

## POST-GRADUATE SURGERY

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*Edited by*

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*With an Introduction*

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*With 846 Figures in the Text*

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THIS WORK IS DEDICATED TO  
THE RIGHT HONOURABLE  
LORD MOYNIHAN OF LEEDS  
K.C.M.G., C.B., M.S., F.R.C.S.  
TO WHOM SURGERY IN GENERAL AND  
ABDOMINAL SURGERY IN PARTICULAR  
OWES SO MUCH

## PREFACE

IN undertaking the production of *Post-Graduate Surgery*, an attempt has been made to cover as far as possible the whole field of surgery in a practical manner. As a certain basic knowledge of surgery has been assumed, opportunity has been afforded to devote more space to a description of special methods of investigation, to an account of recent and approved advances in surgical treatment, and to details regarding the management of cases before and after operation.

This work has been written specially for post-graduates engaged in the practice of surgery, for senior resident officers holding responsible posts on the staffs of hospitals, for Fellowship candidates who require more precise instruction in the details of operative procedure, and for medical officers of the fighting Services, both at home and overseas, as well as for busy general practitioners, who may be called upon to undertake urgent surgical measures and who are desirous of keeping abreast with all the modern developments in surgery.

The main features of this work include :

(1) A wide variety of contributions written in a practical and straightforward manner by well-known authorities, many of whom are teachers and examiners, and each one a specialist in his own subject.

(2) A profusion of illustrations by eminent artists. These drawings include pathological specimens, apparatus, special instruments, and the individual steps of operations, most of these pictures having been drawn during the actual performance of the operation depicted. This wealth of detailed illustrations greatly illuminates the text and facilitates an understanding of the descriptions given.

(3) A consideration of some of the more outstanding and ever-increasingly important medical aspects of surgery.

In a work of this nature, dealing as it does with such a large number of subjects, many of which are closely related, it is impossible to avoid a certain degree of overlapping, repetition, and even difference of opinion. To the student the occasional diversity of opinion expressed may be helpful by encouraging a closer study of such subjects to enable him to form his own conclusions.

In compiling such a vast and responsible work as this, the Editor has been exceedingly fortunate in the invaluable and willing help he has received from so many of his friends and colleagues, not only in this country but also abroad.

To Lord Moynihan I tender my most grateful thanks for his introduction, and for his ready permission to quote certain important extracts from his well-known book, *Abdominal Operations*.

I am also greatly indebted to the writers of the various articles for their loyal co-operation and for the unstinted efforts they have put forward to make their individual contributions a success; to the many authors and publishers who have allowed me to quote from their works and in some instances to adapt or reproduce certain illustrations, the acknowledgments of which I hope are duly mentioned in the captions, text, or references; to the curators of the museums of the Royal College of Surgeons of England and of several teaching hospitals for permitting the artists to draw specially selected or unusual pathological specimens; to the various artists who have devoted much time and skill in the production of so many beautiful illustrations; and to the publishers for their generosity, their readiness in adopting my suggestions, and for overcoming every obstacle in order that success might be achieved.

To my secretary, Miss A. M. Cossham, who has helped me with every step of this work, typing not only my manuscripts but also those of certain of the other contributors, for correcting all the proofs with me, for her untiring work and encouragement throughout our combined labours over what has sometimes appeared to be an endless task, and also for her many helpful suggestions during the final stages of proof reading with Mr. E. Wynne Thomas, who has himself been of inestimable assistance in this matter, my warmest thanks are also due.

RODNEY MAINGOT.

# INTRODUCTION

by

LORD MOYNIHAN

ONE of the defects of English Medicine has hitherto been the neglect of post-graduate teaching. Happily, adequate steps under highest auspices are now being taken, not only to make good the deficiency, but, almost certainly, to put England in her rightful place of leadership.

In London the supply of clinical material is unapproached in quantity and unequalled in quality; the men capable of undertaking post-graduate teaching are at least the equal of any. All that has been lacking up till to-day has been the desire to make this material and these men available for the multitude of those, at home and overseas, who have felt the need of further, more intensive training after undergraduate days are over.

Relevant literature also has been wanting. The problems of undergraduate and post-graduate teaching are not identical. Search for knowledge required in many "Systems" has been arduous and too little profitable. No one book gave what the student felt was necessary.

This new book is therefore urgently needed. Those who work with it will, I truthfully believe, find that it gives them exactly what they seek. The work is essentially practical, as it should be. It will give to the "Fellowship" candidate that full and detailed information as to indications for operations, pre-operative preparation and post-operative care of the patient, and precise details of operative procedures which he may seek in vain elsewhere.

It will be a friendly book of reference for those whose daily tasks make it so difficult a matter to keep abreast in the modern advance of Surgery which continues to be so rapid.

One of the amusing faults in the surgical literature of all countries has been the repetition from author to author, sometimes for centuries, in a few cases for nearly 2000 years, of grievous error.

When I first wrote upon hernia an obvious falsity was, with toil

lightened by the fun it gave me, traced back to its remote ancestor Celsus. To avoid such literary catastrophe the authors of this work have been asked to draw mainly from their own experience. Such choice of authors has been made as will ensure that their teaching and their practice are representative of the best this country can show. Their leadership has been inspiring. Since many of the authors are my personal friends, and as all of them bear names honoured by members of our profession, I need not do more than commend their work and express my delight that so competent a team has been gathered together.

I think the book will carry another message. It will, I believe, convince the young surgeon that, as I have before ventured to say, "Surgery is not only Science, not only Art, it is a Sacrament." To serve a fellow-creature whose health has broken down, whose life is in jeopardy is a task not only for a craftsman, but for one with gifts of compassion, stirred by an earnest desire to succour and to help. Service must rest upon skill which the surgeon strives always to increase, and it must come from the heart. Inspired by these motives he will make his patient "Safe for Surgery" by every device (and they quickly increase in numbers and power) of which he is master. He will consider questions of operability, and he will endeavour so to add to his patient's reserve of strength that the ordeal faced will be met with courage, confidence and success.

For us as surgeons an operation is an incident in the day's work, but for our patients it may be, and no doubt it often is, the sternest and most dreaded of all trials, for mysteries of life and death surround it, and it must be faced alone. Those who submit to operation are confronted, perhaps after long and weary days or months of suffering, with the gravest issues, and far more often than many of us suppose, they pass into the valley of the shadow of death, and, in stark dismay, wonder with Beatrice in her aching solitude and panic what will come to pass

". . . if there should be  
No God, no Heaven, no Earth in the void world,  
The wide, gray, lampless, deep, unpeopled world."

To give courage to those who need it, to restore desire for life to those who have abandoned it, with our skill to heal disease or check its course—this is our great privilege. Ours are not the mild concerns of ordinary life. We who, like the Happy Warrior, are "doomed to go in company with Pain and Fear and Bloodshed," have a higher mission than other men, and it is for us to see that we are not unworthy.

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# CONTENTS OF VOLUME I

## PART I

### ANÆSTHESIA, by C. Langton Hewer

	PAGE
Section 1. GENERAL ANÆSTHESIA . . . . .	3
„ 2. LOCAL ANALGESIA . . . . .	26
„ 3. CHOICE OF ANÆSTHETIC . . . . .	39

## PART II

### ABDOMEN

Section 1. INVESTIGATION OF A CASE OF DYSPEPSIA, by Rodney Maingot, R. Sleigh Johnson and P. M. Deville . . . . .	63
„ 2. STOMACH AND DUODENUM, by Rodney Maingot . . . . .	145
THE PÉAN-BILLROTH I OPERATION, by E. Finochietto . . . . .	526
„ 3. ULCER-CANCER OF THE STOMACH, by F. A. Knott . . . . .	561
„ 4. MEDICAL TREATMENT OF PEPTIC ULCER, by A. F. Hurst . . . . .	573
HÆMATEMESIS, by R. Sleigh Johnson, A. F. Hurst and Rodney Maingot . . . . .	578
„ 5. NEW GROWTHS OF THE DUODENUM—SMALL INTESTINE—DUODENAL ILEUS, by J. Ewart Schofield . . . . .	593
„ 6. GALL-BLADDER AND BILE-DUCTS, by Rodney Maingot . . . . .	627
GALL-STONES AND CHOLECYSTITIS, by A. M. A. Moore . . . . .	723
CONGENITAL CYSTIC DILATATION OF THE COMMON BILE-DUCT, by A. Dickson Wright . . . . .	733
„ 7. LIVER, by A. Dickson Wright . . . . .	739
JAUNDICE, by R. Sleigh Johnson . . . . .	748
„ 8. PANCREAS, by A. Dickson Wright . . . . .	783
DIABETES IN SURGICAL CASES, by R. Sleigh Johnson . . . . .	790
„ 9. SPLEEN, by Rodney Maingot . . . . .	803
RADIO-THERAPY IN SPLENIC DISEASES, by Walter M. Levitt . . . . .	820
THE SPLEEN AND THE BLOOD-PLATELETS, by W. Howel Evans . . . . .	829
EGYPTIAN SPLENOMEGALY, by H. E. S. Stiven . . . . .	898

	PAGE
Section 10. APPENDIX, <i>by Hamilton Bailey</i> . . . . .	909
„ 11. COLON, <i>by Cecil P. G. Wakeley</i> . . . . .	913
„ 12. INTESTINAL OBSTRUCTION, <i>by A. J. Cocklinis</i> . . . . .	973
STREANGULATED HERNIA, <i>by A. J. Cocklinis</i> . . . . .	1034
PERITONITIS, <i>by A. J. Cocklinis</i> . . . . .	1037
PERITONEAL ABSCESSSES, <i>by A. J. Cocklinis</i> . . . . .	1142
„ 13. ILEUS, <i>by the late H. Tyrrell-Gray</i> . . . . .	1181
„ 14. COMPLICATIONS FOLLOWING ABDOMINAL OPERATIONS, <i>by Rodney</i> <i>Mamgot</i> . . . . .	1215
POST-OPERATIVE PHLEBITIS, <i>by A. Dickson Wright</i> . . . . .	1256

## PART III

RECTUM AND ANUS, <i>by W. Ernest Miles</i> . . . . .	1261
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## PART IV

X-RAY DIAGNOSIS, *by H. Cecil Bull*

Section 1. RADIOLOGY OF THE ALIMENTARY TRACT . . . . .	1483
„ 2. RADIOLOGY OF THE URINARY TRACT . . . . .	1540

## PART V

## RADIUM TREATMENT

Section 1. RADIUM TREATMENT OF MALIGNANT DISEASE, <i>by Stanford Cade</i> . . . . .	1557
„ 2. RADIO THERAPY IN DISEASES OF WOMEN, <i>by Malcolm Donaldson</i> . . . . .	1625

INDEX . . . . .	1685
-----------------	------

PART I  
ANÆSTHESIA  
by  
C. LANGTON HEWER

SECTION 1  
GENERAL ANÆSTHESIA

CHAPTER I  
Mode of Action of Anæsthetic Agents

CHAPTER II  
Nitrous oxide—Ethylene—Other Hydrocarbon Gases

CHAPTER III  
Ethyl chloride—Ether (di-ethyl and di-vinyl)—Chloroform—Evipan

CHAPTER IV  
Endotracheal Anæsthesia

CHAPTER V  
Premedication and Basal Narcosis

CHAPTER VI  
Resuscitation

SECTION 2  
LOCAL ANALGESIA

CHAPTER I  
*General Considerations*

CHAPTER II  
*Drugs and Apparatus*

CHAPTER III  
*Technique*

CHAPTER IV  
*Spinal Analgesia*

SECTION 3  
CHOICE OF ANÆSTHETIC

CHAPTER I  
*General Considerations*

CHAPTER II  
*Cranial Surgery*

CHAPTER III  
*Surgery of the Neck*

CHAPTER IV  
*Thoracic Surgery*

CHAPTER V  
*Abdominal and Perineal Surgery*

## SECTION 1

### GENERAL ANÆSTHESIA

#### CHAPTER I

##### MODE OF ACTION OF ANÆSTHETIC AGENTS

It is now generally recognised that general anæsthesia can be produced in at least two different ways.

In the first place, when an inert gas which is soluble in blood is inhaled, it will displace oxygen in all the body fluids until the cells which have the highest oxygen consumption cease to function. There is evidence to indicate that these cells are those of the cerebral cortex, so that the first main effect of the inhalation of the gas will be loss of consciousness. If sufficient oxygen be now added to the inhaled gas to maintain this constant percentage, general anæsthesia will result. Nitrous oxide is the best example of a soluble gas which acts in a purely physical way by replacing oxygen.

Secondly, if a volatile lipid-soluble drug be inhaled, it will enter those cells richest in lipoids and by exerting a toxic effect will cause the cells temporarily to cease functioning. It is probable that the actual mechanism is a reversible coagulation of protein. Chloroform is a drug which acts in this way, and the resulting anæsthesia is far more profound and is accompanied by much greater muscular relaxation than that afforded by the "oxygen-replacer" method. Unfortunately other cells besides those of the cerebral cortex are rich in lipid bodies, and in rare instances the transient toxic effects of the drug may be replaced by permanent changes, as in atrophy of the liver. The lipid-soluble drugs also affect the metabolism of the body as is shown by changes in the blood-sugar and blood-urea values, etc. Unlike nitrous oxide, these drugs are not eliminated as quickly as they are absorbed, and hours and even days may elapse between the end of their administration and their complete removal from the body.

Between the two extremes of nitrous oxide on the one hand and chloroform on the other, the remaining anæsthetic agents can be

placed in a definite order as regards toxicity, rate of elimination and degree of muscular relaxation obtainable. The following table is arranged showing this order for the commoner anæsthetics.

<i>Anæsthetic Agent.</i>	<i>Toxicity.</i>	<i>Muscular Relaxation Obtainable.</i>	<i>Rate of Elimination.</i>
Nitrous oxide.	Nil in presence of adequate oxygen.	Practically nil without "secondary saturation" or premedication.	Equal to that of its absorption.
Ethylene.	Slight. (Nausea sometimes marked.)	Poor.	Rapid.
Ethyl chloride.	Slight. (Nausea usual, vomiting sometimes.)	Fair.	Rapid.
Ether (di-ethyl).	Fairly high. (Often vomiting, headache and persistent taste and smell.)	Good.	Slow.
Chloroform.	High. (Vomiting sometimes severe with acidosis. Also cardiac effects.)	Excellent.	Very slow.

From the foregoing very condensed account, it will be obvious that it is desirable to keep as near the "oxygen-replacer" end of the scale as possible, using only sufficient of the lipoid-soluble drugs to secure adequate relaxation for the proposed operation. This, then, constitutes the ideal in modern general anæsthesia.

## CHAPTER II

### NITROUS OXIDE, ETHYLENE, AND OTHER HYDROCARBON GASES

NITROUS OXIDE is a colourless and odourless gas prepared from ammonium nitrate. The gas is stored in liquid form in cylinders at a pressure of about 1500 lb. per square inch. Until recently these cylinders were made of heavy carbon steel, but nickel-chrome-molybdenum steel of greater strength and corresponding lightness is now being employed. Commercial nitrous oxide is extremely pure, and can now be regarded as free from water, from the higher oxides of nitrogen, and from carbon monoxide. A small percentage of nitrogen is usually present, but most of this is given off in the gas first delivered from the cylinder.

It has already been noted that nitrous oxide is rapidly absorbed by the blood through the pulmonary alveoli. The gas then acts in a purely physical way by replacing oxygen, and produces no toxic effects whatever unless gross anoxæmia is allowed to occur.

#### NITROUS OXIDE AND AIR

Nitrous oxide can be administered for short operations not requiring muscular relaxation with the simple apparatus illustrated. Pure gas is inhaled from the bag with the valves in action until anæsthesia is established (usually shown by a change in the respiratory rhythm accompanied with slight stertor), after which the three-way stopcock is alternately moved to "air" and "rebreathing" as requisite. It is not always possible to avoid a mild degree of asphyxia with this method, so that it should not be used in cases of uncompensated cardiac disease, respiratory obstruction, etc. Since young children are particularly intolerant to oxygen deficiency, it is usually impossible to anæsthetise



Fig. 1.—APPARATUS FOR THE ADMINISTRATION OF NITROUS OXIDE AND AIR.

them satisfactorily with nitrous oxide and air. When employed in suitable cases, however, this method is extremely safe and satisfactory, one series of no less than 300,000 administrations having been recorded without fatality.

#### NITROUS OXIDE AND OXYGEN

If pure oxygen is added to the inspired nitrous oxide in a definite and adjustable proportion, a smooth continuous anaesthesia can usually be maintained provided that the patient has had adequate premedication (see page 19). In order to attain this end, various types of apparatus have been designed. These operate on three main principles.

*Continuous-flow Apparatus.* Here the gases flow at a constant rate so that partial rebreathing by means of a bag and expiratory valve is necessary. The rates of flow and consequently the relative proportions of nitrous oxide and oxygen are measured by means of :

- (1) "Wet" flow-meters, the gases either bubbling through or depressing a column of water.
- (2) Bohn type flow-meters, where the rate of flow can be estimated from the height of columns enclosed in transparent tubes.
- (3) Gauges which are calibrated direct in litres per minute or gallons per hour.

*Intermittent-flow Apparatus.* In this type of machine the gases normally flow during inspiration only. If desired, the pressure can be raised to any reasonable degree without upsetting the proportions of gases. With this type of apparatus, partial rebreathing is usually desirable, but is not essential, and great accuracy in gas percentage is possible.

*Closed-circuit Apparatus.* Total rebreathing is employed in this type of machine, the carbon dioxide being absorbed either by caustic soda solution or, more generally, by granulated soda-lime in a container which is situated either close to the face-piece (see fig. 6) or on the main apparatus. In the latter case, double wide-bore tubing with valves is usual in order to ensure a one-way flow. This principle provides extremely quiet respiration, and affords great economy in nitrous oxide, as when anaesthesia has been established, oxygen only need theoretically be added. In practice, however, very small additional amounts of nitrous oxide are necessary. In order to obtain the best results, it



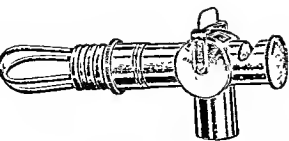


Fig. 2.—DETAILED VIEW OF 3-WAY STOPCOCK SHOWN IN PREVIOUS FIGURE.

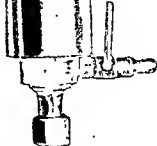


Fig. 4.—REDUCING VALVE. (BEARD'S.)

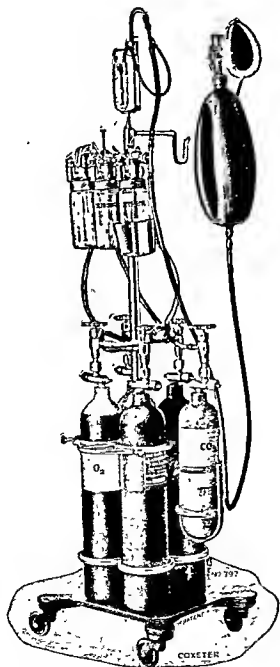


Fig. 3.—CONTINUOUS-FLOW APPARATUS UTILISING "WET" FLOW-METERS. (BOYLE'S.)

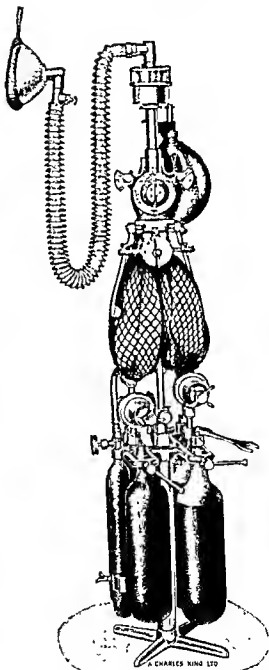


Fig. 6.—INTERMITTENT-FLOW TYPE OF GAS-OXYGEN APPARATUS. (MAGILL'S.)

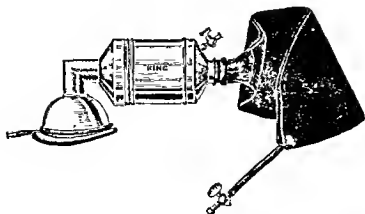


Fig. 6.—CARBON DIOXIDE ABSORBER. (FOREDOER'S.)

is essential to use non-hygroscopic dust-free soda-lime. This material can now be obtained coloured green, a change to brown showing that it has become exhausted.

In order to ensure the even flow of gas, it is desirable to employ reducing valves on nitrous oxide cylinders, used with continuous-flow and closed-circuit machines. The oxygen flow, on the other hand,

DIAGRAMMATIC REPRESENTATION  
OF APPARATUS

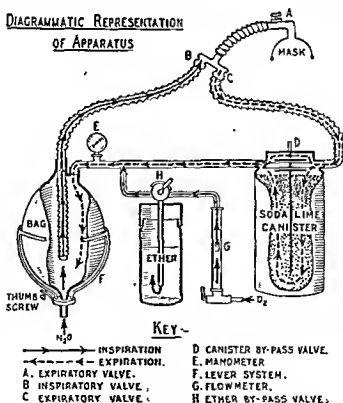


Fig. 7.—DIAGRAM SHOWING ONE OF THE MANY POSSIBLE CIRCUITS OF A TOTAL REBREATHING APPARATUS (IN THIS CASE THAT OF DR. T. A. B. HARRIS) WITH THE ABSORBER FITTED ON TO THE MAIN MACHINE.

remains reasonably constant if a simple fine-adjustment valve of good design is employed.

Practically all types of gas-oxygen apparatus have provision for the addition of ether, chloroform, and carbon dioxide to the inspired mixture.

### ETHYLENE

is an unsaturated hydrocarbon gas having the chemical formula  $C_2H_4$ . When chemically pure, the compound has a faint ethereal odour, but in its commercial form the smell is much stronger and more unpleasant, resembling that of acetylene.

Ethylene can be given in the same way and with the same apparatus as has been described for nitrous oxide. The chief differences between the two gases are the higher percentage of oxygen which can be employed with ethylene and the better relaxation obtainable.

The main disadvantages of ethylene are its smell, its explosibility when mixed with air or oxygen, and the tendency to cause prolonged nausea after operation.

Ethylene is used extensively in America but in this country the general opinion seems to be that it shows little advantage over nitrous oxide oxygen with minimal ether.

### OTHER HYDROCARBON GASES

Purified *acetylene*,  $C_2H_2$ , under the title of "nareylen" is used as an anæsthetic fairly extensively in Germany. The gas is used in a similar way to ethylene, the unpleasant odour being disguised by pine essence.

Experimental work is still in progress with regard to the use of *propylene* and its isomer *cyclopropane* for anæsthesia. Although encouraging results have been obtained, it is too early to express any definite opinion on the merits of these two gases. On the whole cyclopropane seems to offer most possibilities, but at present its price is such that it can only be used in closed-circuit apparatus. When given in this way with a high proportion of oxygen, cyclopropane gives good relaxation with a very rapid recovery. The writer has been considerably impressed with the excellent results obtainable with cyclopropane in major thoracic surgery.

## CHAPTER III

### ETHYL CHLORIDE, ETHER [DI-ETHYL AND DI-VINYL], CHLOROFORM, AND EVIPAN

#### ETHYL CHLORIDE ( $C_2H_5Cl$ )

is a gas at normal temperatures and pressures, but is usually stored in liquid form in small glass containers fitted with spring lever taps. The pure drug has an unpleasant smell which is disguised by most manufacturers by the addition of a small quantity of eau de-Cologne.

An impure form of ethyl chloride can be obtained in ampoules fitted with a very fine jet, and is intended for freezing the skin in order to make small incisions. This method is of little use, as thawing may be extremely painful and the vitality of the tissues may be impaired.

The pure drug may be administered in order to produce general anaesthesia by the closed or the open method. If the former technique be adopted, it is wise always to insert a dental prop before the induction is begun in case masseteric spasm takes place. Ethyl chloride given by the "single-dose" method is extremely useful for short operations in children, but its administration should not be continued indefinitely. It can, however, be used as a preliminary to open ether for longer operations in children, but this sequence is not so satisfactory in adults. The drug can be used in a Clover's inhaler to start a closed ether induction if nitrous oxide is not available.

"Sonnoform" is a mixture of ethyl chloride, methyl chloride, and ethyl bromide having a similar effect to ethyl chloride.

#### ETHER ( $C_2H_5$ )<sub>2</sub>O

A great deal of research work has been carried out upon di-ethyl ether since its adoption for anaesthesia in 1842. At the present time the product of reputable manufacturers can be regarded as practically free from aldehydes and peroxides. Subsequent decomposition is retarded by storage in opaque containers, preferably lined with copper, by saturation with carbon dioxide, or by the addition of traces of

hydroquinone. Many attempts have been made to disguise the unpleasant taste and smell of the drug, one of the most successful being the addition of 10 minims of pure oil of peppermint to the pint of ether.

The induction of anæsthesia by means of ether in adults is still usually accomplished by means of Clover's inhaler or one of its modifications. The high carbon dioxide percentage obtaining in closed inhalers causes deep respiration and consequently rapid narcosis. Nitrous oxide or ethyl chloride can be used as a preliminary to ether in Clover's inhaler, and this sequence is convenient for the general practitioner who has no elaborate apparatus. Ether anæsthesia is generally maintained by an open or semi-open method, as the prolonged hyperpnœa which would occur if narcosis was continued with a closed inhaler is undesirable. Warmed vapour delivered from Shipway's or some

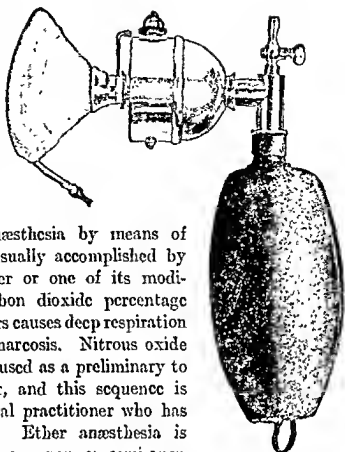


Fig. 8.—CLOVER'S INHALER.

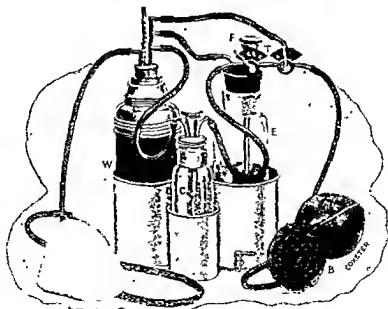


Fig. 9.—WARM ETHER-VAPOUR APPARATUS. (SHIPWAY'S.)

similar type of apparatus is preferable to douching the outside of the mask with liquid ether, but gross overheating should be avoided as this causes excessive sweating. It has already been emphasised that, if facilities permit, the addition of a small amount of ether vapour to a mixture of nitrous oxide and oxygen is preferable to the use of the pure drug. Ether is now generally administered in this way either with a face-piece or by means of an endotracheal tube.

The *intravenous* infusion of 5 per cent ether in saline and the *rectal* instillation of oil-ether are now seldom employed.

If any considerable amount of ether is to be administered, a full dose of atropine should always be incorporated with the pre-operative medication to avoid undue salivation, while "de-etherisation" with carbon dioxide should be practised at the end of the administration. If a carbon dioxide cylinder is not available, recourse may be had to the portable "sparklet" device. The carbon dioxide in the inhaled



Fig. 10.—SPARKLET HOLDER.

mixture should not, as a rule, exceed 10 per cent. There is no doubt that the routine employment of  $\text{CO}_2$ -air or  $\text{CO}_2$ -oxygen mixtures after prolonged ether anaesthesia diminishes the incidence of pulmonary complications.

"Late ether convulsions," so called to distinguish them from the innocuous "ether tremor" occasionally present in light anaesthesia, have recently been the subject of much investigation. The condition is commonest in hot weather or when the ether or its vapour has been beaten. It usually occurs during deep or prolonged anaesthesia especially in the presence of sepsis, and has been described with all types of administration. The cause of these convulsions has been variously ascribed to overdose, to impurities in the ether, to cerebral congestion, and to alkalosis. If the condition is recognised in its initial stages, it can generally be aborted by the inhalation of carbon dioxide and oxygen, by raising the patient's head, or by momentary compression of the common carotid arteries.

Ether causes some metabolic changes in the body and acetonuria, hyperglycaemia, albuminuria, and a raised blood-urea concentration

are common after a prolonged administration. The two former changes can be minimised by a suitable dose of insulin given before operation.

It seems probable that some of the other ethers may eventually prove of service in anæsthesia, *di-vinyl ether* ( $C_2H_3)_2O$  appearing to offer the greatest possibilities in this direction. This drug, also known as "vine-thene" in commercial form, boils at  $28^\circ C.$ , and is consequently more volatile than di-ethyl ether. There is little difference between the inflammability of the two ethers, but the di-vinyl variety is less pungent, less irritating, and causes less after-effects than di-ethyl ether. The main disadvantage of the new drug is its instability, and it should always be used from a fresh stoppered bottle. The writer has been favourably impressed with the results obtained to date with this drug.

#### CHLOROFORM ( $CHCl_3$ )

It is unfortunate that the drug which gives the most perfect surgical anæsthesia should be extremely toxic. Chloroform can cause death in at least three different ways.

(1) By primary cardiac failure. Two varieties of this condition are described: vagal stimulation leading to cardiac arrest and ventricular fibrillation. Both these types are commonest during light anæsthesia, the former probably being unlikely if a full dose of atropine has been given previously. The latter variety is thought to be precipitated by an excess of adrenalin in the circulation, which condition may be occasioned by excessive emotion prior to the induction of anæsthesia or by the application of adrenalin to raw areas. It has been observed for many years that women in the late stages of pregnancy appear to be relatively immune from primary cardiac failure during light chloroform anæsthesia, but the reason still remains obscure. The treatment for the condition is considered later (see page 24).

(2) By gradual paralysis of the respiratory centre from overdosage.

(3) By acute yellow atrophy of the liver in "delayed chloroform poisoning."

Chloroform also tends to lower the blood-pressure and causes grave interference with metabolism. All the changes which have been noted with ether occur to an even greater extent with chloroform. Some of these can be modified by a pre-operative dose of insulin.

From the foregoing considerations it will be seen that chloroform is a toxic drug, and its use should be restricted to a minimum. In minute doses, however, the drug may be of great service when combined with nitrous oxide and oxygen if relaxation is required and ether is contra-indicated. Such operations as diathermy of the air-passages or extensive thoracotomies may be cited as examples.

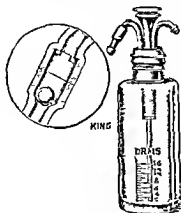


Fig 11.—JUNKER'S BOTTLE FITTED WITH BALL VALVE IN INLET TUBE.

If it is necessary to give chloroform alone, the open lint method is preferable to elaborate dosimetric apparatus. Chloroform vapour is still sometimes delivered into the mouth or pharynx by means of Junker's apparatus. Some safety device should always be fitted to obviate any possibility of liquid chloroform reaching the delivery tube if the apparatus is wrongly assembled.

#### MIXTURES OF CHLOROFORM AND ETHER

Various proportions of chloroform and ether are used in mixtures but they must be regarded as diluted chloroform possessing all the dangers of the pure drug. The added ether tends to make respiration deeper and to minimise the fall of blood-pressure which occurs in pure chloroform anaesthesia.

#### EVIPAN

Although evipan or, strictly speaking, sodium evipan, does not belong to the class of volatile anaesthetics, it can conveniently be considered here. This drug is the sodium salt of N-methyl-c-c-cyclohexenyl-methyl barbituric acid, and has been introduced recently as an intravenous anaesthetic of short duration. The preparation used is a freshly made 10 per cent solution of the powder in sterile distilled water which is injected at the rate of 1 cc. in 15 seconds until consciousness is lost. The volume of solution injected is noted, and an equal amount (in elderly or feeble patients half the amount) is added, the maximum permissible dose in adults being usually regarded as 10 cc. Short operations requiring a moderate amount of muscular relaxation can often be performed under evipan narcosis alone, and if necessary, a second injection can be made. In the writer's opinion, however, the "continuous" method is preferable to the orthodox



technique which has been described. The needle is kept in position throughout the operation and fluid is injected until reflex movements are just abolished. In this way the extremely deep narcosis which occurs at the beginning of the operation by the first method is avoided. The anæsthetist must be prepared to maintain a satisfactory airway and to take all the precautions that are necessary in any other form of general anæsthesia. Evipan tends to depress respiration and to lower blood-pressure, so that it is unwise to give large doses of morphia before operation, and the administration of respiratory stimulants may be advisable. Evipan is not recommended for patients who are very toxæmic or jaundiced or who have respiratory or cardiac embarrassment. It should not be given in the sitting or the reversed Trendelenburg positions.

It is difficult as yet to assess the safety of evipan accurately. When given intelligently to suitable patients, the method appears to be reasonably safe, most of the fatalities being apparently due to faulty technique. For example, a series of 6,500 administrations in Germany resulted in 8 deaths from the method, 7 of which appeared to be due to overdosage. In the same series, five cases of venous thrombosis and two of tissue necrosis were reported. Other sequelæ which have occurred after evipan narcosis are: persistent drowsiness, headache, extreme restlessness, nystagmus, and photophobia, while in children mental changes have been noted.

It must be admitted that some of the fatalities reported recently are disquieting, as in several cases there was no obvious error in administration, and on post-mortem examination the patients appeared to be healthy subjects. It is possible that some of these cases were inadequately prepared beforehand, and that syncope resulted from a full stomach.

It should be remembered that while a non-volatile drug injected intravenously provides a simple and rapid method of inducing a short anæsthesia, it is much less controllable than inhalation techniques, and it is doubtful whether a pure intravenous narcosis will ever be used as a routine procedure.

## CHAPTER IV

### ENDOTRACHEAL ANÆSTHESIA

By endotracheal anæsthesia is meant the administration of anæsthetic vapour through a tube whose distal end lies in the trachea. This method was introduced in 1909 and was developed during the Great War for such operations as plastic repairs of the face.

The chief *indications* for endotracheal anæsthesia are :

(1) Operations in which respiratory obstruction may be expected either from the surgical technique, the nature of the disease, the position of the patient, or the introduction of fluids into the air-passages.

(2) Most prolonged operations upon the head and neck where the patient's face is inaccessible to the anæsthetist, except those for the relief of toxic goitre (see page 45).

(3) Most prolonged upper abdominal operations in which nerve or field blocking is not employed.

It should be noted that two distinct *techniques* are possible with endotracheal anæsthesia :

(1) The *Insufflation* method.

In this case a relatively small-bore catheter is passed through the glottis and the anæsthetic vapour is forced through this under pressure,



Fig 17.—CATHETER FOR INSUFFLATION ENDOTRACHEAL ANÆSTHESIA.

the return flow taking place either round the catheter or through a second wider tube whose end also passes through the glottis. .

(2) The *Inhalation* method.

This technique has tended to replace insufflation and is accomplished by passing a wide-bore rubber tube or open-ended catheter through the larynx, the glottic opening being then completely filled. This tube can thus be regarded as a continuation of the trachea, and to-and-fro

respiration takes place through it. If there is any chance of fluids being liberated into the pharynx, moist gauze can be packed around the tube. It will be obvious that practically any method of inhalation

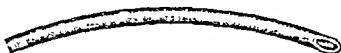


Fig. 13.



Fig. 14.



Fig. 15.

Fig. 14.  
Fig. 15.  
RUBBER TUBE FOR INHALATION ENDOTRACHEAL ANÆSTHESIA WITH ORAL AND NASAL ANGLE PIECES. (MAGILL'S.)

anæsthesia can be adapted for use with this technique, a fact which may be of great service if no apparatus is available. For reasons which have already been stated, however, it is preferable to use nitrous oxide—oxygen with or without minimal ether wherever possible. For this

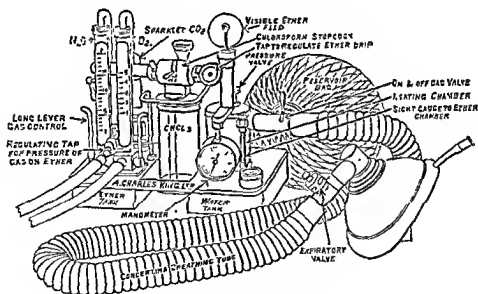


Fig. 16.—CONTINUOUS-FLOW GAS-OXYGEN APPARATUS SUITABLE FOR ENDOTRACHEAL ANÆSTHESIA. (MAGILL'S.)

purpose any continuous or intermittent-flow apparatus can be used, but it is advantageous to have a manometer fitted to the delivery tube, a blow-off valve to avoid excessive pressures, and some device to prevent the delivered vapour being at an unduly low temperature.

*Intubation.* Tracheal tubes can be passed either through the nose or through the mouth according to the requirements of the operation.

Oral intubation is best performed under full general anaesthesia with the patient's head flat (*not* extended). A direct-vision laryngoscope should be used to expose the glottis. If the nasal route is used, a thorough preliminary cocaineisation of the nose

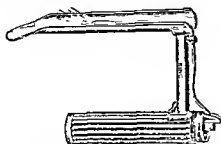


Fig. 17.—DIRECT VISION LARYNGOSCOPE SUITABLE FOR INTUBATION. (MAGILL'S.)



Fig. 18.

and glottis may enable the tube to be inserted direct under very light anaesthesia or even with the patient fully conscious. This method may be of great use if the mouth cannot be opened. If "blind" intubation fails, the tube should be passed through the nose into the pharynx: a laryngoscope is then passed through the mouth and the end of the tube guided by manipulation or by long forceps into the glottis. Whatever method of intubation be employed, it is essential that no force should be used.

## CHAPTER V

### PREMEDICATION AND BASAL NARCOSIS

For many years, nervous patients have been given sedative drugs before operation with beneficial results. For example, a suitable dose of morphia and scopolamine will usually render a patient so sleepy that he loses his apprehension, whilst the amnesic effect is such that he frequently remembers nothing of the induction of general anaesthesia. More recently, the tendency has been to go farther than mere "sedation," and to employ drugs which will render a patient completely unconscious in his bed before removal to the operating theatre. These drugs are termed "basal narcotics," and it should be noted that with the exception of sodium evipan their dosage cannot safely be increased to produce general anaesthesia. The basal narcotics in general use will now be briefly considered.

#### PARALDEHYDE ( $C_6H_{12}O_3$ )

administered per rectum in a 10 per cent solution of water is probably the safest basal narcotic known. The usual dosage is from 50 minims to 1 drachm of the pure drug to each stone of the patient's weight, but it should rarely be necessary to exceed 8 drachms. In this dosage, paraldehyde has practically no effect on blood-pressure or respiration, and leaves the reflexes almost unimpaired. This method is of great service in nervous children, but adults may complain subsequently of the unpleasant smell, since paraldehyde is partly excreted through the lungs. Induction and recovery are usually perfectly tranquil, but excitement is occasionally seen.

#### AVERTIN ( $CBBr_3CH_2OH$ )

or tribrom-ethyl alcohol is usually supplied dissolved in amylene hydrate, the resulting "avertin fluid" containing 1 gramme of the solid drug per cubic centimetre. The rectal injection must be freshly prepared by dissolving the avertin fluid in warm distilled water in a

concentration of about 2.5 per cent. A few drops of Congo Red dye are added to the solution, any change of colour towards blue indicating decomposition and an unserviceable preparation. The usual dose of avertin is from 0.09 to 0.1 grammes per kilogramme body weight, and can quickly be calculated from tables. Avertin has practically no smell, but causes a fall in blood-pressure and depresses both respiration and protective reflexes. It is generally held that avertin should not be used in patients with myxoedema, with abnormally low basal metabolic rates, or in those who are gravely toxæmic. Avertin should not, as a rule, be combined with other methods of general or local anaesthesia, which themselves cause an appreciable fall of blood-pressure, and care must be used if the drug is employed for nose and throat operations in which blood or other fluids can gain access to the respiratory passages during the stage of recovery.

#### BARBITURATES

comprise a very large group of drugs, many of which have been tried as basal narcotics. Some of these compounds in common use will now be considered separately.

*Nembutal* can be given by mouth in capsule form about  $1\frac{1}{2}$  hours before operation. Three to  $4\frac{1}{2}$  grains (2 to 3 capsules) is the usual adult dose. The oral administration of this drug cannot always be relied upon to produce actual unconsciousness, but "sedation" and subsequent amnesia is usually satisfactory. Nembutal can also be given intravenously, the freshly prepared solution being injected at a rate not exceeding 1 cc. per minute until a reply to questions can no longer be elicited from the patient.

*Sodium amytal* can be used in the same way as nembutal, i.e. by a freshly prepared intravenous injection, or by oral capsules, each containing 3 grains of the drug.

*Sodium soneryl* is usually administered by mouth in capsules containing  $2\frac{1}{4}$  grains each. The usual adult dose is from 3 to 5 capsules, depending to some extent upon the patient's weight. This drug appears to exert a more constant effect than does oral nembutal.

*Hebaral sodium* is also given by mouth in 3 grain capsules. The claim that the drug is eliminated more rapidly and completely than most of the other barbiturates seems to be substantiated in that "hang-over" effects are minimized.

*Pernocton* is a barbiturate whose molecule contains a bromine atom. It has the advantage of being stable in solution so that the drug

can be injected intravenously direct from ampoules. The rate of injection should not exceed 1 cc. per minute.

*Sodium evipan* can be used as a basal narcotic, but is generally employed as a short general anæsthetic. It has already been discussed on page 14.

It should be noted that in every type of basal narcosis no further depressant drug should be given after operation unless the patient is restless or complaining of pain.

The most reliable antidote to inadvertent overdosage with avertin or the barbiturates appears to be coramine, a drug which is usually given intramuscularly, but which may, in cases of urgency, be injected into a vein. Repeated lumbar puncture is also said to be of service in barbiturate poisoning, as some of the drug is excreted into the cerebrospinal fluid. Respiratory diffusion should be treated by 5-10 per cent  $\text{CO}_2$  in air or oxygen, combined, if necessary, with artificial respiration.

Basal narcotics should not be given if there is reason to think that the patient's hepatic or renal functions are not satisfactory.

## CHAPTER VI

### RESUSCITATION

RESTORATIVE measures may become necessary during an operation performed under any form of general or local anæsthesia. It is customary to distinguish between collapse of gradual and sudden onset, and these two types will now be considered separately.

#### OPERATIVE SHOCK

is a term which is loosely but conveniently applied to the gradual deterioration in a patient's condition during a prolonged or severe operation. The usual differentiation between primary and secondary shock is of little service since overlapping invariably occurs.

The *signs* of operative shock are pallor, sweating, coldness of the skin, rising pulse-rate, falling blood- and pulse-pressures, spaced or sighing respiration, and a dilated pupil. (The last two signs may be masked by the type of anæsthesia employed.)

The *pathology* of shock is extremely complicated and still imperfectly understood, but from the anæsthetist's point of view, the main change can be regarded as a diminution in volume of circulating blood, with an accumulation in the capillaries of the relaxed muscles.

The *causes* of operative shock can be briefly summarised as :

- (1) Loss of fluid (a) by visible and invisible sweating.  
(b) by hæmorrhage.
- (2) Trauma (a) to nerves, producing "reflex shock"; probably by excessive stimulation of the vasomotor centre.  
(b) to muscles and other tissues producing toxins which are absorbed and give rise to "histamine shock." It has been proved that chloroform and ether sensitise the capillaries to the action of histamine so that its effect is increased. Nitrous oxide, however, has no such action.



(3) Overdosage with general or local anæsthetics or oxygen deprivation from obstructed airway or other cause. This, with the diminished volume of blood in circulation, may give rise to acidosis. Operative shock due mainly to anæsthetic overdosage can be distinguished from that produced by other causes by means of the "depression test." A large volume of oxygen is suddenly added to the inspired mixture and the blood-pressure is watched. An immediate rise indicates overdosage (especially with nitrous oxide and ethylene), while no appreciable change means that the shock is due mainly to other factors.

(4) Prolonged loss of temperature from exposed viscera, etc. Both general and spinal anæsthesia disturb the normal heat-regulating mechanism of the body, so accentuating the effects of heat loss.

Operative shock is seldom due to one cause alone, and many of the above factors may obtain at the same time.

The *treatment* of shock occurring during an operation must be adapted to individual cases, but may include the following measures :

(1) The head should be lowered, and if practicable a steep Trendelenburg position may be adopted.

(2) Oxygenation of the blood should be maintained at the highest possible level by the observance of a perfectly clear airway and, if necessary, by increasing the oxygen percentage in the inspired mixture. In certain cases the addition of 5 per cent carbon dioxide may be desirable in order to stimulate respiration and raise the blood-pressure.

(3) In all severe cases of shock the administration of fluids should be undertaken. Unless an immediate effect is imperative, the rectal route is to be preferred, since there is no danger of overloading the heart, and a prolonged action is obtained. The slow administration of 5 per cent glucose in normal saline at 110° F. is the usual procedure, and it should be noted that an anæsthetised patient will retain much more than a conscious one, a litre being a fair average in the former case. Intravenous injection must be carried out with care, the optimum effect being obtained when the systolic pressure has risen to within 10 per cent of that shown at the beginning of the operation. A smaller volume of fluid is inefficient while a larger one introduces the risk of cardiac dilatation and pulmonary œdema. Six per cent gum acacia in normal saline (Bayliss' solution) is sometimes used for intravenous injection with the idea of producing a longer effect by increasing the viscosity of the added fluid. More recently, a hypertonic glucose

solution (50 to 100 cc. of 50 per cent chemically pure glucose) has been given intravenously with beneficial results. This is curious since it has been shown that the blood-sugar is increased during shock, and that insulin simultaneously lowers this value and raises the blood-pressure. It seems possible that the chief effect of the glucose is the alteration of viscosity produced. Finally, if hæmorrhage has been one of the main factors in the production of the shock, a blood-transfusion may be of the greatest benefit.

(4) The body temperature must be maintained during and after operation by such means as an electric cradle or electrically heated blankets, which are safer and more efficient than hot water bottles.

(5) The administration of stimulating drugs may be adopted to tide over an emergency, but too much reliance should not be placed on them nor should they be used in cases where the myocardium is known to be degenerated. The preparations in common use for this purpose are pituitrin, coramine, and icoral, all of which are given intramuscularly.

#### PRIMARY CARDIAC FAILURE

is entirely distinct from operative shock, and is characterised by extremely sudden onset.

The *signs* of this condition are painfully obvious. The patient's colour suddenly changes to a grey pallor, the pulse is imperceptible, the pupils dilate widely and do not react, while the respiration usually continues for a time and then becomes sighing in character before finally ceasing.

The *causes* of primary cardiac failure are various. The condition seems to occur most frequently in association with light anaesthesia produced by chloroform or a mixture containing it. A severe stimulus applied during light anaesthesia, the presence of natural or injected adrenalin in the circulation and "status lymphaticus" may be factors in the production of the condition. Primary cardiac failure has also been seen during the injection of local anaesthetics, and indeed from the mere surface application of cocaine to susceptible patients.

The *treatment* of this alarming condition must be instituted immediately, as the sole hope of permanent recovery lies in the restoration of circulation before the cells of the cerebral cortex have suffered irreparable damage from anaemia, a period which probably does not exceed eight minutes.

(1) The patient should be placed in a steep Trendelenburg position.

(2) Artificial respiration must be instituted, preferably by means of 90 per cent oxygen and 10 per cent carbon dioxide given under slight pressure.

(3) If there is no response, cardiac puncture should be performed. The right auricle is reached by inserting the needle through the third right intercostal space close to the sternum and directed towards the mid-line. If the puncture fails to elicit any result, 1 cc. of 1 in 1000 adrenalin solution may be injected through the needle.

(4) Cardiac massage must be instituted as soon as it is evident that cardiac puncture has failed. Having opened the abdomen, the heart can often be efficiently massaged in children, but for adults it is usually necessary to incise the diaphragm. If no undue delay has occurred, most hearts can be restarted by massage, but the abdomen should not be closed until it appears certain that the regular beating is permanent.

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## SECTION 2

### LOCAL ANALGESIA

#### CHAPTER I

##### GENERAL CONSIDERATIONS

LOCAL analgesia is a general term covering all methods of rendering a limited area of the body insensitive without abolishing consciousness.

It is outside the scope of this chapter to go into the many arguments which have been evolved for and against the routine adoption of local analgesia for surgery, and it is only possible to touch upon the main points here.

In the first place, it has been known for many years that the reflex effects of severe traumatic stimuli are not completely abolished by general anaesthesia, although the deeper the narcosis the less marked will be these effects. If, however, these stimuli are prevented from reaching the central nervous system, either by paralysing the nerve endings at the site of operation (infiltration) or by paralysing the nerves supplying this area (nerve or field block), reflex effects such as fall in blood-pressure will not occur.

Secondly, local analgesia may make an operation easier from the comparative ischaemia which it produces. Although all local anaesthetics except cocaine are vasodilators, it is customary to incorporate adrenalin in the injected solution so that absorption into the blood stream is retarded, and the resulting bloodless field may be of considerable service in such operations as thyroidectomy. The possibility of reactionary haemorrhage after the vasoconstriction has passed off must, however, be borne in mind.

If an operation demands complete relaxation, it may be preferable to paralyse the nerve supply to the appropriate muscles with a local anaesthetic rather than to obtain general relaxation by means of a lipid-soluble general anaesthetic. This is especially the case if the patient is suffering from pulmonary disease or grave toxæmia.

Lastly, minor operations can often be performed single-banded under local analgesia, little preliminary preparation is necessary, and after-effects are rarely marked.

On the other hand, local analgesia has definite disadvantages and

contra-indications. For example, it should not, as a rule, be employed in children, nor for operations in which the injection would have to be made through infected tissues. In debilitated subjects extensive sloughing may subsequently occur.

It must not be forgotten that local anæsthetics must be regarded as poisons having a special affinity for nerve tissue, and that they can, under certain conditions, give rise to severe toxic symptoms and even death. These effects are most likely to occur with the more toxic drugs, when large volumes of solution are used, and especially when an appreciable quantity is inadvertently injected into a vein. In these circumstances, the vital medullary centres may be affected or primary cardiac failure (see page 24) may occur.

To many patients the performance of a major operation under local analgesia alone is an insupportable ordeal, and at the present time local methods are frequently combined with nitrous oxide-oxygen anæsthesia. It might be thought that the ideal combination would be basal narcosis and local analgesia, especially as it has been proved that the barbiturates decrease the toxicity of cocaine derivatives. This, however, is not borne out in practice. It is impossible to be certain that a patient will remain under the full influence of a basal narcotic for the whole duration of a major operation. If the effect of the drug wears off, any stimulus may produce inconvenient reflex movements, and, as the patient is in a non-co-operative state, a general anæsthesia will have to be superimposed. For example, the writer has induced full avertin narcosis in 400 consecutive goitre patients for partial thyroidectomy. After careful local infiltration, the operation was only completed in 48 cases (12 per cent) without recourse to nitrous oxide-oxygen. In this instance, the inhalation anæsthesia was no disadvantage, but if a purely local analgesia is essential, it is better to give the patient a full dose of some sedative (such as morphine-scopolamine) rather than to attempt basal narcosis.

Finally, it is useless for the anæsthetist to decide upon some purely local technique unless the surgeon is prepared to operate with gentleness, to avoid undue traction, to speak quietly, and generally to lend his sympathetic co-operation.

The matter may therefore be summarised by saying that, in this country local analgesia is frequently used alone for minor operations and in combination with a light general anæsthesia such as nitrous oxide-oxygen in major surgery, in order to diminish shock, to minimise capillary oozing, and to obtain muscular relaxation without recourse to lipid-soluble drugs.

## CHAPTER II

### DRUGS AND APPARATUS USED IN LOCAL ANALGESIA

VERY many drugs have been used to produce local analgesia, some of which will now be discussed.

#### COCAINE

has been employed since 1884, and is a derivative of the base ecgonine. The hydrochloride in fresh solution is the form generally used, since decomposition occurs on boiling. Cocaine is an extremely toxic drug, and its dangers are such that its use is now practically confined to the surface application to mucous membranes, e.g. of the nose, eye, and larynx. The one outstanding advantage of cocaine over its substitutes is its vasoconstrictive action, a property which renders unnecessary the addition of adrenalin.

#### NOVOCAINE,

the hydrochloride of para-aminobenzoyldiethyl-aminoethanol, is now used very extensively as a substitute for cocaine. Novocaine is known under a variety of other names such as *ethocaine* (B.P.), *planocaine*, and *kerocain*. In France the drug may be designated as *néocaine*, *syncaïne*, and *scurocaine*, while in America it is usually known as *procaine* (U.S.P.). Novocaine is also the analgesic principle in the preparations known as *spinocain*, *gravocaine*, *duracaine*, and *novutox*.

For infiltration and field blocking, novocaine is generally used as a freshly prepared 0.5 per cent solution in 0.9 per cent saline which may be boiled for five minutes. Since novocaine is a vasodilator, it is customary to add adrenalin solution, making a final dilution of about 1 in 400,000. In an average patient, not more than 300 cc. of this preparation should be injected at one time. For nerve blocking techniques, 2 per cent novocaine is frequently employed, in which event a dosage of 40 cc. should not, as a rule, be exceeded. It is important to remember that novocaine, in common with most of the other cocaine substitutes, is decomposed by alkalis, so that pure water unimixed with sodium bicarbonate must be used for the sterilisation of syringes, needles, etc.



## PERCAINE

(known as *nupercaine* in America) is not related chemically to cocaine, but is a quinoline derivative having the name butyl-oxyeinchoninic acid diethylethylendiamide hydrochloride. This drug was introduced in 1929 and has displaced novocaine to some extent. Percaine appears to be one of the most promising substitutes for cocaine as it is very effective for surface application (unlike novocaine), and has an extremely prolonged action, often exceeding three hours. For infiltration analgesia and field blocking the very low concentration of 1 in 2000 is adequate, adrenalin usually being added to counteract the vasodilatation. Percaine is also often used in a 1 in 1500 solution for spinal block (see page 35).

Among the more recently introduced local anæsthetics, *pantocain*, *neolhesin*, and *diothane* appear to show the most promise.

Amongst the many other cocaine substitutes which have had a more or less prolonged trial, but which are now seldom employed, are *acocaine*, *alypin*, *apothesine*, *borocaine*, *butyn*, *cucaine*, *holocaine*, *quinine* and *urea hydrochloride*, *stovaine* (*amylocaine*), *tropacocaine*, and *tulocaine*.

The apparatus necessary for the induction of local analgesia is simple. An ordinary 10 cc. Record type syringe is quite usable, but it is better to employ a special syringe fitted with an eccentric needle

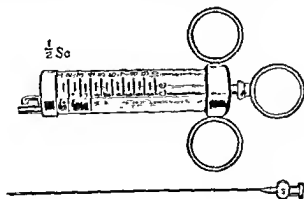


Fig. 19.—SYRINGE AND NEEDLE FOR LOCAL ANALGESIA. (LABAT'S.)

mount, a needle lock, and finger grips. The needles should be made of stainless steel, and must be sharp. A very fine short needle is necessary for the intradermal wheal, while a longer and stouter one is used for the deeper injections. For extensive infiltrations and field blocking, the continuous-flow syringes save a considerable amount of time as the upward motion of the piston automatically refills the



Fig. 22.—DIAGRAM TO ILLUSTRATE THE DEPOSITION OF SOLUTION IN INFILTRATION ANALGESIA.

they must also be infiltrated, the exact procedure being determined by the nature of the operation. The hæmatoma-injection method for setting fractures is a special form of infiltration analgesia and is considered elsewhere.

### FIELD BLOCK

This consists in creating walls of analgesia encircling the operative field. The solution is distributed in certain definite planes so that it will paralyse all the nerves crossing them, although no attempt is made to localise individual nerves.

Field blocking can be accomplished in three different ways :

(1) The creation of walls of analgesia perpendicular to the skin surface and involving the entire thickness of the tissues in which the nerves lie. These analgesic walls meet at their extremities and form a polygon described around the operative field. This technique was once described as "Hackenbruch's rhombus."

(2) The creation of walls of analgesia obliquely to the skin surface involving only a part of the tissues, but meeting below like a cup. This

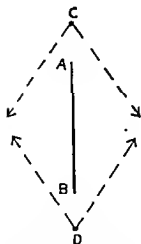


Fig. 23.—HACKENBRUCH'S RHOMBUS.  
LINE OF INCISION AB, WHEELS AT C  
AND D.

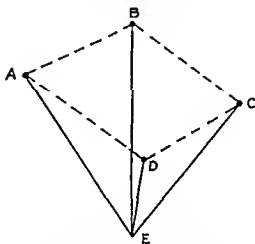


Fig. 24.—WHEELS AT ABCD. INJECTIONS MEET AT  
E ANALGESISING THE AREA SHOWN.

method is applicable to the excision of small superficial tumours or to the removal of foreign bodies which are fairly near the surface.

(3) The creation of a single wall of analgesia may be sufficient in certain areas of the body. For example, a wide area of insensitivity results from a field block along the costal margin, and this may suffice for such an operation as gastrostomy.

Further examples of field blocking are discussed in the next section of this book.

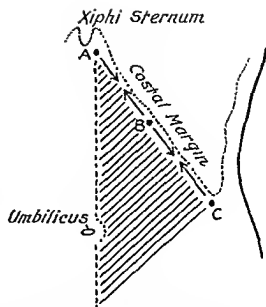


Fig. 23.—COSTAL MARGIN BLOCK. WHEELS AT ABC. ANÆSTHETISED AREA SHOWN SHADED.

### NERVE BLOCK

In certain areas of the body, it is possible to block the sensory nerves supplying the site of operation at some point remote from that site. This can be done in two ways:

*Intra-neural block.* The anæsthetic solution is here deposited within the nerve sheath. This method frequently leads to degeneration of the nerve, so that it is only employed when a permanent analgesia is required. Examples of this technique are the injection of the Gasserian ganglion for trigeminal neuralgia and the blocking of large nerves in limbs previous to their section during amputation. Alcohol is generally used for this purpose.

*Para-neural block.* In this case, the anæsthetic solution is deposited in such close proximity that it bathes the nerve but is outside its sheath. Examples of this method are splanchnic and spinal blocks both of which are considered later (see page 35).

### FREEZING ANALGESIA

If a fine spray of ethyl chloride is directed on to one area of skin a white, insensitive, frozen patch soon develops and a small incision can then be made relatively painlessly. The

method cannot be recommended as the subsequent thawing may be extremely painful and the vitality of the tissues may be impaired.

#### INTRAVENOUS AND INTRA-ARTERIAL LOCAL ANALGESIA

These methods are only applicable to operations upon the extremities, and are now rarely used since other methods give better results.

## CHAPTER IV

### SPINAL ANALGESIA

ATTEMPTS to block the nerve trunks of the cauda equina were made in 1896, but the method was not a practicable one until stovaine was employed in 1906.

#### ANATOMY AND PHYSIOLOGY

There are two ways in which several spinal nerves can be simultaneously blocked by one injection. In the first place, the anæsthetic fluid can be deposited in the peridural space by means of an injection either through the sacral hiatus (caudal block), or through a puncture to one side of the interspinous ligament just piercing the ligamentum flavum (extra-dural spinal block). Secondly, the solution can be injected into the cerebro-spinal fluid (subarachnoid spinal block). The latter method is more certain in its effects, and is the one usually known as spinal analgesia. The solution is introduced at a level no higher than the space between the second and third lumbar vertebrae in order to avoid any possibility of damaging the lower end of the spinal cord.

*Solutions used.* Since the specific gravity of cerebro-spinal fluid varies in health between 1.004 and 1.010, it is impossible to employ an exactly isoharic solution. Until recently a small volume of a relatively concentrated solution of novocaine, stovaine, or tropacocaine was commonly used, since these drugs are ineffective in low concentrations, and the solutions were invariably hyperbaric. The main disadvantage of this method is the inconvenient spread of the fluid towards the head by gravitational diffusion if a Trendelenburg position is necessary. The effective duration of such an analgesia rarely exceeds one hour. Novocaine-alcohol solutions (such as spinocaine and light duracaine) were introduced in order to limit the spread, and although these preparations are undoubtedly hypobaric in vitro, it is now widely thought that, after injection, the alcohol rapidly diffuses into the cerebro-spinal fluid, leaving the heavier constituents to spread as before by gravitational diffusion. The introduction of percaine in 1929 profoundly

modified the technique of spinal analgesia, as this drug acts in such extremely low concentrations that it is now possible to use a true hypobaric solution without the addition of alcohol. The usual preparation is 1 in 1500 percaine in 0.5 per cent saline, which has a specific gravity of 1.00345 at 15° C.

With this technique, the subarachnoid space is treated in exactly the same way as the tissues in an infiltration analgesia, a relatively large volume of dilute solution being injected, the height of analgesia being regulated by the amount of solution used. It is now generally recognised that this method gives considerably better control than the older techniques, and possesses the additional advantage of securing a longer analgesia, often exceeding three hours. The "hypobaric percaine" method elaborated by Howard Jones will, therefore, be the one subsequently described.

*Indications.* The view held at present is that spinal analgesia can be usefully employed for extensive operations below the level of the diaphragm involving much shock, particularly in patients suffering from, or liable to, pulmonary diseases.

*Contra-indications.* Spinal analgesia should not, as a rule, be employed for patients who have abnormally high or low blood-pressure, or who have suffered from any disease of the central nervous system. High spinal blocks should be avoided if the patient has any form of respiratory obstruction or other interference with normal respiration such as a recent phrenicotomy, thoracoplasty, or artificial pneumothorax. Finally, the method should never be employed if there is sepsis in the region of the proposed lumbar puncture.

*Technique.* About 45 minutes before operation, the patient is given a suitable dose of morphine and scopolamine, his eyes are lightly bandaged, and his ears plugged with wool. Upon arrival in the anaesthetic room, the lateral position is adopted and the knees are drawn up towards the chin. The skin having been sterilised and the surface markings defined, a lumbar puncture is performed, usually between L.2 and L.3. The best type of needle is one of stainless steel 8.5 cm. long, 1.2 mm. in diameter, with a 45° bevel, and provided with a stylet. Having obtained a flow of clear cerebro-spinal fluid, a 20 cc. Record syringe containing the percaine solution is connected to the needle. The fluid should be at body temperature and is injected

slowly. The dosage will depend on the desired height of analgesia and the build of the patient. High abdominal operations necessitating a block up to D.4 or 5 will require from 14 to 16 cc., while an analgesia

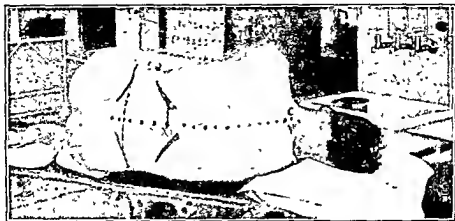


Fig. 26.—POSITION OF PATIENT FOR LUMBAR PUNCTURE (by permission of the late Dr. Howard Jones).

extending as far as the umbilicus is usually attained with 10 to 12 cc. Simple caudal block can be made by injecting 6 cc. between L.4 and L.5.

As soon as the injection has been finished, the patient is turned over on to his face and the table or trolley given a slight head-down



Fig. 27.—SPINAL NEEDLE AND STYLET. (HOWARD JONES.)

tilt. This position is maintained for about five minutes in order to be certain that a complete posterior root block will develop. After this interval the patient may be placed in any convenient position for the operation, provided that a slight Trendelenburg slope is maintained.

Ephedrine  $1\frac{1}{2}$  gr. or coramine 1 cc. may be injected intramuscularly with the idea of minimising the inevitable fall in blood-pressure, but more reliance should be placed upon position than upon drugs. It now appears to be certain that the blood-pressure drop is due to two factors, viz.: vasomotor paralysis and absorption of the analgesic drug into the blood stream.

Respiration is rarely embarrassed, but occasionally becomes unduly shallow or slow. This is unlikely to be due to an upward spread of the block causing phrenic paralysis, but is probably the result of fatigue of the respiratory centre from continued slight anoxæmia. The judicious

administration of carbon dioxide will almost always restore full respiratory action.

Patients do not, as a rule, complain of any pain during an operation performed under percaine spinal analgesia, but discomfort may arise from a variety of causes. The hot theatre and unusual position may induce a feeling of suffocation and dryness. These may be relieved by sucking ice or sponging the patient's face, or directing a gentle air blast towards his head. Nausea and even vomiting may be caused by powerful traction on the viscera immediately below the diaphragm. In difficult operations it may be necessary to administer nitrous oxide and oxygen to avoid this discomfort.

#### SEQUELÆ

*Headache* is unfortunately fairly common after all types of spinal analgesia, and, indeed, after simple lumbar puncture. Prophylactic measures include the use of a fine needle, the rigid adherence to a head-down position for some hours after operation, a semi-darkened room, and the prohibition of reading for at least 24 hours. Mild cases yield to the usual treatment, but occasionally it is necessary to resort to the injection of hypertonic rectal or intravenous solutions.

Transient *paralyses* are not uncommon, particularly of the ocular muscles, but permanent effects are fortunately extremely rare, although not unknown.

*Vomiting* is rare, but may occasionally prove troublesome. It should be treated on the usual lines.



## SECTION 3

### CHOICE OF ANÆSTHETIC

#### CHAPTER I

##### GENERAL CONSIDERATIONS

THE ability to choose and to carry out the best anæsthetic technique for each individual patient and operation distinguishes the true anæsthetist from the mere casual administrator of anæsthetics. Considerable experience is required to assess not only the patient's general condition and the particular factors likely to arise from the operative procedure, but also the mentality of the patient.

As a general rule it can be said that the least toxic drug or combination of drugs should be used which will provide adequate anæsthesia for the particular operation which is proposed. The very large number of possible combinations of basal narcotics, and general and local anæsthetics render it essential that the anæsthetist should have experience of an adequate variety of methods. For example, it is useless to decide that a combination of field blocking with nitrous oxide and oxygen will give the best results in a particular case if the anæsthetist is only familiar with the administration of open ether. It is, in fact, better to give a good ether anæsthesia than an indifferent local block with a poor gas and oxygen technique.

Disparaging remarks are still occasionally heard about complicated apparatus and the ease with which the old "rag and bottle" methods can be used. Such arguments are, of course, quite futile, and, if acted upon, would render progress impossible. The most conservative general practitioner employs for his daily rounds a car which is an agglomeration of machinery far more complicated than any gas and oxygen apparatus. It is true that some people cannot understand or even drive a car, and it is usually impossible for the general practitioner to keep abreast of the latest developments in anæsthesia or, for that matter, in many other branches of medicine and surgery. It is for this

reason that the specialist is becoming more and more essential if the best possible results are to be obtained.

In the following chapters an attempt has been made to indicate desirable methods for the anæsthesia necessary for some of the surgical proceedings described in other parts of this book. Many readers will doubtless disagree profoundly in certain cases, and may well have obtained excellent results with quite different techniques. The writer's reply to these critics is merely that he has used the methods recommended, and that in his experience they do give better all round results than others which he has tried. Anæsthesia has not, however, yet become an exact science, and there is no doubt that various differing techniques, when carried out efficiently by those proficient in their use, yield results which are not very dissimilar.<sup>1</sup>

<sup>1</sup> Should the reader require fuller details of technique or references to original papers, he is referred to *Recent Advances in Anæsthesia and Analgesia*, J. and A. Churchill, London.

## CHAPTER 'II

### CHOICE OF ANÆSTHETIC FOR CRANIAL SURGERY

CRANIAL surgery has altered considerably during the past thirty years. When the late Sir Victor Horsley first made craniotomy a relatively safe operation in this country, a duration of half an hour was rarely exceeded, and chloroform was a perfectly satisfactory anæsthetic. This "rapid" technique was developed by the late Sir Percy Sargent and others who extended the scope of cranial surgery considerably, and although extensive tumours were removed, the time of operation was usually under  $1\frac{1}{2}$  hours. Ether, generally administered by the endotracheal route, gave excellent results in such cases. More recently, however, the "slow" method, in which Dr. Harvey Cushing has taken such a prominent part, has, to a certain extent, come into vogue. It is not for the anæsthetist to say whether the meticulous care expended avoids more shock than the immense time entails, but this method has profoundly altered the anæsthetic technique. It is not permissible to use lipoid-soluble drugs such as chloroform or ether for periods of the order of five hours, as severe toxic symptoms may ensue. The other difficulties which confront the anæsthetist are the impossibility of approaching the face when the operation has started, and the patient's position, which may be extremely awkward. It is also essential that the blood-pressure should not be raised nor must the brain be engorged.

Local analgesia alone is useful when the patient is comatose, and for comparatively short operations such as the repair of scalp lacerations, sub-temporal decompression, etc. Care must be taken over pre-medication. Morphia in the form of omnopon is usually satisfactory, but it must be ascertained that the patient reacts normally to this drug, as certain people vomit frequently after its administration, a circumstance which may prove disastrous if it occurs during the operation. Morphia is also inadvisable if there is any evidence of respiratory failure from cerebral compression. The same remark applies to avertin and all other drugs which are respiratory depressants.

The technique of local analgesia for cerebral operations is simple. A polygon is described around the proposed field by means of wheals

## CHAPTER III

### CHOICE OF ANÆSTHETIC FOR SURGERY OF THE NECK

OPERATIONS upon the neck can, for anæsthetic purposes, be divided into three groups.

#### MINOR OPERATIONS

Into this category fall such proceedings as the aspiration or drainage of abscesses, the excision of single superficial glands, phrenicotomy, etc. Suitable premedication followed by the inhalation of nitrous oxide and oxygen usually provides satisfactory anæsthesia without undesirable sequelæ. If desired, local analgesia can be employed provided that the area is not septic; for example, phrenicotomy can be conveniently performed under a simple infiltration analgesia. If a short anæsthesia is required for operations in which a face-piece would be in the surgeon's way, e.g. for the insertion of radium needles near the mandible, intravenous sodium evipan may be useful.

#### MAJOR OPERATIONS

It is proposed to include under this heading all long cervical operations except those for the relief of toxic goitre. Typical examples coming into this group are lateral pharyngotomy, excision of pharyngeal pouches, removal of thyroid adenomata and carcinomata, block dissection of glands, etc. In practically all these operations some respiratory obstruction may occur either from the nature of the disease, the position of the patient, the operative manipulations, or the effusion of fluids into the air-passages.

The actual anæsthetic technique must be adapted to the individual case, but the following details are worthy of attention. Premedication should include a large dose of atropine, and the patient should be placed upon the operating table in the position in which he breathes most comfortably. For example, if a large retro-sternal adenoma of the thyroid is present, the patient's head may have to be kept in the

flexed position. The nose and glottis are next cocainised, and anæsthesia is induced with nitrous oxide and oxygen. A rubber tube is then passed into the trachea either through the nose or mouth and anæsthesia is maintained by the inhalation endotracheal technique. It is essential that the distal end of the tube should lie beyond the region at which obstruction may occur. When this point has been ascertained, wet gauze is packed around the tube above the larynx and the operation may be started. Some surgeons prefer to use an infiltration analgesia in addition, with the object of avoiding shock and of minimising capillary oozing.

The excision of extensive malignant growths of the pharynx and larynx may necessitate preliminary laryngotomy or tracheotomy, the former procedure being preferable if the site of operation permits. In any event, anæsthesia can be conducted upon the inhalation endotracheal principle by using an adapter which fits on to the laryngotomy or tracheotomy tube. If diathermy is used in the course of the operation, it is safer to employ no ether whatever although the expiratory valve may be some distance away from the electrode.

#### OPERATIONS FOR TOXIC GOITRE

Patients suffering from primary or secondary toxic goitre exhibit symptoms which are of great importance to the anæsthetist. In the first place, such patients are, as a rule, extremely nervous and apprehensive, and have a raised basal metabolic rate. Again, cardiac disturbances are common, tachycardia and auricular fibrillation with partial decompensation being, perhaps, the most frequent varieties. The operation itself also presents peculiar problems, two of which have a bearing upon the method of anæsthesia: firstly the area is a highly vascular one, and secondly the necessary baring of the trachea may lead to post-operative tracheitis.

The writer is of the opinion that the best anæsthetic technique for these cases is the avertin-local-nitrous oxide and oxygen combination. Pre-operative fear is largely abolished by means of the basal narcotic, capillary oozing is minimised by the local infiltration, a higher percentage of oxygen than usual can be used owing to the avertin so that no further cardiac embarrassment is caused, while the absence of an endotracheal tube and the avoidance of ether causes no aggravation to any inevitable tracheitis.

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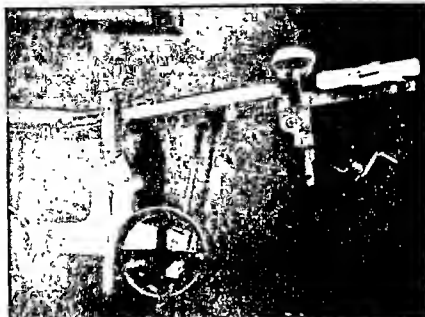


Fig 23.—OPERATING TABLE (KNY SCHEERER) ARRANGED FOR THYROIDECTOMY, SHOWING STRAPS FOR SECURING WRISTS.

is generally agreed that iodine should be administered, usually in the form of Lugol's solution, until the maximal improvement is obtained. For some days before operation every effort should be made to increase the patient's store of carbohydrate. If the patient's nervousness is extreme, it may be deemed necessary to avoid any mention of operation, and a simple saline enema is given for two or three days beforehand. About fifty minutes before the time fixed, an avertin enema is given in exactly the same way as the previous saline, and directly the patient is asleep the eyes are lightly bandaged, the ears plugged, and a hypodermic



Fig 24.—GOITRE FACE-PIECE WITH RETAINER HOOKS (MAGILL'S).

injection of atropine combined with a *small* dose of morphia is given. The dosage of avertin will depend not only on the patient's weight, but also on the basal metabolic rate. As a rule, not less than 0.1 grammes per kilo. body weight should be used, and if the B.M.R. be considerably raised, 0.11 grammes can be used with advantage. The patient is then placed upon the operating table with the head extended and the wrists



and legs secured against unexpected movements. The face-piece is next fixed in position by Clausen's retainer, great care being taken to see that the eyes are closed and properly lubricated. In cases of extreme exophthalmos it may be necessary to strap the lids together. A local infiltration is then made, using 0.5 per cent novocaine and 1 in 400,000 adrenalin. The greatest care must be exercised to avoid intravenous injection as several fatalities have occurred from this misadventure in thyroid cases. The safest method is to keep the needle point continually on the move and to inject during withdrawal and not during entry. The operation is then started, and if at any time the patient begins to make reflex movements, the hose of a gas-oxygen apparatus is connected to the face-piece and an inhalation anæsthesia is induced. No cyanosis should be permitted at any stage as this almost always leads to a deterioration of the pulse. The anæsthetist must always be in a position to advise the surgeon if it is desirable to perform the operation in two or more stages. In extremely toxic patients it may even be inadvisable to start with more than the ligature of one thyroid artery.

a real space with collapse of the lung and dyspnoea. The mediastinum normally has little lateral stability and consequently moves towards the unopened side, tending to collapse that lung also. The sudden change in the relationship of the thoracic viscera is always accompanied by a certain amount of shock. If the pleural cavity which is opened contains fluid, the resulting disturbance will depend upon the amount of effusion present. For example, if the whole cavity is full of fluid under pressure, changes exactly the reverse to those described will occur, i.e. as the fluid will escape, the intra-pleural pressure will fall, and the mediastinum will move *towards* the affected side.

The undesirable effects of sudden pressure changes (mainly shock and dyspnoea) can be minimised in three ways. (1) By opening the pleural cavity slowly, e.g. by preliminary drainage of an acute empyema through a large-bore needle. (2) By the use of positive pressure anaesthesia, and (3) By inducing an artificial pneumothorax prior to operation. The use of one or more of these measures will depend upon the type of operation. In this group a great variety of procedures occurs from a small rib excision to an extensive thoracotomy for the removal of growths.

The excision of a portion of one rib for the relief of acute pneumococcal empyema can often be conveniently performed under local

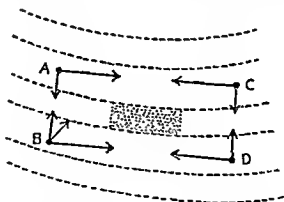


Fig. 32.—LOCAL ANALGESIA FOR RIB RESECTION.  
PORTION OF RIB FOR REMOVAL SHOWN SHADED.

analgesia in adults. Four wheals are raised, ABCD, two in the intercostal space above, and two in the space below the desired rib. An infiltration of the subcutaneous tissues and muscles is made joining these wheals. Finally from the lower posterior wheal B, a deep injection is made under the lower border of the affected rib in order to block the intercostal nerve in its groove. The needle must not be advanced deeper than 0.5 cm. from the posterior surface of the rib, and the

aspiration test must be negative before 2 cc. of novocaine solution are injected. Streptococcal empyemata are better dealt with under general anaesthesia, as extensive sloughing sometimes follows local blocks.

If it is decided to employ a general anaesthetic, the same technique as that described for the previous group of operations can be followed except that a slight amount of positive pressure is usually beneficial when the pleural cavity is open. Intermittent-flow gas-oxygen machines are particularly useful for this type of work as the pressure can be altered without altering the percentages of the gases. Only just sufficient pressure to avoid dyspnoea should be used, as any excess may embarrass the surgeon by pushing the thoracic viscera into the wound.

If the patient is coughing up large quantities of sputum, the combined endotracheal and suction technique introduced by Magill may be adopted. A wide-bore rubber tube is passed into the trachea through the nose, and a small coude gum elastic catheter is then passed in front of it by direct laryngoscopy until its distal end lies near the bifurcation of the trachea. Gauze is then packed around both tubes and anaesthesia is maintained upon the inhalation endotracheal principle with nitrous oxide and oxygen. Any desired degree of positive pressure can be obtained by adjusting the tension of the expiratory valve, and the trachea can be kept free from fluid by connecting the catheter to a suction pump from time to time. When suction is not being used, the proximal end of the catheter is closed by a spigot in order to avoid any air leak.

An alternative and theoretically superior method of dealing with excessive secretion is intra-bronchial anaesthesia. In this case, the affected lung is cut off entirely so that no blood or pus can be aspirated into the sound side. A long intubation tube fitted with an inflatable rubber cuff is used, the curved distal end being passed into the desired

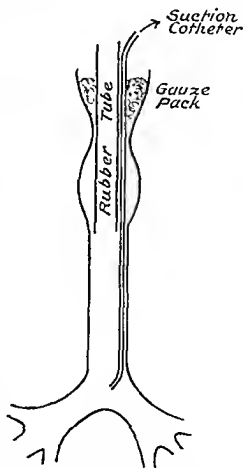


Fig. 33.—DIAGRAM TO ILLUSTRATE THE METHOD OF COMBINING SUCTION WITH ENDOTRACHEAL ANÆSTHESIA.

bronchus. The pneumatic cuff should then lie at the bifurcation of the trachea, and on being inflated with air will seal off the opposite side so that anaesthesia is maintained entirely by the sound lung. There are, however, various technical difficulties connected with this method and, in the opinion of the writer, the technique described previously will be found quite satisfactory.

If a large bronchial fistula is present, it will be necessary to plug the opening; otherwise the air leakage will prevent a satisfactory nitrous oxide-oxygen from being administered.

#### OPERATIONS INVOLVING THE OPENING OF BOTH PLEURAL CAVITIES

In certain severe thoracotomy operations such as those for the relief of diaphragmatic hernia or thoracic stomach, the simultaneous opening of both sides of the chest may be unavoidable. When this occurs in a normal patient (e.g. in gunshot wounds) the most violent respiratory movements fail to draw sufficient air into the lungs, and death from asphyxia inevitably occurs. It follows, therefore, that differential pressure must be employed, and the most convenient ways of doing so are by means of a tightly fitting face-piece or an intubation tube with gauze pack. It is possible to use continuous-flow gas-oxygen machines for this purpose, but the intermittent-flow types offer easier control. It cannot be too strongly urged that no operation which may involve the simultaneous opening of both pleural cavities should ever be performed unless the anaesthetist is prepared to institute and to maintain adequate positive pressure as required.

Diathermy is now extensively used in thoracic surgery, both for minimising haemorrhage from the thoracic wall and also for the division of lung tissue. If this type of current is employed, the use of ethylene, cyclopropane, ether, and ethyl chloride is entirely contra-indicated, and the choice of basal narcosis, nitrous oxide-oxygen, chloroform, local analgesia, or combinations of these must be made. As already pointed out, however, practically any thoracic operation can be successfully carried out with pure nitrous oxide and oxygen.

## CHAPTER V

### CHOICE OF ANÆSTHETIC FOR ABDOMINAL AND PERINEAL SURGERY

THE various types of operation upon the abdomen vary so much in the anæsthetic problems which they present that it is necessary to consider them in separate groups.

#### MINOR OPERATIONS UPON THE ABDOMINAL WALL

Various common procedures fall into this group, including the repair of inguinal, femoral, and umbilical herniæ, operations for the cure of varicocèles, hydrocèles, etc. Since complete muscular relaxation is not essential, a nitrous oxide-oxygen anæsthesia preceded by adequate premedication is usually quite satisfactory, and the patient should suffer from little, if any, post-anæsthetic discomfort. In the author's experience, quite severe chronic intercurrent diseases are not contra-indications to this method provided that the operation itself is thought necessary. If local analgesia is deemed advisable, however, a combination of infiltration and field blocking can usually be performed.

For example, a reducible right inguinal hernia can be dealt with by the method shown in the subjoined diagram. A wheal A is raised three finger-breadths internal to the anterior superior iliac spine, and solution is distributed in the directions shown both subcutaneously and between the muscle layers, particular care being taken to reach the iliac bone so that the ilio-inguinal and ilio-hypogastric nerves are blocked. A second wheal B is then made over the pubic spine and through this injections are made, firstly under the

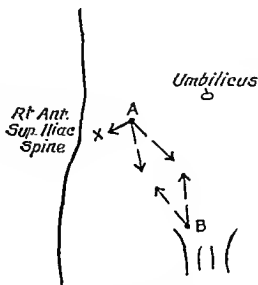


Fig. 34.—BLOCK FOR REDUCIBLE RIGHT INGUINAL HERNIA.

skin to meet the former infiltration in the form of a rhombus, secondly beneath the aponeurosis in the line of the inguinal canal, and finally fanwise over the pubis, coming into contact with the bone.

#### LOWER ABDOMINAL AND PELVIC OPERATIONS

Comparatively short operations such as appendicectomy, removal of ovarian cysts, sub-total hysterectomy, etc., can be conveniently dealt with under nitrous oxide-oxygen anaesthesia with the addition of minimal ether.

In cases associated with grave toxæmia, e.g. appendicitis with general peritonitis or suprapubic cystotomy for long-standing urinary retention, it is better to avoid lipoid-soluble drugs altogether and to use pure nitrous oxide-oxygen with the addition of local infiltration if necessary.

Prolonged operations which may be followed by shock, such as abdomino-perineal excision of the rectum or pan-hysterectomy, frequently do extremely well under spinal block up to D.8 or D.9, preferably using percaine (see page 35).

Prostatectomy is also frequently performed under spinal analgesia, but some surgeons dislike it on account of the tendency towards reactionary hæmorrhage when the blood-pressure subsequently rises. The writer considers that nitrous oxide-oxygen is a better technique in most cases.

Operations for the repair of very large herniæ may take an extremely long time, especially when fascial grafts are used. Furthermore, these patients are frequently the subjects of chronic bronchitis, so that a prolonged inhalation anaesthesia is undesirable. Infiltration and field block analgesia are difficult to perform and not uniform in their effects, so that in the writer's opinion a percaine spinal block frequently represents the best technique for this type of operation.

#### HIGH ABDOMINAL OPERATIONS

The shortest and simplest operation commonly performed in the upper abdomen is that of gastrostomy. This proceeding is usually carried out upon patients who are wasted and in poor condition from prolonged starvation, so that a satisfactory anaesthesia is generally obtained with nitrous oxide-oxygen. If, however, a local analgesia is thought to be preferable, the simple costal margin block described on page 33 can be employed.

We now have to consider such major operations as gastrectomy, gastro-enterostomy, cholecystectomy, etc., and these present various problems for the anæsthetist.

In the first place, the wound is made in that part of the abdomen where respiratory movement is at its greatest, so that the anæsthetic should not produce any increase in excursion which would make suturing, etc., difficult.

Secondly, in order to obtain the best exposure, the abdominal wall must be completely relaxed. This, in itself, is comparatively easy, but severe traction on the diaphragmatic attachments is very apt to initiate reflex laryngeal spasm which is incompatible with complete muscular relaxation.

Lastly, the anæsthetist has to consider the fact that major operations upon the upper abdomen may give rise to shock, and are more likely than others to be followed by pulmonary complications.

It will be seen, therefore, that *the ideal anæsthetic for major high abdominal surgery* should: (1) give rise to minimal respiratory movements; (2) afford complete muscular relaxation with protection from reflex laryngeal spasm; (3) give protection from operative shock; and (4) must not increase the liability to pulmonary complications.

The methods which appear to fulfil these somewhat formidable conditions best are:

(1) Endotracheal nitrous oxide-oxygen-ether with subsequent hyperventilation with carbon dioxide-oxygen. This technique has been described in a previous section.

(2) "High" spinal block with percaine, a procedure which has also been described previously.

(3) Field block of the abdominal wall combined with splanchnic block. The best field blocking for extensive explorations is the costo-iliac block illustrated. Wheals are raised at ABCD and solution is injected subcutaneously joining these points and also fanwise into the muscle layers. If the incision is to be appreciably to the right of the mid-line, the block is

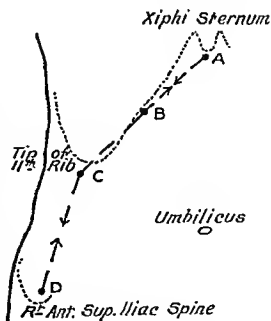


Fig. 35.—RIGHT COSTO-ILIAIC BLOCK.

completed by another wall of analgesia starting at A and extending along the mid-line to below the umbilicus. If, on the other hand, the incision is to be practically central, a bilateral costo-iliac block will be necessary. A separate splanchnic block can be obtained in two ways. Firstly, the abdomen is opened under the field block just described, the hand is then introduced and the aorta gently retracted with the finger. Novocaine solution is then deposited in close contact with the lateral aspects of the body of the first lumbar vertebra. This method is known as Braun's anterior technique, and is frequently used on the Continent. The second method (Kappis') uses the posterior method of approach. Before the abdominal wall blocking is carried out, the patient is placed on his side and a point 7 cm. external to the spinous process of the first lumbar vertebra is identified.

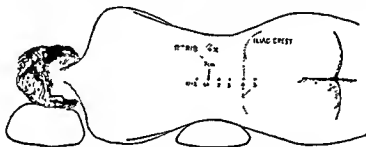


Fig. 36.—PATIENT IN POSITION FOR RIGHT SPLANCHNIC INJECTION.  
X IS SITE OF PUNCTURE.

This point should lie immediately below the 12th rib, and through it a 12 cm. needle is introduced at an angle of  $45^\circ$  to the median plane. It should be possible to feel the point strike against the side of the vertebral body. The needle is then partially withdrawn and re-introduced in a slightly more forward direction until its point is felt to slide tangentially past the bone. It is then pushed 1 cm. farther in and, the aspiration test being negative, 20–30 cc. of 1 per cent novocaine solution are injected. The patient is then turned over on his other side and the same process repeated.

(4) Combined paravertebral and splanchnic blocks. A bilateral paravertebral block from D.7 to D.12 can be substituted for the anterior field block if desired. The patient is placed on his side and a series of wheals are raised 4 cm. from the mid-line opposite the required spinous processes. Through these wheals an 8 cm. needle is introduced perpendicularly to the skin surface until its point is felt to impinge upon the rib. It is then slightly withdrawn and re-introduced downwards and inwards towards the lower border of the rib. The needle



is finally pushed 2 cm. past this point and, if the aspiratioo test is negative, 5-6 cc. of 1 per cent novocaine solution are injected. When all the iojections are completed, the splanchnic block is proceeded with as before.

The anæsthetist will have to decide upon his choice of method in each individual case. The last two techniques described are not used very extensively in this couotry owing to the length of time required, the large amounts of solution necessary, and the uncertainty that a perfect analgesia will develop in spite of the greatest care and technical skill in making the multiple injections. On the other hand, there is now no question as to the definite value of splanebnie block in the prevention of shock due to visceral stimulation. Attempts have been made to obtain similar results by the wholesale introduction of novocaioe solution into the peritoneal cavity, but until recently the risk of subsequent intestinal paralysis and toxic effects from absorbed novocaine has been judged excessive. It has now been stated, however, that 200 cc. of 0.25 per cent novocaine can be used safely provided that it is only left in for 10 minutes and the excess fluid then removed. If the second or "high spinal" technique be adopted, a splaochnic block is automatically included since all the splanchnic nerves are given off helow D.4. The single injection with about 15 cc. of solution provides a complete hlock of praetically every nerve supplying the outside and ioside of the abdomen with the exception of the phrenic nerves and the gastric portioos of the vagi. The results obtained with this metbod using percaine (previously described) are, on the whole, very reliable, but there is little doubt that, in spite of statements to the contrary, the immediate aoæstbetic mortality is higher than if an inhalation technique is used. The author's practice is, therefore, to use the first (endotracheal) method for most major operatioos upon the upper ahdomen unless the patient is suffering from some chronic pulmonary disease or unless the surgeon considers that considerable visceral shock is likely to develop in the course of the operation. In

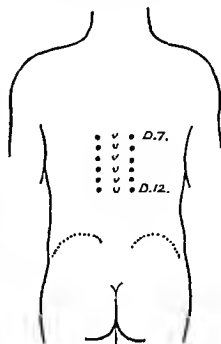


FIG. 37.—BILATERAL PARAVERTEBRAL BLOCK, SHOWING POSITION OF WHEELS.

these events a high spinal analgesia is used unless otherwise contra-indicated, nitrous oxide and oxygen being added if desirable.

#### OPERATIONS FOR THE RELIEF OF PERFORATED PEPTIC ULCER

The question of basal narcosis does not usually arise in these cases as the patient is only too anxious to be anaesthetised in order that his pain may cease.

Although spinal analgesia has been recommended for these operations, particularly in America, the low blood-pressure which is usually present in such patients is a definite contra-indication, and in the writer's opinion better results are obtained with general anaesthesia.

Nitrous oxide and oxygen with minimal ether is generally quite satisfactory, the endotracheal method being employed if there is any reason to anticipate difficulty with the airway. If the surgeon is desirous of performing a gastro-enterostomy after suturing the perforation, the anaesthetist must be in a position to inform him whether such a prolongation of the operation is justifiable.

#### OPERATIONS FOR THE RELIEF OF ACUTE INTESTINAL OBSTRUCTION

These operations always cause anxiety to the anaesthetist. Spinal block has, in the past, been freely advocated for such emergencies. This practice has several grave disadvantages. In the first place, the sympathetic paralysis and unopposed vagal action always results in increased peristalsis. This may be beneficial in cases of paralytic ileus, and may even result in the spontaneous reduction of strangulated herniae, but if real mechanical obstruction is present, rupture of the distended proximal bowel may be precipitated. Again, supposing that the obstruction has been overcome by the increased peristalsis, a massive action of the bowels may occur with great fall of blood-pressure, collapse, and in extreme cases death. Lastly, a high spinal block so weakens the effective coughing mechanism that the patient may inhale his own vomit with resulting immediate asphyxia or subsequent septic broncho-pneumonia.

The only certain way of preventing the aspiration of infected material in a patient who is continually regurgitating it, is the endotracheal technique with gauze packing (see page 16), or with a special tube fitted with an inflatable rubber cuff. A suction pump should be ready to hand in case vomiting occurs during the induction period. When anaesthesia is progressing smoothly, a stomach tube should be passed and maintained in position throughout the operation.

## ABDOMINAL OPERATIONS IN INFANTS

The commonest abdominal emergencies which render operative interference necessary in infants are pyloric stenosis and intussusception. From observations made at a large children's hospital, it was found that the best results were obtained if local and spinal blocks were avoided and a general anæsthetic was used. Nitrous oxide-oxygen is difficult to administer satisfactorily to very young babies, and excellent results are obtained with an open ethyl chloride induction followed by warm ether vapour. The essential points to observe are, firstly, that the child is kept as warm as possible before, during, and after operation, and, secondly, not to begin the administration of the anæsthetic until the surgeon is quite ready to begin, with sutures threaded, etc. If due recognition is paid to these two factors of heat and speed, and if the supply of fluids is kept up by every available route, the mortality of these operations is gratifyingly low.

## PERINEAL OPERATIONS

Most operations upon the perineal region can be performed very satisfactorily under gas-oxygen anæsthesia with the addition of minimal ether. If the sphincter ani is suddenly stretched, temporary laryngeal spasm may occur, but this is rarely troublesome.

Should a local analgesia be considered desirable, a variety of methods can be used. A local infiltration around the anus causes immediate relaxation of the sphincter and allows minor operations to be performed, e.g. hæmorrhoidectomy, incision of fistulæ, etc. It is generally held, however, that there is a distinct risk of infecting healthy tissues, and it is preferable to employ a nerve block some distance away from the site of operation. Extra-dural sacral (or caudal) block is sometimes useful in such cases. An 8 cm. needle is introduced at an angle of  $20^{\circ}$  to the plane of the back between the sacral cornua, and, having pierced the tough ligament, is directed horizontally and parallel to the back until 5 cm. of its length lie in the sacral canal. If the aspiration

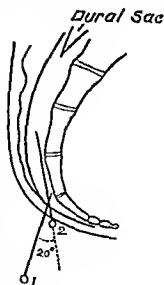


Fig. 38.—DIAGRAM SHOWING INITIAL AND FINAL POSITIONS OF NEEDLE IN SACRAL BLOCK. (1 AND 2.)

test is negative to blood and cerebro-spinal fluid, 30 cc. of 2 per cent novocaine are injected slowly. For extensive operations which may cause shock, such as perineal excision of the rectum, vaginal hysterectomy, etc., a "low" spinal block may be useful. The lithotomy position which is frequently used for such procedures is advantageous in that it minimises the fall of blood-pressure. An alternative to low spinal analgesia is trans-sacral block, but this is rarely used now in this country owing to the multiple injections necessary and the variable results obtained.

PART II  
ABDOMEN

SECTION 1

INVESTIGATION OF A CASE OF DYSPEPSIA

INTRODUCTION

by  
RODNEY MAINGOT

CHAPTER I

History of the Case

by  
RODNEY MAINGOT

CHAPTER II

Examination of the Stomach with Special Instruments

by  
RODNEY MAINGOT

CHAPTER III

Physical Examination of the Patient

by  
R. SLEIGH JOHNSON

CHAPTER IV

Biochemical Investigations

by  
P. M. DEVILLE AND R. SLEIGH JOHNSON

## SECTION I

# THE INVESTIGATION OF A CASE OF DYSPEPSIA

## INTRODUCTION

by

RODNEY MAINGOT

THE methods of investigation may be classified as follows :

- (1) History (anamnesis).
- (2) Physical examination.
- (3) Radiological examination.
- (4) Laboratory investigations.
  - (a) Gastric analysis.
  - (b) Tests for occult blood.
  - (c) Examination of urine and tests for renal efficiency.
  - (d) Wassermann reaction and Kahn test for syphilis.
  - (e) Complete blood count.
  - (f) Duodenal intubation.
- (5) Investigation by means of instruments.
  - (a) Gastroscope (gastroscopy).
  - (b) Gastro-photor (gastro-photography).
- (6) Exploratory laparotomy.

It is very important that the history of these cases should be taken with meticulous care and patience, that the investigations should be as thorough and complete as possible, that the methods of treatment, whether they be medical or surgical, should be accurately recorded and strictly investigated by a "follow-through" system, and that the final results of such treatment should be assessed and impartially reviewed after a period of many years.

There does not seem at the present moment to be any generally adopted system or scheme for the investigation of the large number of

cases of chronic dyspepsia that come to hospital or to the private practitioner for advice. A perusal of numerous notes or records of such cases reveals woeful deficiency regarding just those points that are particularly required for accurate statistical purposes. Nevertheless, it must be admitted that no scheme or special form for dyspepsia cases can ever be complete in every detail, or meet with universal acceptance, as dyspepsia, although a pre-eminent symptom in gastro-intestinal disorders, may be a symptom of any general disease. Therefore, an investigation which does not include the routine examination of all the systems of the body can never adequately cope with the complexity of the subject in its fullest sense. An accurately recorded form which includes the history of the case, the clinical findings, the results of investigations, the details of treatment, and which sets out clearly the particulars of a "follow-through" inquiry, will be of worth, not only in training the investigator to adopt systematic methods, but in providing a complete and comprehensive synopsis of all the relevant matter pertaining to each case. Such a record is easily filed, and is at once handy for ready reference and for statistical purposes.

The form here given may be found useful. For the sake of compression the space for written entry has been omitted.

#### FORM FOR DYSPEPSIA CASES

Disease (to be filled in on discharge).		Index No.	
		Date of Examination.	
Name (in block letters).	Age.	Sex.	Married.
			Single.
			Widow.
			Children.
Address.			
Address of next of kin.			
Name and Address of Doctor.			
Occupation.			

#### (A) HISTORY

- (1) *Complaints.*
- (2) *Family history.*
- (3) *Social and personal state.*

- VOL. I.—D



- (iii) amount.
  - 1. small.
  - 2. large.
- (iv) relation to food.
- (v) relation to onset of pain.
- (vi) self-induced.
- (vii) relief of pain.
- (d) Regurgitation ; flatulence ; belching ; sense of fulness ; heartburn.
- (e) Weight.
  - (i) loss.
    - 1. rapid.
    - 2. gradual.
  - (ii) stationary.
  - (iii) gaining.
- (f) Appetite.
  - (i) anorexia.
  - (ii) normal.
  - (iii) diminished through fear of producing pain.
  - (iv) increased.
  - (v) perverted.
- (g) Hæmatemesis and melæna.
  - (i) frequency.
  - (ii) quantity.
  - (iii) with or without collapse.
- (h) Jaundice.
  - (i) number of attacks.
  - (ii) preceded by pain or colic.
  - (iii) with no pain.
  - (iv) with or without pyrexia.
  - (v) depth.
  - (vi) dark urine or light stools noticed.
- (i) Condition of the bowels.
  - (i) normal.
  - (ii) constipated.
  - (iii) diarrhœa—number of stools.
  - (iv) alternating diarrhœa and constipation.
  - (v) character of the stools.
    - 1. colour (e.g. black, tarry, clay).
    - 2. odour.
    - 3. admixed with blood and slime.
    - 4. other features.
- (j) Other complaints and symptoms.

(B) PHYSICAL EXAMINATION

(1) *General examination.*

- (a) General appearance.
- (b) Build.
- (c) Condition of skin ; colour.
- (d) Eyes, tongue, teeth, tonsils, pharynx, etc.
- (e) Heart and lungs ; blood-pressure =  $\frac{\text{systolic.}}{\text{diastolic.}}$
- (f) Nervous system ; reflexes.

(2) *Abdominal examination.*

- (a) Inspection.
  - (i) respiratory movements.
  - (ii) distension or retraction.
  - (iii) presence of herniæ.
  - (iv) visible peristalsis.
  - (v) visible tumour.
- (b) Palpation and percussion.
  - (i) superficial abdominal reflexes.
  - (ii) cutaneous hyperæsthesia.
  - (iii) muscular rigidity.
  - (iv) muscular tenderness.
  - (v) region of umbilicus and linea alba.
- (c) Deep abdominal palpation.
  - (i) abdominal viscera.
  - (ii) visceral tenderness.
  - (iii) tumour.
    - 1. size.
    - 2. shape.
    - 3. surface.
    - 4. position.
    - 5. tenderness.
    - 6. fixed or mobile.
  - (iv) fluid.

(3) *Auscultation.*

- (4) *Rectal* } examination.  
*Vaginal* }

(C) LABORATORY INVESTIGATIONS

- (1) *Gastric analysis.*
- (2) *Tests for occult blood.*
- (3) *Examination of urine ; tests for renal efficiency.*
- (4) *Wassermann reaction and Kahn test for syphilis.*
- (5) *Complete blood count.*

## (D) RADIOLOGICAL INVESTIGATIONS

- (1) *Barium meal.*
- (2) *Barium enema.*
- (3) *Cholecystography.*
- (4) *Other X-ray investigations.*

## (E) EXAMINATION OF THE STOMACH WITH SPECIAL INSTRUMENTS

- (1) *Gastroscope.*
- (2) *Gastro-photor.*

## (F) EXPLORATORY LAPAROTOMY

## (G) DIAGNOSIS

- (1) (in block letters).
- (2) (in block letters).

## (H) TREATMENT

- (1) *Reasons for advising—*
  - (a) Medical treatment.
  - (b) Operation.
- (2) *Details of medical treatment.*
  - (a) Ambulatory.
  - (b) Strict medical.
    - (i) name and address of doctor.
    - (ii) hospital or nursing institution.
  - (c) Duration.
  - (d) Result on discharge.
- (3) *Details of operation.*
  - (a) Date.
  - (b) Hospital or nursing institution.
  - (c) Name of anaesthetist and type of anaesthetic administered.
  - (d) Name and address of surgeon.
  - (e) Operative procedures.
    - (i) (in block letters).
    - (ii) (in block letters).
    - (iii) (in block letters).
  - (f) Post-operative complications.
  - (g) Result on discharge.

## (J) AFTER-HISTORY ("Follow-through" system)

## CHAPTER I

### HISTORY OF THE CASE

by

RODNEY MAINGOT

It is generally agreed that of all the available methods of investigation a well-taken history is the most important. The surgeon should preferably take the history himself, according to the scheme outlined above, to ensure that no important detail is omitted and to obtain the full benefits which accrue from personal interrogation by one versed in this type of disease.

It will be found that the majority of cases of dyspepsia can be correctly diagnosed on the history alone, and a serious attempt at this should be made in every case before physical, radiological, and laboratory methods are undertaken. In duodenal ulcer the symptoms are so characteristic that in fully 90 per cent of cases a confident and correct diagnosis can be formed on the history alone. Although in chronic gastric ulcer, gall-stones, and other dyspeptic conditions the diagnosis from an anamnesis may not reach such a high pitch of perfection, yet with the increasing knowledge of the symptomatology of these diseases a correct diagnosis can frequently be made. It should therefore be emphasised that a painstaking interrogation and interpretation of all the various symptoms is amply repaid and well worth while.

The patient should tell his story in his own words, but in order to prevent irrelevancies the surgeon, while avoiding leading questions, should, by timely interrogation, guide the inquiry into right channels. Each item in the above form should be carefully filled in, as each may have a significant bearing on the case. The name and address of the doctor should be entered, not only for supplying him with all the available details of his case, but for future reference in connection with the "follow-through" system should the patient, owing to his living at some great distance, be unable to make periodic visits for examination by the specialist.

(1) *Complaints.*—Under this heading *all* the patient's complaints should be noted *seriatim*.

(2) *Family history.*—A brief inquiry into the patient's family history may yield useful information, e.g., whether or not there is a peptic ulcer diathesis. Hurst has shown that gastric and duodenal ulcers may occur in several members of the same family. Some families may show a selective tendency to gastric ulcer, others to duodenal ulcer. Again, achylia gastrica may be an inherited condition, and families with such a history are prone to develop pernicious anæmia or other diseases. Serious illnesses or causes of and age at death in the patient's family should be inquired into as it is well known that certain diseases are inherited. It would be well to inquire into a history of syphilis, and if the patient has acquired this disease in the past he should be made to give a detailed account of any treatment he had and the results of the Wassermann tests. Syphilis may play a leading part in anomalous dyspeptic conditions.

(3) *Social and personal state.*—Under this heading inquiry will have to be made into particulars relating to the patient's meals, whether these are regular or irregular, the amount eaten at each meal, the type of food favoured, and whether or not certain types of foods produce dyspepsia. Again, whether there is a distaste for certain articles of diet.

Very hot or iced drinks, alcoholic beverages, condiments, and such articles of diet as pickles, may be a predisposing cause of gastritis, or a factor in the perpetuation of peptic ulceration. With regard to alcohol it will be necessary to ascertain not only the amount imbibed, but the type of alcohol, whether it be beer, whisky, wines, liqueurs, etc., and the average number of drinks of this nature that the patient takes during the day, as alcohol is a recognised cause of cirrhosis, gastritis, and various other gastro-intestinal disorders. The general tendency to under-estimate the total amount imbibed daily should be remembered.

Excessive smoking may also lead to dyspepsia and hyperchlorhydria. Nicotine is a potent agent in stimulating the gastric juice and in maintaining a high acid curve. Patients often also under-estimate the amount of tobacco smoked during the course of a day, and usually on careful inquiry it will be found that their assessment falls very far short of what actually proves to be the case.

In addition to inquiry into the patient's business it may be well to know what his usual habits are, his pastimes, and even his virtues and

vices, and whether by residence abroad at any period of his life he has been exposed to any tropical disease.

(4) *Previous illnesses.*—Information concerning the patient's health in the past is important, as it may have a direct bearing on his present condition. A history of pyorrhœa or infected tonsils, sore throat, or nasal discharge, may explain the production of the dyspeptic symptoms of which the patient now complains.

The severity of previous illnesses can be somewhat gauged by a knowledge of whether the patient was obliged to go to bed (if so, for how long) and to summon medical advice. If investigations were undertaken in connection with any past illness, material help would be gained by obtaining such records. If, on the other hand, an operation has been performed, details of the operative findings and procedures should be acquired; also particulars of any medical treatment which the patient has undergone. A history of jaundice, tuberculosis, syphilis, or specific fevers may throw light on the pathology of the present condition. A considerable number of patients do not understand the term "jaundice," and it is not sufficient merely to inquire whether their eyes or skin have been yellow at any time in the past, as more often than not they will state that their friends have noticed such a condition to be present on various occasions. The condition will only be of importance where it has warranted medical attention.

(5) *Duration and progress of symptoms.*—Having obtained a brief summary of the patient's complaints and past history, it will now be necessary to enter into some detail regarding the duration and progress of the symptoms. Here three leading questions are permissible, and inquiry should be made as to (i) when perfect health was last enjoyed; (ii) what was the first thing noticed wrong; and (iii) what was the subsequent course of the illness.

*The initial symptoms of any disease are the most important and are often forgotten.* Patients will frequently describe the present or the last bad attack of dyspepsia, or date their illness from a preceding severe attack, while the inaugural symptoms which accompanied the onset of the disease, and which may have been mild in character, are overlooked. It is therefore most essential to ascertain as far as possible the exact date of onset, as the duration of the symptoms may play an important part in the diagnosis.

(a) *Short history.* A short history is rarely obtained in cases suffering from chronic dyspepsia, nor is it pathognomonic of cancer of the

stomach. However, in a patient over forty years of age who has previously enjoyed perfect health, and who gives a short history of indigestion which is unappeased by medicine, the possibility of the early onset of cancer of the stomach should not be forgotten, and further investigations should be undertaken with this in mind.

With cancer of the stomach and gastritis the history may be brief, but it is most exceptional to get a short history with chronic peptic ulcer, as in these latter cases the symptoms frequently date back many years, in fact, in some instances to the patient's adolescence.

(b) *Long history.* In dyspepsia cases, as noted above, the history is usually a long one. Patients so suffering may have continuous symptoms, or they may have intermissions during which they are more or less free from their discomfort or pain. These intermissions are of the utmost importance as they are very characteristic of gastric and duodenal ulcer.

While a long history with *continuous symptoms* is very characteristic of visceroptosis, and may be a prominent feature in some cases of gallstones or chronic cholecystitis, a long history with *intermissions* is almost pathognomonic of peptic ulcer.

Having ascertained that the patient has a long dyspeptic history with intermissions, it will be necessary to inquire if during these intervals there is—(i) freedom from symptoms; (ii) discomfort between the attacks; or (iii) almost continuous mild symptoms with periodic severe or acute attacks.

(i) In uncomplicated duodenal ulcer there will be attacks of pain coming on about 3 hours after meals, alleviated by partaking of food. This will usually afford relief for a period varying from 2–4 hours, and so the cycle of pain, food, relief of pain, and pain, may continue every day for several days or weeks during a typical attack. The symptoms will eventually disappear, and there will be a period in which the patient will enjoy good health and not complain of any symptoms at all.

In chronic gastric ulcer, however, the intermissions are usually much shorter than obtains in the case of duodenal ulcer, and there is rarely a period of complete freedom from symptoms for more than 2 or 3 months between the attacks.

The time of the onset of pain after meals is also significant. The nearer the cardiac end of the stomach the ulcer is, the sooner the pain will be ushered in after the ingestion of food. For instance, where the ulcer is situated in the middle of the lesser curvature the pain may

come on  $\frac{1}{2}$ –1 hour after meals; whereas with one situated at the pylorus there will be a later incidence, i.e. 2–3 hours.

(ii) In visceroptosis and appendix dyspepsia there may be a long history with intermissions, but in these cases there is always discomfort between the attacks.

(iii) In patients suffering from gall-stones the symptoms are usually mild, but there will be periodic severe or acute attacks depending on the onset of cholecystitis or colic. These patients generally complain of flatulence, distension of the stomach—particularly after meals, and vague and irregular bouts of dyspepsia during the intermissions. Then suddenly, without warning, they may be prostrate with an acute attack of colic. Relief eventually comes, to be followed by the expectant and anxious period of intermission.

*(c) Description of the attacks and of the intervals.*

(i) Long attacks with long intervals are characteristic of uncomplicated peptic ulcer. Attacks which are seasonal, occurring mostly in spring and autumn, are suggestive of chronic duodenal ulceration. In duodenal ulcer the attacks may only recur once or twice in a year, or even more rarely. They will, however, become more frequent and more severe as the disease progresses and when complications supervene. The intermissions in the case of chronic gastric ulcer usually afford a few weeks' or months' reprieve.

(ii) Short attacks with short intervals may occur in appendix dyspepsia, and in those cases of visceroptosis in which the symptoms resemble those of chronic peptic ulcer. It is unusual for a patient suffering from visceroptosis to have complete freedom from all symptoms between the attacks, however short these intervals may be.

(iii) Recent attacks becoming longer and more severe suggest that a gastric or duodenal ulcer has become adherent to a neighbouring viscus, such as the pancreas, that pyloric stenosis or hour-glass stomach may be present, or that a cancer has become grafted on to a chronic ulcer.

(iv) As the patient will invariably describe the last attack in some detail, it is well to encourage him in this respect as much valuable information will be gained, particularly if he can be induced to draw comparisons between the last attack and the previous ones. He may have noted, for instance, that the last attack was perhaps more persistent, or obstinate to medicine, that the pain was different in



character, or that it was associated with complications not previously noted, such as loss of weight and vomiting, clearly suggesting that his condition is assuming a more sinister aspect.

(6) *Analysis of individual symptoms.*—Having completed a summary of the patient's complaints and the length of the history, it will now be necessary to deal in some detail with the individual symptoms.

(a) *Pain.* Pain is the most important symptom in abdominal disease; in fact, it may be the only symptom complained of by the patient. It is therefore essential to go into fullest details with regard to its *position and radiation*.

The *position* of the pain may vary according to the disease present, and in a number of cases it will give an important clue as to which organ is involved. In the early stages of acute intra-abdominal lesions the site of the pain is perhaps more characteristic and definite, and is a greater aid to diagnosis than will be the case in more chronic conditions.

In cases of simple ulcer of the stomach and duodenum the pain is located in the epigastrium; in gastritis, carcinoma, and gastroptosis it tends to be diffuse. In peptic ulcer, when the lesion involves the duodenum, it will be more localised and situated at the outer border of the rectus muscle in the transpyloric plane, whilst if the stomach is the seat of chronic ulceration the tender spot is usually in the mid-epigastric line or to the left of it.

The position of the pain, however, is not always constant, and too much confidence should not be placed in a diagnosis based on the position alone, as this is subject to some variation with certain lesions. For instance, I have seen not a few cases of duodenal ulcer in which the pain was definitely located over the upper half of the *left* rectus muscle, and even on examination the tenderness was found to be over this area.

In gastric ulcers situated near the pylorus the pain may be on the right side, over the middle third of the rectus muscle in the epigastrium, or may be even further out than this in the region of the gall-bladder. In gall-bladder disease the pain is often located below the tip of the ninth costal cartilage, but it may be internal to this, further afield in the right post-renal angle, or even in the right iliac fossa.

The pain will *radiate* widely when the patient is suffering from one of his attacks or during a particularly severe bout of indigestion. When such complications as penetration of the pancreas occur in peptic ulceration, severe pain may be felt in the back. As a rule, therefore,

patients giving a history of duodenal ulcer will complain of pain to the right, and during a more severe attack, and particularly when complications arise, there will be radiation of the pain to the back in the region of the twelfth dorsal vertebra, upwards between the shoulder blades, and piercing even up to the right shoulder.

In gastric ulcer there will be a history of pain in the middle of the epigastrium, or slightly to the left, but as the disease progresses there will be a diffuse radiation over the sternum, the lower half of the left chest, and upwards to the left shoulder. *Backache is a notable feature when a callous ulcer has involved the pancreas or liver.*

In gastro-jejunal ulcer there is often a history of pain just below or to the left of the umbilicus, with radiation towards the left iliac fossa or into the hypogastrium. With diseases of the gall-bladder there is pain in the right hypochondrium, below the costal margin, and this may extend to the back or shoot upwards to the right shoulder and supra-clavicular region. Radiation towards the umbilicus or even into the right iliac fossa may cause an error in diagnosis. The position of the pain in pancreatic disease is epigastric, usually diffuse, more intense just above the umbilicus, and may also extend to the back. If the dyspepsia is due to chronic appendicitis the pain will often be localised to the right iliac fossa, but it may radiate to the epigastrium. Pressure over the caecum or appendix in such cases will also cause epigastric pain similar to that of which the patient has been complaining.

Pain may be *mild*, described merely as discomfort. Such pain may be experienced in early cases of cancer of the body of the stomach, in chronic gastritis, chronic pancreatitis, chronic colitis, visceroptosis, and in uncomplicated cases of gall-stones. Here, in the interpretation of his pain, we are dependent upon the patient's description of it. It is very difficult indeed to assess accurately the amount and character of the pain, as it is a subjective phenomenon. In two patients with an identical lesion the pain may be described by one as being very severe and by the other as being mild. It is difficult, too, to interpret terms, such as "boring," "aching," "burning," "cutting," "acid," "sharp," "tearing," "dull," frequently used by patients in referring to their pain. Pain, in fact, is one of the most teasing symptoms to describe, but without it very few patients would, in my opinion, seek medical advice, even in the presence of obvious disease.

In certain cases of dyspepsia the pain is described as being *severe* or *acute*. This is well demonstrated in peptic ulcer when adhesions are present, or when penetration of such a viscus as the liver or pancreas occurs. The pain which was bearable before now becomes intolerable. In order

to get some relief the patient will apply pressure to the epigastrium, take copious draughts of alkaline mixtures, or resort to self-induced vomiting. Nothing will afford these patients greater relief, however transient, than vomiting. Once alleviation has been obtained by this method they will be found to resort to it with increasing frequency. The pain will be described as *severe* during an attack of acute gastritis; so also where cancer of the stomach and peptic ulcer have produced obstructive symptoms.

In cholecystitis associated with gall-stones there may be quiescent intervals, mild grumbling attacks of discomfort, and the uneasiness that accompanies gastric flatulence. When, however, acute infection supervenes there may be severe pain in the region of the gall-bladder associated with *pyrexia*. In gall-stone colic the pain is excruciating, it radiates to the back and shoulder, and is accompanied by *collapse*. Probably the worst pain that an individual can be called upon to bear is that which ushers in an acute hæmorrhagic pancreatitis. The words "acute" or "severe" are inadequate to describe the agonising torment, the relentless, burning, strangling pain which grips the upper abdomen as in a vice, and plunges the patient into a fearful dread of continued agony and impending death. The suggestion of operation with its promise of relief is eagerly and gratefully acquiesced to by the patient.

The pain in acute perforation of the stomach or duodenum, and certainly in some cases of acute obstructive appendicitis, is undoubtedly very intense, but its severity cannot in any way be compared with that of an acute pancreatitis.

In certain organic conditions of the stomach the pain will be described as *constant*, and is unrelieved by partaking of food; in fact, in many cases the ingestion of food will increase the pain or give rise to vomiting. This is often seen in cancer of the stomach, pyloric stenosis, and hour-glass stomach. It is also a characteristic feature when malignant degeneration is superadded to a callous ulcer. Not only is the pain a constant feature after partaking of food in cases of cancer or organic obstruction of the stomach, but the taking of large meals or unsuitable articles of diet will often aggravate the symptoms and precipitate an acute attack.

In simple ulcer and gastro-jejunal ulcer the pain comes on at a longer though variable *interval after food*, the usual time being 3 hours, this being extended in exceptional cases to 6 hours. Some pyloric ulcers, owing to their close proximity to the bulb, may mimic duodenal ulcers in their symptoms.

Pain arising at *short intervals after food* is found in chronic peptic ulceration occurring near the cardia, and also in cancer of the stomach and gastropotosis. In atonic dilatation of the stomach, owing to the diminished gastric muscular tone, a sensation of fulness is produced after a small quantity of food has been taken, and this may develop into actual pain if the patient takes more than he is accustomed to.

"Hunger pain," first described by Lord Moynihan, is an outstanding feature in duodenal ulcer. The patient experiences severe pain which is immediately appeased by partaking of food. The bulkier the meal, the longer will be the interval of relief; but the pain which will eventually return will be correspondingly worse. On the other hand, where a small meal is taken, although the pain is immediately relieved, it will return after a shorter interval and with a lesser intensity.

"Hunger pain" is occasionally seen in diseases other than duodenal ulcer, such as cholecystitis, chronic appendicitis, some cases of cancer of the stomach, duodenitis, and in most cases of hyperchlorhydria unassociated with peptic ulceration. In the majority of cases of cancer of the stomach, visceropotosis, chronic diseases of the gall-bladder, appendix, and pancreas, complicated peptic ulcer (and particularly where there is an hour-glass stomach), pyloric stenosis or adhesion of the ulcer to the pancreas or liver, the intake of food causes an aggravation of the symptoms. Although the appetite may be good, and even capricious, with some of these lesions, the fear of the pain that will inevitably ensue, often enforces an abstinence which borders on starvation.

(b) *Nausea.* Nausea may be a symptom in many cases of dyspepsia. It is associated with a loss of appetite and diminished gastric tone. It is described as a "sinking" feeling in the "pit" of the stomach, and is accompanied by such vasomotor symptoms as faintness, giddiness, cold sweats, etc. In gastric conditions it usually *precedes* vomiting, which is in contrast to the vomiting which may occur without any antecedent nausea in cases of cerebral lesions. Nausea is a very early symptom in carcinoma of the stomach; in fact, it may be the first symptom to indicate that something is seriously amiss. It continues throughout the disease and increases in frequency and in intensity as the growth develops. In gastritis and in chronic diseases of the gall-bladder and appendix nausea is a prominent symptom, and apart from mild pain, discomfort, and indigestion, which usually accompany these diseases, it may be the only presenting feature. It should be remembered that nausea occurs

in such conditions as pregnancy, uræmia, nephritis, anæmia, tuberculosis, functional and neurotic states, debility from any cause, intestinal worms, and chronic drug addiction.

(c) *Vomiting.* In cases of dyspepsia this may be an important and frequent symptom, and the various points in connection with it demand careful inquiry. These should be considered under the following headings :

(i) *Character of vomit.* This alone may elucidate a clinical diagnosis, and the surgeon should always make a personal inspection of a fresh specimen of vomit when it is available, as the macroscopic appearances may be suggestive of certain lesions. For instance, in acute gastritis there may be an excess of mucus. Bright red blood may be quite obvious to the naked eye ; on the other hand, it may be dark brown if it has been subjected to the influence of gastric juice for any length of time. Sometimes the vomit will resemble coffee grounds.

The vomiting of large quantities of bile is seen in cases of migraine, and particularly where vicious circle vomiting has occurred after a gastro-jejunostomy.

In obstruction of the stomach or duodenum, the vomiting may be copious, and recognised undigested particles of food taken two or three days previously may be contained in the vomited material. When the obstruction is due to a fungating necrotic cancer, the vomited matter will be foul, evil-smelling, and frothy from fermentation. Fæculent vomiting is seen in cases of intestinal obstruction, but true fæcal vomiting (i.e., vomiting of fæces) can only occur if the stomach is in direct communication with the colon, which may obtain as a complication in peptic ulceration, i.e., gastro-colic fistula, and in certain cases of advanced cancer of the stomach where the growth has become adherent to and has finally eroded the large gut.

In peptic ulcer unassociated with obstruction the vomiting will be described by the patient as being "acid" and "burning." The amount is usually very small and generally contains much stringy or ropy mucus.

(ii) *Amount.* The further away the obstruction is from the cardia the greater will be the amount vomited. In pyloric occlusion due to cancer, to chronic duodenal ulcer, or to a stenosing gastric ulcer, large amounts are vomited at frequent intervals. As the gastric muscle tone decreases, however, the vomiting will be less frequent but more copious.

In well-established hour-glass stomach the amount will be large or small according to the position of the stricture. If the proximal pouch is small there will be frequent vomiting of *small* amounts of highly acid gastric contents; whereas where the lesion occurs near the pylorus, owing to the increased capacity of the proximal pouch, the amounts vomited will be *larger*, and the condition will be indistinguishable from that of pyloric obstruction.

(iii) *Frequency.* It should be borne in mind that a considerable number of patients who complain of dyspepsia and who have a visceral organic lesion do not vomit. Vomiting is rare in simple duodenal ulcer, and is stated to be absent in fully 25 per cent of cases of chronic gastric ulcer. When an obstruction supervenes, however, vomiting invariably occurs.

Vomiting is *frequent* in acute gastritis, organic obstruction of the stomach, and in those cases of gall-bladder disease associated with bouts of acute cholecystitis and colic due to gall-stones.

(iv) *Relation to food.* Vomiting occurring *immediately* after the intake of food is seen in functional disorders and in organic oesophageal obstruction. In gastric ulcer vomiting usually occurs at the height of the pain, 1-2 hours after the intake of food. In duodenal ulcer the incidence is rare, but when vomiting occurs it does so after a longer interval, usually 3-4 hours after food. Vomiting occurring *several hours after* a meal, as has been pointed out before, is very characteristic of a stenosing ulcer producing pyloric obstruction.

In alcoholic gastritis, cirrhosis, and pregnancy, vomiting is most usual in the early morning *before* breakfast, whereas in atonic dilatation of the stomach and pyloric stenosis vomiting more frequently takes place towards the close of the day.

(v) *Self-induced vomiting.* Self-induced vomiting is commonly seen in cases of gastroparesis, atonic gastric dilatation, peptic ulcer, gall-stones, gastric flatulence from any cause, and in functional disorders.

(vi) *Relief of pain.* It is well known that relief of pain immediately follows vomiting in cases of peptic ulceration, but although some relief is afforded in gastritis and gastroparesis the same cannot be said of the vomiting associated with biliary, pancreatic and renal conditions, appendicitis, acute dilatation of the stomach, or intestinal obstruction.

(d) *Belching; flatulence; regurgitation; heartburn; sense of fulness.* Very mild belching or eructations occur normally after meals, but may be a very troublesome and common symptom in certain cases of

dyspepsia. It is frequently associated with gastritis, peptic ulcer, and functional disorders (ærophagia).

In uncomplicated gastric disorders the eructations are odourless, and the same applies in benign pyloric obstruction. In carcinoma of the stomach, owing to the decomposition of proteins by lactic and butyric acids which are present, the belched gas is foul-smelling and may contain a quantity of sulphuretted hydrogen. In the rare cases of gastro-colic fistula the fæculent odour of the eructations may be a characteristic feature.

The patient may sometimes experience great difficulty in differentiating between gastric and intestinal flatulence. The intestinal distension is mistaken for gastric flatulence, and in fruitless efforts to expel the gas by mouth more air is swallowed. Although belching will relieve gastric flatulence, it will have no effect upon the pent-up gases in the flexures of the colon. Gastric flatulence or wind is often associated with palpitation and other cardiac symptoms, and is due to the gas-filled stomach displacing the diaphragm and heart in an upward direction.

Some *regurgitation* of gastric chyme usually occurs at the commencement of a meal, and in its exaggerated form it may be present in many gastric disorders.

*Water-brash* is described as a sudden filling of the mouth with alkaline watery saliva. It is common in patients who exhibit the hypersthenic ulcer diathesis, and in duodenal ulcer. It is said to be a natural reaction on the part of the body to neutralise the excess of acid.

*Pyrosis*, or the acid regurgitation of gastric contents into the mouth and pharynx, is attended by a bitter or burning sensation in the throat and is another symptom of hyperacidity.

*Heartburn* is a burning or eroding sensation felt behind the lower portion of the sternum. It is a frequent concomitant of chronic dyspepsia and may be associated with excessive belching and acid regurgitation. It is instantly relieved by taking alkalis. Numerous theories have been advanced to explain this symptom, and a plausible explanation is that it is due to increased tone and excessive peristalsis of the lower end of the gullet, secondary to an œsophagitis produced by the frequent regurgitation of gastric juices of very high acid content.

A *sense of fulness* in the epigastrium is a common symptom in diseases of the gall-bladder, carcinoma of the stomach, and in any condition that is associated with enlargement of the stomach, e.g., gastric atony and benign pyloric stenosis. The sensation is probably

the result of stretching of the muscle-fibres of the stomach by gas or by the intake of food which increases the pressure inside the stomach. Pressure on the stomach from conditions such as secondary deposits in the glands or liver, epigastric cysts, gas in the transverse colon, enlarged spleen, etc., will also give rise to a sensation of fulness by interfering with the muscular mechanism of the stomach.

(e) *Weight.* In patients suffering from dyspepsia the weight is sometimes stationary, but on the other hand there may be a gain or a loss which is either *gradual* or *rapid*. The loss in either case may be marked. Loss of weight which is gradual is less likely to be noticed by the patient than a rapid loss which is suggestive of organic obstruction in the œsophagus or stomach. *The most rapid loss of weight is seen in cases of œsophageal obstruction.* There is always loss of weight with hour-glass stomach, pyloric stenosis, and obstruction of the common bile-duct. In the latter condition the wasting may be very rapid, so that the patient will show obvious signs of emaciation. *A marked loss of weight, if it is not due to strict dieting, is a symptom strongly suggestive of organic disease.* In cancer of the stomach the loss will be rapid or gradual, depending largely upon the position of the growth, and whether or not obstruction is present. The loss will be rapid when the growth is situated at the cardiac orifice, the pyloric orifice, or elsewhere in the stomach when it is large and fungating and is associated with obstructive symptoms. It is gradual when the growth is situated in the body of the stomach, in the early stages of leather-bottle stomach, and in all cases where the growth springs from the region of the greater curvature and does not involve the pylorus.

In gastric and duodenal ulcer the weight may be stationary, or in some instances the patient may actually gain. When, however, such complications as penetration of the pancreas, hour-glass stricture, and pyloric stenosis ensue, the fear of the pain or vomiting that will follow the intake of food produces a self-induced starvation which necessarily results in loss of weight.

In taking the history careful distinction should always be made between true anorexia and a condition in which the patient is hungry and has a good appetite, but is afraid to eat owing to the subsequent pain that will ensue. An increase of weight will be noticed in cases of simple duodenal ulcer, owing to the fact that the frequent intake of food will relieve symptoms. In patients suffering from gall-stones and chronic appendicitis, the appetite is good as a rule, and the patient may actually put on weight; on the other hand, in certain cases, owing to the sense



of fulness and epigastric discomfort that result from the intake of food, the patient may resort to dieting, and loss of weight follow.

(f) *Appetite.* Appetite is the natural desire for food, and is a sign of efficient gastric tone and secretion. A loss of appetite or anorexia, on the other hand, is an indication of the converse of this. Increased appetite is nearly always associated with hyperchlorhydria, whereas a loss is likewise associated with a diminution of hydrochloric acid. Anorexia is a common complaint in many cases of dyspepsia and functional disorders. It is a prominent symptom in gastritis, new growths of the stomach, and other conditions accompanied by atony and dilatation of the stomach. In cancer of the stomach there is often a peculiar distaste for meat, and in patients suffering from cholelithiasis there is usually a dislike of fats, such as butter and cream. Acute loss of appetite, particularly if it is associated with loss of weight, may be the only symptom of an early cancer of the stomach, and in any patient over the age of forty, who has suddenly noticed this condition for the first time, a complete investigation of the stomach is imperative.

(g) *Hæmatemesis and melæna.* There are many causes of hæmatemesis, though peptic ulcer is by far the commonest. It is estimated to occur more frequently in gastric than in duodenal ulcer, and in a severe form in at least 10 per cent of cases of chronic peptic ulceration.

The vomited blood may be bright red. When it is dark brown or black this is due to alterations which have occurred from the action of the acid gastric juice. It may be represented as "coffee grounds" or, although present in the vomit, it may be detected only by means of the microscope (occult blood). Collapse and other signs associated with loss of blood are evident in cases of profuse hæmatemesis.

Hæmatemesis is often the only symptom of an acute peptic ulcer, and its occurrence in cases of cirrhosis of the liver, splenic diseases, blood dyscrasias, and after operations upon the stomach requires only to be mentioned. The hæmatemesis which sometimes occurs with gallstones, appendix dyspepsia, and acute appendicitis is dependent upon the formation of multiple acute peptic erosions.

Melæna occurs commonly in duodenal ulcer, less frequently in gastric ulcer, and even more rarely in cancer and innocent new growths of the stomach. In melæna the stools are black and tarry. In cases of hæmorrhage from the large gut the blood which is passed in the motions is red. It is very rarely tarry in nature. The naked-

eye appearances, therefore, of blood that is passed in the motions may be helpful in indicating its origin. Occult blood is found in all cases of active gastric and duodenal ulcer, and is a very important test in the diagnosis and management of these cases. Its persistence indicates that the ulcer is still active, whereas if the test is consistently negative on several occasions there is strong presumptive evidence that healing is taking place satisfactorily.

Hæmatemesis and melæna may occur in cases of cancer of the stomach, but while it is rare to have a copious hæmatemesis in such cases, occult blood is repeatedly found on examination of the stools.

(h) *Jaundice*. This is an important symptom in gastro-intestinal diseases, and its occurrence will often help to elucidate the diagnosis. Its mechanism, causation, and classification into obstructive, hæmolytic, and toxic and infective, are well known, and are clearly and brilliantly described in the writings of McNee.

In investigating a case it will be necessary to inquire into the number of attacks that the patient has had, whether an attack has been preceded by pain or colic, and whether or not it was accompanied by pyrexia. The fact that the patient suffers from attacks of jaundice is suggestive of the presence of gall-stones, although there are many cases of cholelithiasis who have never had jaundice. Attacks of colic, too, although a prominent feature of gall-stones, may be entirely absent in a large number of such cases. While pain preceding or accompanying an attack of jaundice indicates the presence of gall-stones it is when jaundice occurs in the absence of pain that the greatest difficulties in diagnosis arise. Painless jaundice, particularly when it is persistent and of increasing intensity, is very suggestive of cancer of the head of the pancreas, but it is occasionally seen in obstruction of the common bile-duct due to stone. In both conditions rapid emaciation is a marked feature, but the latter is often accompanied also by pyrexia.

Intermittent attacks of jaundice, preceded by pain and accompanied by pyrexia, are pathognomonic of cholelithiasis. Jaundice which occurs without attacks of pain or fever, and which persists in a patient past middle life, is in all probability due to a cancer of the head of the pancreas, rarely of the common bile-duct, or to a sclerosing chronic pancreatitis. If itching is a marked feature it is strong evidence of an obstructive lesion, as it does not normally occur in the other types of jaundice.

In investigating a case of jaundice special inquiry will have to be made to ascertain whether there have been any previous toxic or

infective factors to account for the present attack. For instance, jaundice is found in certain acute specific fevers, in certain cases of syphilis, where excessive doses of salvarsan or equivalent drugs have been injected intravenously, after the administration of certain poisons such as phosphorus, etc., and in certain septic conditions.

Jaundice of a mild nature is seen in the familial condition of acbolic jaundice with increased fragility of the red cells, and also in the acquired form when it is frequently associated with the formation of pigment gall-stones. Jaundice occurring in several members of the same household, or in epidemics, is at once suggestive of the epidemic catarrhal form which is associated with indigestion, slight pyrexia, and occasional bouts of mild colic.

(4) *Condition of the bowels.* It will be found on taking the histories of patients suffering from dyspepsia that the condition of the bowels, whether they are normal, constipated, or whether there is diarrhoea or diarrhoea alternating with constipation, will yield useful information in confirming a diagnosis. In certain lesions there is always constipation, whereas in others this symptom is not present, and there may even be diarrhoea. Although the absence or presence of constipation or diarrhoea may not be helpful in differentiating between one form of dyspepsia and another, it may be useful in ruling out the possibility of other gastro-intestinal lesions. In the majority of cases of dyspepsia constipation is present; for instance, in duodenal ulcer, duodenitis, and in indigestion associated with hyperacidity, in gastric cancer, pyloric stenosis, in chronic appendicitis, visceroptosis, and in chronic diseases of the gall-bladder, constipation is almost invariably found to be present. It should also be noted that any lesion accompanied by vomiting will cause constipation. This is well seen in cases of pyloric stenosis, whether it be the acquired or the congenital form. In cases of obstruction of the gut constipation is a characteristic, and may be a pathognomonic symptom.

The majority of drugs that are taken for the symptomatic cure of dyspepsia, chiefly in virtue of their bismuth content, produce constipation, which in turn may result in an aggravation of the original symptoms.

Diarrhoea is a common complaint in nervous and functional disorders, in colitis, pancreatic insufficiency, and in a host of other conditions associated with dyspepsia. It not infrequently results from a catarrhal colitis induced by excessive purgation, or by the taking of certain drugs in the symptomatic treatment of indigestion.

In this country gastrogenous diarrhœa is fairly common, and is due to diminution or absence of gastric secretion, especially of hydrochloric acid. Patients will often pass three or four soft, foul-smelling stools during the course of the day, and will complain of mild dyspepsia and a distaste for certain forms of food.

Diarrhœa occasionally occurs after the operation of partial gastrectomy for carcinoma or peptic ulcer, or after gastro-enterostomy for pyloric stenosis, and is often secondary to an enteritis produced by the rapid emptying into the jejunum of undigested and irritating gastric contents. It is surprising that diarrhœa does not result more frequently after these operations have been performed, as they are in themselves a plea for careful dieting and prolonged medicinal treatment.

Alternating diarrhœa and constipation is found in cancerous strictures of the descending and sigmoid portions of the colon, and is occasionally seen in cases of diverticulitis. In gastro-colic fistula alternating diarrhœa and constipation may be present, though diarrhœa alone is more usual. The fæces in these cases will often contain undigested food which has been recently ingested.

In most cases an inspection of the stools, especially with regard to colour, odour, and whether or not blood and slime are present, will yield valuable information. The motions will be tarry or black when active peptic ulceration is associated with bleeding of any severity, the altered blood being intimately mixed with the fæcal material.

In certain cases of torrential hæmorrhage from a duodenal ulcer, associated with diarrhœa, the blood passed may be red, but this is most unusual. Bright red or brown blood in the motions, usually in streaks on the surface or discrete from the main masses, implies that there is an ulcerative condition of the colon. It is only in the most exceptional cases that black or tarry motions will be found with organic lesions involving the large gut, but it is almost pathognomonic of duodenal ulceration.

After the ingestion of large doses of bismuth or iron the stools will be found to be grey or even black, but the fact that the patient admits having taken these drugs will account for these physical characteristics. In obstructive jaundice the stools are clay-coloured and putty-like in consistency. Large frothy, fatty, evil-smelling motions are present in chronic pancreatic diseases.

(j) *Other complaints and symptoms.* As there is practically no organ in the body which, when diseased, will not give rise to some symptoms of dyspepsia or mimic gastro-intestinal disorders, it is most

important to inquire into the main symptoms which may be produced by diseases of the other systems, such as cardio-vascular, nervous, genito-urinary, etc. In a severe attack, the gastric crisis of tabes has been mistaken for an abdominal catastrophe such as perforated gastric or duodenal ulcer, and numerous instances are on record where an unnecessary operation has been performed. In coronary thrombosis, pericarditis, basal pleurisy, and other intra-thoracic diseases, symptoms indistinguishable from an acute dyspepsia or an abdominal calamity are sometimes reproduced.

In enlarged prostate, associated with retention, back pressure, and chronic uræmia, the symptoms of a chronic duodenal ulcer, or cholecystitis associated with gall-stones, are sometimes mimicked and may mislead the surgeon into believing that a double pathological condition is present.

It is futile to examine one system completely and thoroughly and to ignore the others, as multiple organic lesions may be present in the same patient.

## CHAPTER II

### EXAMINATION OF THE STOMACH WITH SPECIAL INSTRUMENTS

by

RODNEY MAINGOT

THERE have been a large number of ingenious instruments devised at one time and another for investigating the interior of the stomach, but although they have been popular on the Continent they have only occasionally been used in this country. Only two of these recent innovations will be here briefly described.

(1) *Gastroscope*.—The new flexible Wolf-Schindler gastroscope (fig. 39) is an instrument that is used extensively in Germany, but, as far as I am aware, very rarely in England.

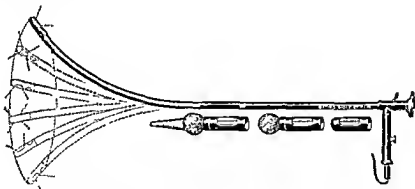


Fig. 39.—THE WOLF-SCHINDLER FLEXIBLE GASTROSCOPE.

It resembles a long cystoscope with an eye-piece, and has a flexible shaft, a strong terminal light, and an excellent Wolf lens placed proximal to the illumination. After the induction of narcosis by the intravenous injection of evipan, or the cocaineisation of the throat and pharynx, the instrument is passed into the stomach and air is pumped into the viscus to produce a moderate dilatation. Owing to its flexibility the gastroscope can be manoeuvred into the various portions of the stomach cavity, and a good view be thus obtained of the greater part of its

lining. Gastric juice, mucus, etc., which may cloud the field of vision, can be wiped away by rubbing the lens gently against the gastric mucosa.

Preliminary preparation is necessary. It is best to wash out the stomach thoroughly with a weak solution of bicarbonate of soda, and to aspirate the contents completely about  $\frac{1}{2}$  hour before the investigation is performed. The intramuscular injection of  $\frac{1}{4}$  gr. of atropine sulphate  $\frac{1}{2}$  hour beforehand will facilitate the examination by diminishing the gastric secretion.

There is no doubt that a very excellent view of the interior of the stomach can often be obtained, and in certain conditions such lesions as chronic gastritis, polypi, chronic gastric ulcer, and the papilliferous and ulcerated types of cancer of the stomach can be very clearly seen.

While it may be possible to interpret gross lesions by this means, it is of little or doubtful value in differentiating between a gastric ulcer and an early cancer of the stomach, or in identifying the malignant changes that are occurring at the border of a chronic peptic ulcer. This is to be expected as it is sometimes impossible after the specimen has been removed at operation to make such a differentiation, even with the naked eye. Again, a great deal of technical skill is required in passing the instrument and in the interpretation of what is actually seen. It may, in the future, have a limited field of usefulness in amplifying, elucidating, or even correcting the diagnosis of a gastric lesion arrived at after an X-ray examination by means of a barium meal.

(2) *Gastro-photography*.—The gastro-photor is an instrument which has been invented for taking photographs of the interior of the stomach. It is a great mechanical achievement and consists of a semi-rigid rubber tube, fitted at its lower end with a minute camera which, instead of lenses, has light pin-holes arranged vertically in pairs.

The stomach is thoroughly irrigated with warm bicarbonate of soda solution, and the contents aspirated before the examination is undertaken. The examination is conducted in the X-ray room, and after the instrument has been passed its position in the stomach is located under the screen. As it is composed of metal it shows up very clearly, and it can be manipulated to lie in any desired portion of the stomach. Its position in the stomach cannot be accurately determined, and a certain amount of guess-work is necessary in placing it in the ideal position for the exposures. The examination takes only a few minutes, and is painless and free from danger.

After the exposures the instrument is withdrawn, the films are

developed, magnified, and then examined—preferably stereoscopically. I have examined a number of films and positive pictures taken by the gastro-photor, but the results obtained have not impressed me very favourably. Most of the pictures are very blurred, indistinct, and out of focus, and the area included in each picture is so small that it is practically impossible to draw any definite conclusion as to the pathological condition present. Occasionally the swollen edge of a gastric ulcer, its cavernous crater, and the radiating, puckered folds of mucous membrane leading to it, may appear in a lucky exposure; but as often as not they are likely to be missing owing to the diminished size of each picture.

The edge of a malignant ulcer, or the tortuous, corrugated and enlarged folds of gastric mucous membrane, cloaked here and there with thick mucus, may be depicted in certain cases of gastritis.

The instrument may be described as ingenious, but it competes in no respect with modern radiological methods.

### EXPLORATORY LAPAROTOMY

It is rare nowadays for an exploratory laparotomy to be undertaken in order to make a diagnosis in any gastro-intestinal disorder, as the methods of investigation that are now available, and particularly by means of X-rays and biochemical tests in expert hands, have reached such a high pitch of efficiency and accuracy. It should therefore be possible to arrive at a correct diagnosis in the majority of cases before advising any operative measures.

The number of cases in which it is considered necessary on flimsy evidence to "look and see" should be very few, and it is only where whimsical signs and symptoms suggest to an astute diagnostician that an early cancer of the stomach or other grave visceral lesion may be present in the absence of positive physical findings, or where an abdominal tumour, by presenting unusual features, proves baffling, that such a form of inquiry can be justified.

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I wish to acknowledge specially the courtesy of Dr. M. A. Araf, of Cairo University, in granting me permission to base certain parts of this article on his excellent book, *Modern Aspects of Gastro-enterology*, published by Messrs. Baillière, Tindall & Cox. 1933.



## CHAPTER III

### PHYSICAL EXAMINATION OF THE PATIENT

by

R. SLEIGH JOHNSON

(1) *General examination.* In the majority of cases it will be found that the careful taking of a history according to a detailed scheme as shown on page C4 will suggest a probable, or at least a differential diagnosis. The object of physical examination is then to confirm or negative these tentative conclusions.

In this respect two words of caution may perhaps legitimately be sounded. The temptation must be rigidly resisted in the first place unduly to hasten or restrict examination to particular organs without having due regard to the patient as a whole ; and secondly, to proceed too hastily to special instrumental or pathological means of investigation without first obtaining the fullest possible information from one's ordinary senses and from inspection in particular. Spot-diagnosis carries its dangers as well as its brilliance.

In a broad preliminary survey, attention is naturally first paid to the patient's *general appearance, build, and state of nutrition*. Variations from the average in physique or build, although not in themselves necessarily pathological, may be of significance in affecting the likely diagnosis in cases of disturbed gastro-intestinal function. Thus, in many dyspeptic patients a wide initial classification can be made into one of two main groups, the so-called "sthenic" and "asthenic" types. Not only is physique concerned but also considerations of temperament, habits, and indeed of the whole mental outlook. While it is the "functional" types of dyspepsia to which these observations chiefly apply, long continued disorder of physiological function is not infrequently followed by organic disease, as with duodenal ulcer.

The sthenic form is met with commonly in men of alert, active and energetic type, with slow full pulse and good appetite. Normally robust and strong, they have often lived well and fully, and later tend to develop high blood-pressure and arterial disease. The asthenic type,

which includes many hypochondriacs, is personified by the thin, flabby, constipated woman with a long narrow thorax, poor appetite, flatulence, low blood-pressure and soft frequent pulse. Her mental attitude is one of depression and she is much given to introspection and worry. When, under strain, the digestive function gives way, the former type tends to show a hyperchlorhydric type of dyspepsia with exaggerated peristalsis and rapid emptying of the stomach which may develop into peptic ulcer. The latter group, in whom sympathetic rather than vagal control is uppermost, are often the subjects of visceroptosis, with a general loss of tone of a part or whole of the gastro-intestinal mechanism in which the abdominal wall also shares. In these cases emptying of the stomach is slow and secretion of acid deficient.

The factor of sex is also of significance, as shown by the far greater incidence of gastric carcinoma in men and of gall-stones or visceroptosis in women; similarly, special diseases are common at special ages.

*The degree of nutrition* is probably linked up more intimately with digestive disorders than with disturbance of any other system. Obesity, for example, carries with it an increased liability to hernia, diverticulitis or gall-stones. Emaciation should bring to mind two frequent causes; a mechanical interference with the entry or absorption of food, and the possibility of malignancy. When due to stricture, the higher its level in the alimentary canal the more marked and rapid is the loss of weight that ensues. Such wasting is seen in oesophageal stricture and stenosis of the pylorus. Simple peptic ulcer is not usually associated with any striking loss of weight unless there be obstruction or prolonged restriction of diet. Defective or inadequate assimilation of food from other reasons, such as poverty of appetite or hysterical vomiting, will also produce wasting though seldom of a comparable degree. The possibility in this regard of other digestive disorders such as a chronic gastro-intestinal catarrh or pancreatitis must also be borne in mind.

Malnutrition from simple deprivation of food, in the broader sense outlined, is unaccompanied by any notable degree of anæmia. A combination, on the other hand, of wasting and a high degree of anæmia in a chronic case is strongly suggestive of malignancy. With the further element of sallowness and pigmentation, the appearance of cachexia is produced. Although not confined to malignant diseases, these probably form the largest single group in its production. Though unhappily only too often seen, the aim in carcinoma is, of course, to diagnose before any marked loss of weight or appearance of cachexia is evident. Gastric carcinoma unless recognised early is often accompanied by a striking grade of *anæmia*, in the production of which

interference with the recently-discovered role of the stomach in normal blood formation probably plays an important part. Usually of secondary type, rarely it may closely mimic pernicious anæmia with a colour index above unity, the distinction being the more difficult in view of the achlorhydria common to both conditions. In these cases, however, leucopenia is seldom as marked in degree. Where, in addition, dyspeptic symptoms are slight or latent over a prolonged period diagnosis is even more obscure, and radiological investigation becomes of outstanding importance.

In other gastro-intestinal diseases the general appearance may reveal an *acute secondary anæmia* as after a single or repeated large hæmorrhage from a peptic ulcer. Here the absence of wasting, the chalky-white pallor without pigmentation, and outward evidence of recent bleeding—dyspnoea, throbbing arteries and tachycardia—will make the diagnosis clear. Confirmation by a blood count will show a high degree of secondary anæmia with low colour index and temporary rise in leucocytes. By contrast, it is striking how little shortness of breath may accompany anæmia of the same grade but of insidious onset and development. Severe anæmia from whatever cause, whether primary or secondary, must in itself be considered as a possible explanation of dyspeptic symptoms, with complaint of poor appetite with fulness or discomfort developing early after food. When accompanied by general fatigue and markedly relieved by rest the probability of such an explanation is increased.

Besides pallor other observations of significance from the appearance of the skin concern *texture and pigmentation*. A general loss of elasticity commonly accompanies the later stages of malignancy, often with a diffuse dryness and scaling. *Jaundice*, with its degree and involvement or otherwise of the sclerotics, must be noted, and as a corollary the presence or absence of bile-pigment in the urine and stools. A degree of jaundice obvious in daylight may readily be overlooked when the examination is made by artificial light.

Since the body is not a collection of isolated units, but depends for its health upon the harmonious interaction of all its members, a breakdown in the integrity of one will, as a rule, speedily become evident in the working of the remainder. Thus there are few systems, disease or disturbance of which does not become reflected in symptoms referable, by the patient at least, to his digestion. Indeed, by their early and isolated character they may lead not only the patient but his doctor also into error as to the primary site of disorder. The fundamental necessity, therefore, of a complete even if brief systematic examination

of the patient as a whole needs no further emphasis, and may save many a misdiagnosis or in some cases unnecessary operation. This general physical overhaul may in some circumstances suggest the need for a special investigation, e.g. a blood count, Wassermann reaction, or X-ray examination, before a final diagnosis can be made.

With a complaint of loss of appetite and vomiting, the *facies* may suggest an underlying chronic nephritis or cirrhosis of the liver, and will then incite further examination for disturbance of renal function or for portal obstruction. Cyanosis and dyspnoea will direct special attention to the heart and circulation. The general appearance again will, in some cases, indicate an endocrine disturbance, such as Graves' or Addison's disease, as responsible for otherwise unexplained vomiting and lead to a careful search for confirmatory signs. An early pregnancy will not be overlooked if during examination its possibility be borne in mind.

Special reference must be made, in view of their commonness and importance, to a few particular *affections of the heart and lungs* in which abdominal disease, and even an acute surgical emergency, may be closely simulated.

In *chronic congestive failure*, especially of the right chambers of the heart, as in late mitral disease, there is a gradual loss of appetite, gastric and intestinal flatulence, with nausea, vomiting and diarrhoea or sometimes constipation. These are accompanied by a sense of fulness and oppression after meals, pain in the epigastrium or between the shoulder blades, and, rarely, by a hæmatemesis or melaena. These symptoms are dependent upon a chronic venous congestion or inadequate blood supply of the whole portal system, including the liver, and more especially of the stomach and intestines. Their true significance will be revealed on examination by the early presence of breathlessness on effort, cyanosis, and fulness and pulsation of the jugular veins, while the heart shows objective evidence of embarrassment by enlargement, tachycardia or abnormal rhythm. Later follows oedema of cardiac type, whether of the lungs, peritoneum (ascites), or extremities. On abdominal examination the liver is uniformly enlarged, perhaps to the umbilicus, with a well-defined lower border and a smooth firm surface, often tender and rarely pulsatile.

In *left-sided cardiac failure and in aortic disease* abdominal symptoms are also common. The pain of angina pectoris is often referred, and rarely strictly localised to the epigastrium, and by its intensity may suggest perforation of an ulcer; or it may radiate widely over any region of the abdomen (angina abdominis) and be mistaken for colic.

Retching and vomiting may occur at the height of the attack and further focus attention on the abdomen. Again, the pain may be of any grade of severity from discomfort only to the most intense agony with feeling of impending death.

Diagnosis from an abdominal catastrophe is assisted by the following points: a history of previous attacks, where obtainable, and of their direct relation to physical effort or mental excitement; relief by rest and nitrite administration; the usual spread of pain from an initial substernal site; hyperalgesia and hyperæsthesia of the skin and muscles over the area of distribution of the pain. Generally the more severe the pain the wider is its zone of radiation. The extreme mental anxiety and sense of vice-like constriction may be helpful in differentiation. On the objective side are evidences of defective cardiac nutrition and vasomotor disturbance, e.g. giddiness, syncopal attacks, cardiac asthma (especially nocturnal), and signs of arterial degeneration with raised pressure. During an attack vasoconstriction is the rule, the patient being grey, motionless, with a small hard pulse, infrequent or unchanged in rate, and often an extremely high systolic pressure. Epigastric pain of lesser severity than angina, sometimes mistaken for a simple dyspepsia, may result from aneurysm of the aorta or from aortic regurgitation.

*Thrombosis of a branch of the coronary arteries*, usually the descending division of the left, is another cardiac condition which may cause confusion in diagnosis. Its incidence is almost confined to men in the later decades. A history, if it can be obtained, of previous attacks of pain on effort or of known cardiac disorder will aid in diagnosis, although rarely an unheralded coronary occlusion may be the first sign of disaster. An attack begins with agonising and persistent pain beneath the lower sternum which may radiate not only upwards to the neck and arms but frequently to the epigastrium, with rapid abdominal distension and rigidity. The pain is of such severity as to be wholly unrelieved by nitrites and the customary doses of morphia. Nausea and vomiting are usual, with an extreme degree of shock and *angor animi*. Diagnostic points from an abdominal perforation include a marked degree of restlessness, the patient writhing with pain or even walking about, and the constant presence of dyspnoea which may be the predominant symptom. The breathing is sometimes irregular or of Cheyne-Stokes type, and a dry cough may develop from congestion of the lung bases. The pulse is commonly infrequent with faint heart sounds and an extremely low systolic blood-pressure, often only 70 to 80 mm. Hg. Pericardial friction may sometimes be heard. With the

persistence of the pain signs of venous engorgement and failure become evident. If facilities are available at the time an electro-cardiographic tracing may show pathognomonic features of myocardial infarction and make the diagnosis conclusive.

Other acute abdominal conditions in addition to perforation which may be simulated by coronary thrombosis include gall-stone colic, and in particular acute hæmorrhagic pancreatitis in view of the cyanosis and dyspnœa often found in the latter condition.

*Pericarditis* often gives rise to severe pain in the upper abdomen or spreading widely over its whole extent; with the severe degree of general illness the resemblance to a diffuse peritonitis may then be very marked. As with other referred pain the abdomen may be tender and rigid. The intensity of the pain is usually lessened by adopting a sitting position. Auscultation at intervals over the cardiac area will reveal the characteristic to-and-fro friction.

Sudden obstruction by *embolism* of a branch of the mesenteric artery in malignant endocarditis or mitral stenosis, or a mesenteric venous thrombosis in cirrhosis of the liver, gives rise to the picture of an abdominal catastrophe with prostration and collapse, passage from the stomach and bowel of fresh or altered blood in large amounts, and the rapid development of intestinal obstruction. Careful examination of the heart will afford a clue where this is the primary site of disease.

*Respiratory affections* in causation of abdominal pain may in some circumstances be equally productive of error. Of outstanding importance in this respect is *acute fibrinous pleurisy*, either alone or associated with an underlying *pneumonia*. Following the distribution of the intercostal nerves, the pain may in accordance with its origin be referred and localised to either upper or lower quadrant, and so simulate almost any abdominal emergency. Involvement of the diaphragmatic pleura will be suspected if, besides an abdominal reference, there be complaint of pain at the tip of the shoulder, referred via the phrenic nerve to the area of distribution of the fourth cervical root. This reference is also observed in cases of perforated gastric and duodenal ulcer, as emphasised by Cope. Often, however, no shoulder pain is felt in either condition, when, with a tender, rigid upper abdomen and no friction to guide, diagnosis between a lesion above and a lesion below the diaphragm may be especially difficult and thrown back entirely upon a critical survey of the thorax, which will usually reveal diminution of movement and of air entry over the affected lower lobe.

In children the onset of a *lobar pneumonia* on the right side may be

equally hard to distinguish from acute appendicitis. The most helpful signs are the early appearance of cyanosis and the altered character and increased rapidity of respiration relative to the rise of pulse-rate, aided by a critical search for abnormal pulmonary signs, of which again the earliest to appear is a weakening of the breath sounds. The presence of cough or labial herpes will favour pneumonia, and working of the alae nasi is similarly significant. Again, with a pulmonary infection the initial fever is usually of higher grade and the rise in leucocytes more marked and of earlier development than in appendicitis. A figure, therefore, of say 25,000 to 30,000 at the onset of illness is strong evidence in favour of a diagnosis of pneumonia. With regard to distinguishing abdominal signs, local hyperæsthesia of the skin is more commonly found in appendicitis, while tenderness on rectal examination and pain on extending the right thigh, from psoas rigidity, can sometimes be elicited. Cope has pointed out that deep pressure directed towards the right iliac fossa from the left side of the abdomen will produce pain if the appendix be inflamed, but not in pneumonia. Where clinical signs are inconclusive, a good X-ray of the chest if obtainable may decide the issue. If after weighing all evidence there still be any doubt, as will in some circumstances be the case, it is wiser to take the risk of opening the abdomen by a small incision under morphia and local anaesthesia than to wait for possible peritonitis to develop. In childhood an exploration in error is by no means as serious an undertaking as in adult life, and many will recover.

Other thoracic conditions which may be responsible for abdominal symptoms include *fibrositis of the intercostal muscles* or membranes (pleurodynia), where pain of quite severe degree may be referred to the abdomen. Here local spots of tenderness due to painful inflammatory fibrositic nodules will be found on palpating between the ribs. If the nerve sheaths are inflamed, these tender areas are situated at the site of emergence of the cutaneous branches from the intercostal muscles. The muscular exhaustion that follows chronic cough is a cause of abdominal pain often overlooked and is readily recognised by tenderness on palpation of the axillary folds. Pain due to any of the above thoracic causes is accentuated by deep breathing or coughing. *Herpes zoster* may be responsible for pain whose origin is obscure until the appearance of the characteristic eruption, which may be delayed for some days.

Other pulmonary conditions to be borne in mind in considering more chronic cases of apparent dyspepsia include *early phthisis*, where in the acute toxic variety of young adults anorexia, flatulence, epigastrie pain, and vomiting may dominate the picture and even precede

cough and expectoration. The semblance to peptic ulcer may sometimes be very close. The presence of wasting and evening fever will lead to its suspicion, and in such circumstances, whether physical signs are found or not, a skiagram should be taken of the lung fields. Vomiting may occur reflexly with chronic cough due to any cause, e.g. pertussis in children, irritation of the vagus from dragging on the diaphragm in basal fibrosis, or may be due to the swallowing of offensive sputum setting up a chronic gastritis, as in bronchiectasis. In such circumstances the infecting organism can frequently be recovered from the vomit, a useful method of diagnosis in children.

Many affections of the *central nervous system* have their abdominal manifestations and may therefore be briefly outlined. Of greatest importance to the surgeon is *tabes dorsalis*. In the fully developed clinical picture with gross objective signs no difficulty is likely to arise; rather is it in the early or pre-ataxic stage that error may occur, where owing to interference with the posterior nerve roots subjective sensory disturbances predominate. In this active phase of the disease paroxysms of great pain may occur—the well-known visceral crises. The organ most frequently affected is the stomach. A gastric crisis comes on suddenly with severe abdominal pain and incessant vomiting, lasting usually a day or two, sometimes longer, up to a week. Such attacks tend to recur over long periods and are accompanied by much prostration and general illness, so that an acute surgical emergency, e.g. perforated ulcer or intestinal obstruction, may be diagnosed, and an unnecessary operation performed. Usually, however, muscular rigidity of the abdominal wall and tenderness on rectal examination are absent. Much less commonly, other forms of visceral crisis occur, for example rectal or vesical crises with severe tenesmus or strangury, renal crises with severe pain in the loin resembling renal colic, and other rare varieties.

In these cases error can be avoided by a careful examination of the nervous system, which will always reveal undoubted signs of tabes. Loss of knee-jerks and fully-developed Argyll-Robertson pupils are not to be expected as these are late manifestations, but earlier changes, such as irregularity or inequality and sluggish pupillary reaction to light, loss of one or both ankle-jerks and cutaneous analgesia, will suggest the correct pathology. Evidence of syphilis in the past and a history, if available, of characteristic lightning pains will be of further aid, while between attacks examination of the cerebro-spinal fluid and Wassermann reaction will be conclusive.

*Affections of the spine*, especially Pott's disease, may by pressure on intercostal nerves give rise to pain, the cause of which will be



overlooked if the back be not examined. In any obscure case of abdominal pain an inspection of the vertebral column should be made.

Other nervous causes of vomiting include cerebral tumour, meningitis, migraine, ocular and labyrinthine disease (Ménière's syndrome), and a very large group of so-called functional disorders associated with hysteria, mental anxiety or shock. Where appropriate the possibility should be borne in mind of obscure vomiting being due to drugs such as alcohol, lead, arsenic, or digitalis, and confirmatory signs looked for in other systems. Again, so simple a disturbance as constipation or faulty habits of diet may be responsible. In children ketosis is a common cause of repeated bouts of sickness, evident on testing the urine if the odour of the breath does not suffice.

Vomiting is a cardinal symptom of acute and chronic affections of the kidney and liver, becoming predominant in the stage of developing failure with the clinical picture of uræmia or cholæmia. *Renal disease* will not be overlooked if a routine examination of the cardio-vascular system and urine is made, supplemented if necessary by estimation of the blood urea, while in surgical cases the possibility of secondary renal failure due to progressive urinary obstruction must be considered.

In *cirrhosis of the liver* the differentiation not infrequently arises from peptic ulcer, especially with a complicating hæmatemesis or melaena. In the former the type of dyspepsia is that of a chronic gastritis, with loss of appetite, flatulence, morning vomiting, salivation, and alternating diarrhoea and constipation, while the facies shows a subicteric muddy hue, with prominent venules, injected watery conjunctivæ, furred tongue and heavy breath. The liver and spleen are commonly enlarged with characteristic feel, and, later, ascites may confirm the diagnosis. The onset of cholæmia is marked by wasting and prostration, complete anorexia, incessant vomiting, and the development of convulsions and coma with hæmorrhages and deepening jaundice.

Efficient digestion of food is naturally dependent upon a healthy condition of its portal of entry. Considerable attention in cases of dyspepsia is therefore to be paid to the state of the *mouth and throat*. The protruded tongue is inspected, noting whether it is clean or furred, dry or moist, whether the papillæ are normal in appearance, injected or missing, and whether tremor or any soreness or ulceration is present. In debilitated conditions opaque white plaques of thrush may be found. In many dyspeptic states small aphthous ulcers may be seen on the tongue and inner aspects of the cheek.

The presence or deficiency of *teeth* is noted and the healthiness or otherwise of the *gum-margins*. *Root absorption* and gross *pyorrhœa* are often associated with a chronic gastritis due to swallowed pus. *Dentures* should invariably be removed and the mouth inspected for neglected stumps beneath.

An examination should be made of the *fauces* for enlarged or septic tonsils, and, if indicated, of the nose for obstruction or signs of rhinitis or sinus infection. Hoarseness or stridor of the voice may signify possible pressure on nerve trunks, and calls for a confirmatory examination of the larynx and a testing of pupil reactions.

In examination of the *neck* the significance of thyroid swelling or prominence of veins has already been mentioned. In suspected malignant disease *enlargement of lymphatic glands* should be searched for, particularly above the left clavicle. This group of glands is especially prone to become involved in carcinoma of the stomach by upward spread along the thoracic duct, forming hard, fixed and painless nodes. Carcinoma of the *œsophagus* may first make itself shown by enlargement on one side of a deep group of glands near the middle of the neck.

The *chest* is examined as already outlined for any abnormal signs in the heart or lungs; cough is noted, with any special features it may possess, and a record made of any undue prominence of veins on the chest wall.

(2) *Physical examination of the abdomen.* For the full elicitation of physical signs two criteria are essential—a correct position of the patient, and comfort on the part of the examining surgeon. Moreover, the examination must be carried out in a good light. For inspection of the abdomen the patient should be lying recumbent, flat on his back, with the body straight and abdominal wall relaxed. The bladder should be previously emptied, if necessary by passing a catheter. Inspection should be carried out along both axes of the body, from the end of the bed, and from the patient's side if minor degrees of asymmetry are to be observed.

*Inspection at rest.* Much may be learned from the general contour and symmetry of the abdomen at rest. In health the abdomen should be neither unduly prominent nor retracted, and free from any local bulging. A symmetrical enlargement of the abdomen as a whole may have its origin in the abdominal wall or in the subjacent contents. The commonest reason of protrusion in the former instance is *obesity*,

usually made obvious by the patient's general state of nutrition. Moreover, the fat is evenly spread over the abdominal wall obscuring the rib margins and iliac crests, while the umbilicus retains its normal central position, but is sunken and depressed by its attachment to subjacent fasciæ.

The commoner causes within the abdomen of a general enlargement readily recognised on inspection are distension of the peritoneal cavity by fluid (*ascites*), and *flatulent distension* of the stomach and intestines, neither of which conditions needs detailed description here. It may be noted that a not inconsiderable amount of fluid may collect in the adult abdomen without materially altering the external appearance, and its detection will then depend on other methods of examination. A larger collection produces the characteristic bulging, chiefly in both flanks, while with increasing amount the abdomen becomes greatly distended and uniformly rounded and immobile, with flattening or eversion of the umbilicus, a tense shininess of the skin, and prominence of veins coursing vertically in the abdominal wall.

Flatulence, on the other hand, even when due to intestinal obstruction, seldom produces the degree of distension seen with ascites. Bulging in the flanks is less apparent, and other points of differentiation, as described later, make the distinction an easy one.

*Retraction of the abdominal wall* in dyspeptic states is found in the wasting of malignant disease (apart from obstruction or ascites) and in conditions of dehydration following acute or chronic diarrhœa, e.g. gastro-enteritis of infants or ulcerative colitis.

Other general points to be observed on inspection of the abdomen at rest include the presence of striae, indicating past or recent distension according to their colour, and the position and shape of the umbilicus, whether displaced upwards, downwards or laterally, or distorted in appearance by being tethered to subjacent organs. Any discharge or excoriation should also be noted. If distended surface veins are present the direction of blood-flow should be determined. The distinction usually to be made is that of portal from inferior vena caval obstruction. In the former case the veins commonly radiate from around the umbilicus, forming a collateral circulation via the round ligament. Obstruction to the inferior vena cava is compensated for by prominence of the inferior epigastric veins and often also by a large vein running upwards in the mid-axilla to join the superior vena cava. Local pigmentation of the abdomen apart from a general increase is not without significance. It results not only from pregnancy but often also from abdominal tuberculosis or neoplasm.

A *local swelling of the abdomen*, evident sometimes on inspection alone, may, like a general enlargement, have its origin either in the parietes, or more often in the abdominal cavity. As the determination of their nature must include consideration of other features, the character and differential diagnosis of such swellings will be considered later.

### *Abdominal movements.*

(a) *Respiratory movements.* Normally the abdominal wall shows a regular range of movement with respiration, greater in its upper zone, rising evenly with the inspiratory descent of the diaphragm and falling again with expiration. In diaphragmatic paralysis a reversed or paradoxical movement occurs, the epigastrium being sucked in with inspiration and blown out with each succeeding expiration.

A normal range of movement is strong evidence against the presence of any acute inflammatory lesion affecting the underlying segment of the abdomen, and, conversely, a localised limitation or absence of movement will arouse suspicion of such a possibility. Thus, where applicable, a critical comparison of the degree of movement on the two sides and of the upper and lower zones becomes of great significance. Involvement in acute inflammation of the peritoneum above all structures causes complete abolition of movement. Distension of the abdomen alone, as by ascites, may be so great as to prevent any degree of movement on breathing. As referred to later, the relation to respiratory movement of any localised abdominal swelling must be carefully noted.

(b) *Movement of the abdomen apart from respiration* may be due to abnormal pulsation in its upper part or to exaggerated peristaltic action of stomach or intestines. The normal abdomen does not pulsate. In thin nervous women there may be marked throbbing in the epigastrium of the abdominal aorta, sometimes sufficiently prominent to be mistaken for a gastric tumour or aneurysm. Its impulse slightly to the left of the mid-line, traceable usually down to the bifurcation of the aorta, will prevent error. True expansile pulsation is very rare and is due to aneurysmal dilatation of the abdominal aorta. Not infrequently a transmitted pulsation may be conveyed to the abdominal wall from the aorta through an overlying tumour of whatever origin, when the knee-elbow position may be needed to distinguish its true nature. Again, epigastric pulsation is frequently seen in hypertrophy or dilatation of the right ventricle. Very rarely, the liver itself may pulsate in congestive heart failure.

*Visible peristalsis* may arise from the stomach, or from the small or large intestine. It is the outward sign of more forcible contraction, and in long-standing cases muscular hypertrophy, of the gut-wall to overcome a distal obstruction, partial or complete. The ease with which the contractions are seen depends also upon the thickness of the parietes, being most easily observed in a thin-walled abdomen. Visible gastric peristalsis whether in the infant or adult always indicates an organic obstruction, usually of the pylorus. It is often early in appearance, before marked dilatation of the stomach develops, whilst the tone of the hypertrophied muscle coats is still good and successfully overcoming the obstruction. Its elicitation is helped by stimulation of the overlying abdominal wall by flicking or stroking the skin along the left costal margin, and by the giving of food or a drink immediately before examination. A rounded prominence is seen arising from under the ribs and travelling slowly and uniformly like a ball from left to right across the abdomen, at a level varying with the position and degree of dilatation of the stomach. As each wave reaches the pylorus a further prominence may be seen to begin under the costal arch. If the peristaltic waves are marked they may be accompanied by audible gurgling and by spasms of colicky pain.

Coils of obstructed small intestine are often seen to stand out prominently across the centre of the abdomen in transverse ridges, likened to the steps or rungs of a ladder, which, while retaining the general pattern, are individually changing in shape and movement. In large-bowel obstruction the swelling of the abdomen as a whole is greater and more obvious in the flanks than in the centre. Peristalsis of that portion proximal to the lesion is seen as waves of movement beneath the abdominal wall, beginning at the caecal region and travelling in rapid progress clockwise around the periphery of the abdomen. By noting the direction of the waves as they cross the abdomen transversely the distinction of gastric peristalsis from that in a distended and obstructed transverse colon can readily be made.

*Visible abdominal tumour.* As with a general enlargement, a localised swelling of the abdomen may be situated in the abdominal wall or arise from within the peritoneal cavity. In the former case it can be picked up with the parietes and on breathing shows no movement relative to the abdominal wall. Such a superficial swelling may be a lipoma or a secondary nodule of carcinoma. If in the skin or subcutaneous tissues, its ease of palpation is not affected by voluntary contraction of the recti. The possibility of hernia must be considered

and the relation of the swelling determined to the common sites—inguinal, femoral, or umbilical—together with any pulsation on coughing and reducibility within the abdomen.

If the swelling arises from an intra-abdominal organ which normally descends with respiration, as from the stomach, kidney, spleen, liver or gall-bladder, and if it be not fixed by adhesions or inflammatory reaction, it is seen to move vertically up and down under cover of the abdominal wall. Tumours arising in the lower abdomen, as from ovary, uterus, or bladder, are immobile on breathing.

The appearance of the swelling may of itself be so characteristic as to proclaim its nature. Thus, the rounded outline of a distended gall-bladder may be identified, while a dropped and dilated stomach, especially if it be obstructed also, may be clearly outlined beneath the abdominal wall, with a characteristic hollowing above and below its borders. This appearance is accentuated with the patient standing. Enlargement of any organ in the upper abdomen should, if the general condition permit, be searched for in this position. If normally hidden high up under cover of the costal arch, the swelling may then through the added downward weight of the abdominal contents become visible or palpable. Moreover, a better general idea of the tone of the abdominal muscles is obtained by examination in both postures.

*Palpation and percussion.* Palpation of the abdomen should be carried out with warm hands, the surgeon being seated comfortably by the side of the patient, who should be recumbent with the abdominal wall relaxed as far as possible by drawing up the knees.

A general idea should first be obtained, by superficial examination, of the muscular tone of the abdomen as a whole and of each quadrant in turn. Any divarication of the recti is noted. Having estimated the general "feel" of the abdomen, special attention should be paid to any area of pain by examining for hyperæsthesia of the skin and localised muscular rigidity and tenderness. This should be followed by deep palpation to determine the presence or absence of deep abdominal tenderness, and of enlargement of each viscus in turn, or the presence of a tumour or free fluid.

*Superficial abdominal reflexes.* These are elicited by drawing a sharp object such as a pin lightly over the skin of the abdomen on each side in turn, parallel to the costal arch and to Poupart's ligament. Normally the underlying quadrant of the *rectus muscle* gives a quick contraction. Their loss is of most significance in organic lesions of the nervous

system. Unless the abdominal wall be of good tone they may, in health, not be obtained, and their significance in acute abdominal diseases is slight and uncertain. Sometimes with an underlying inflammatory lesion the reflex over the affected segment is absent; at others an increased response is obtained. No great diagnostic import, therefore, is to be attached to this sign alone.

*Protective response of the abdominal wall.*

The protective response of the abdominal wall to an inflammatory lesion of a viscus within its cavity gives rise to a group of physical signs of varying diagnostic import. These have so much in common in their mode of production and demonstration that in this regard they are better considered in conjunction than as separate entities. They include the questions of *cutaneous hyperæsthesia*, *muscular rigidity*, *deep visceral tenderness*, and *reflex tenderness* of the abdominal wall. Special consideration is given to their occurrence in peptic ulcer.

For their correct interpretation the mode of production of *visceral pain* must first be briefly discussed. The invaluable work of Head and Hurst has shown that the local seat of origin of all gastric sensations lies in the muscular coat, and that not only pain but also normal sensations of hunger and repletion are dependent upon the activity of peristalsis and degree of intra-gastric tension. The mucosa itself, whether intact or ulcerated, is an insensitive structure, and the pain of uncomplicated ulcer is due, not to the passage over it of irritating food, but to spasm of the pyloric sphincter or of the muscular wall of the stomach opposite to the ulcer area, with the added factor in some cases of violent peristaltic contractions.

The presence in the stomach of a lesion such as an ulcer leads to a constant stream of abnormal impulses originating in the sensitive muscular coat and conveyed by afferent sympathetic fibres to the spinal cord, where they set up an irritable focus in the affected segments. The resulting pain is referred not to the stomach itself, but to a corresponding part of the abdominal wall whose somatic nerve supply arises from this same unduly sensitive area of the cord. Moreover, normal stimuli of touch or pressure applied to the skin or muscles of this part of the abdominal wall are no longer interpreted as such, but produce discomfort or actual pain. The postural tone of the abdominal muscles, being dependent upon a reflex arc, is locally increased in view of the irritable spinal centre, and a further degree of protective spasm occurs on external pressure. Thus arise a group of phenomena of referred pain, reflex hypersensitiveness of the skin, reflex tenderness

and muscular rigidity. In the case of the stomach the area of the cord affected is nearly always the seventh, eighth and ninth dorsal segments. Each viscus has its own characteristic surface zone.

The site of reference on the abdominal wall remains constant despite the changing position of the stomach resulting from ptosis, respiratory movement or alteration of posture, and may even be completely outside the gastric area. As X-ray examination will prove, pain and tenderness are not felt exactly over the site of the ulcer, and do not vary in position with its situation in the stomach.

These signs are often, but by no means constantly, present, and must be considered in conjunction with other evidence. Their absence cannot be taken to exclude ulceration, for uncomplicated peptic ulcer is often present without local tenderness or rigidity.

#### *Cutaneous hyperæsthesia and hyperalgesia.*

Of the reflex signs in chronic abdominal disease hypersensitivity of the skin is the least constant and reliable; its presence and distribution are of much more value in acute conditions. It may be tested for either by lightly pinching the skin or by stroking down the trunk on either side with the point of a pin. If a sensitive zone be found it is wise to map out its borders by drawing the pin from the normal to the sensitive area. Allowance must be made for undue nervousness of the patient and care taken to avoid suggestion, for in such subjects hyperæsthesia is often found in the absence of any organic disease.

#### *Reflex tenderness.*

The site of reflex tenderness of the structures of the abdominal wall usually corresponds with the reference of spontaneous pain. In the case of gastric ulcer it is usually in the mid-line of the epigastrium immediately below the xiphisternum. With a deep ulcer boring into the pancreas there is sometimes pain and tenderness over a small localised area to the left of the spine. In duodenal ulcer tenderness is more often felt about an inch and a half to the right of the mid-line, in the subcostal plane. Deep tenderness of the muscles is generally more marked in ulcer than hypersensitiveness of the overlying skin, and of the two is the much more reliable physical sign. As well as the muscle fibres the connective tissues of the abdominal wall are also sensitive, as is seen in the tenderness to pressure over the linea alba in cases where the recti are widely divaricated. Moreover, voluntary contraction of the rectus muscles diminishes the tenderness on pressure, and suggests



that the sensitive zone may be at a deeper level in the subperitoneal cellular tissue which is richly supplied with sensory nerve endings.

*Reflex muscular rigidity* is found as a protective spasm over the zone where deep tenderness is present. If the lesion be a gastric ulcer there is some degree of increased resistance of one or both upper recti, usually rather more on the left. With duodenal ulcer the upper half of the right rectus muscle is somewhat on guard, but the side affected is no certain guide.

Referred tenderness and rigidity, although most marked and of widest distribution when the spontaneous pain of an ulcer is severe, are not dependent upon it, but are often found in the intervals between bouts of pain or after pain has ceased. They are, however, some guide to the persistence of ulceration, for they are frequently found to lessen steadily with the progress of healing and disappear when the ulcer is replaced by a scar. Reflex rigidity is involuntary, and of these signs is the last to disappear. On the whole, the deeper the ulcer crater penetrates into the muscle coats the better marked are the referred physical signs.

As with tenderness and hyperæsthesia, reflex rigidity must be distinguished from the voluntary tightening of the abdominal wall commonly met with in very nervous patients whenever an attempt at palpation is made. This type of resistance is usually overcome by diverting the patient's attention from the examination.

When pain in the gastric area is produced in response to a lesion in a distant area of the alimentary canal, as in the referred pain of appendix dyspepsia, these reflex signs of soreness of the skin and deep muscular tenderness are either absent or much less marked than with a gastric lesion, although the type of pain may be identical. Some guide in differential diagnosis is thus afforded as to the likely site of disease. Pain in the epigastrium often results from pressure in the right iliac fossa over an inflamed appendix, but the upper abdominal wall is soft. The same principle applies, to a less extent, to lesions in the thorax productive of abdominal pain. Abdominal rigidity due to thoracic disease is usually found to lessen with continued palpation, while with an abdominal cause it grows more marked, with increasing tenderness, the more prolonged and deep the examination.

### *Visceral tenderness.*

The peritoneum, in contra-distinction to the mucosa, is sensitive to direct pressure. When the peritoneum overlying an ulcer becomes involved in inflammation or adhesions, then tenderness and rigidity are

found directly overlying the affected area. This local or visceral tenderness, unlike the reflex type, changes its position with each movement of the stomach, and is usually deeper and more localised. In carcinoma of the stomach, owing to the more frequent and wider involvement of the peritoneum, local tenderness over the lesion is much more common than with an innocent ulcer. Again, where perigastritis occurs from leaking of the contents in simple ulcer, the resulting abdominal rigidity and tenderness are far more marked than in the absence of complications. Any intra-abdominal lesion, such as a perforation, causing acute irritation of the peritoneum produces extreme tenderness and an immediate protective spasm of the muscles of the overlying abdominal wall, with board-like rigidity in the initial stages. Peptic ulcer never produces such extreme rigidity of the abdomen unless complicated by peritonitis.

Rigidity and tenderness of the abdominal muscles thus develop rapidly over any acute inflammatory intra-abdominal lesion, while their degree and the quadrant affected depend upon the organ at fault and especially upon the rapidity and extent of peritoneal involvement. In the right upper quadrant, for example, affections of the duodenum, liver, gall-bladder, and colon, and their possible complications, have to be considered. With a rapid outpouring of irritant fluid, as in intra-peritoneal hæmorrhage or perforation, there is exquisite tenderness and extreme muscular rigidity, while the slightest unnecessary movement, even of breathing, is avoided. Tenderness is marked not only on pressure but equally on the release of that pressure by the fingers. With the spread of peritonitis there is an advance of rigidity and tenderness situated exactly over the seat of inflammation. This is an observation of value—for example, as evidence of the tracking down of fluid from the upper abdomen into the right iliac fossa. Where an inflammatory lesion is confined to the pelvis, although the peritoneum be extensively involved rigidity and tenderness are slight or absent on palpation of the abdomen but marked on rectal examination.

When peritonitis is general the whole abdomen is in the initial stages board-like. Such a degree of spasm cannot, however, be kept up indefinitely; in course of time fatigue of the neuro-muscular reflex occurs, accentuated by the increasing shock and general toxæmia, and the abdominal wall again becomes lax though still tender. In elderly people does this particularly apply, and its importance in diagnosis and treatment needs no stress.

Tenderness is a sign of value in the differential diagnosis of obstruction from peritonitis. In both conditions pain, vomiting, distension

and constipation are prominent; if due to intestinal obstruction alone, uncomplicated by peritonitis, rigidity and tenderness are absent.

*Deep palpation : Palpable abdominal tumour.*

After testing for rigidity and tenderness, the abdomen is examined systematically by deep palpation for enlargement of any organ or presence of any abnormal tumour. Of greatest importance in cases of dyspepsia are swellings in connection with the stomach, which will be considered first.

The commonest palpable tumour originating in the stomach is a carcinoma. Arising most often at the pyloric end, when of sufficient size it is felt as a firm, often irregular and tender mass above and to the right of the umbilicus, usually freely mobile, moving up and down on deep respiration beneath the flat of the hand held across the middle of the abdomen. As the mass grows it becomes more irregular and warty, and by forming adhesions to surrounding viscera is less freely mobile. It may by direct spread become continuous with the liver, or the main mass felt may be composed of secondary glands along the greater or lesser curvature. Anchoring of the umbilicus may be noted. Pyloric obstruction with its associated signs frequently develops.

A pyloric tumour has to be distinguished from swellings arising from the gall-bladder. The swelling in the former case is resonant and, at least in the early stages, is separated from the liver by a band of tympany.

Carcinoma arising from the body of the stomach is usually less mobile than a pyloric growth. In either case, the direction of local spread tends to be upwards through the stomach wall towards the cardia. Fixation of the body of the stomach, in the later stages, to any surrounding structure may limit or prevent movement. Finally, large irregular masses may be felt in the abdomen due to direct infiltration of omentum, mesentery and lymphatic glands.

The aim of diagnosis should be to detect gastric carcinoma in the early stages before an abdominal tumour becomes palpable. On the other hand, it is not true to say that when the growth has become palpable per abdomen it has necessarily passed the stage for radical removal. A swelling is most readily detected when the stomach is empty. In any doubtful case the examination should be repeated under an anæsthetic, to relax the protective spasm of the upper recti. With any suspicion an X-ray examination also will, of course, be indicated.

Inflation of the stomach with gas through a tube or by giving

separately the two halves of a Seidlitz powder is seldom used at the present time, as it produces discomfort, is not absolutely free from risk, and much more exact and reliable information can be obtained by X-rays.

Special search, as already mentioned, should be made for enlargement of the liver or left supraclavicular glands and for ascites. Other late complications include the rare event of perforation of the growth with local peritonitis or formation of fistulæ especially into the transverse colon, setting up diarrhœa and fæculent vomiting.

In the uncommon so-called "leather-bottle" type of carcinoma of the stomach the whole organ becomes infiltrated with densely contracting growth, and is readily palpable as a firm, shrunken tube-like structure lying across the left upper abdomen.

Slow leakage into the peritoneum from a simple peptic ulcer may produce an inflammatory mass or perigastritis, which closely resembles a neoplasm of the stomach, and is only distinguished with certainty by exploratory operation. Usually the mass is less firm and less mobile owing to early fixation by inflammatory adhesions, while tenderness is more marked. Apart from such complications a simple ulcer does not produce an abdominal tumour.

A cardinal sign in the diagnosis of congenital pyloric stenosis, considered in detail in a later chapter, is the presence of palpable enlargement of the hypertrophied sphincter as a small firm rounded swelling to the right of, and above, the umbilicus.

*Percussion* over the stomach yields a high-pitched tympanitic note, but is not a physical sign of great accuracy or precision in mapping out its borders. The combined percussion-auscultation method again proves unreliable as an index of the size of the stomach or other hollow organs. Splashing over the region of the stomach if elicited three hours or more after the last meal is evidence of dilatation.

Other swellings in the upper abdomen to be considered in cases of dyspepsia and to be distinguished from gastric swellings include enlargements of the liver, spleen, kidney or suprarenal, gall-bladder, tumours or cysts of the pancreas, and tumours of the colon.

In relation to every abdominal tumour the following characteristics have to be considered :

1. Situation.
2. Size.
3. Shape.
4. Surface.

5. Consistency.
6. Tenderness.
7. Fixity or mobility.

From a consideration of these features a diagnosis can usually be made with a tolerable degree of certainty.

*Enlargement of abdominal viscera of significance in dyspepsia.*

1. *Liver.* In the adult the lower border of the liver is not normally palpable, the tone of the abdominal muscles preventing its being felt on inspiration. The observer sits on the right side of the patient, pushing forward the loin from behind with the left hand and feeling with the right below the costal arch in the nipple line. If the liver be slightly enlarged, its lower border will be felt to descend beneath the fingers of the right hand on deep inspiration. If great enlargement of the liver is possibly present, examination for its lower border must be begun low down in the right iliac fossa and continued upwards. Many enlargements have been missed by neglecting this procedure. If the liver be felt, the following points are to be noted—the degree of enlargement, the character of the edge—whether sharp or rounded—of its anterior surface—whether firm or soft, uniformly smooth or irregular, with the degree and extent of any irregularity present. Tenderness or pulsation will also be noted. Palpation will be confirmed by percussion of the upper and lower limits of liver dullness, both in the mid-line and in the right mid-clavicular line. Any swelling arising from either liver or gall-bladder is, of course, dull to percussion, the dullness being continuous with that of the liver.

It must be remembered that the normal liver may be rendered palpable from the downward pressure of disease within the thorax, for example emphysema or a large empyema, as well as by any collection of fluid between its upper aspect and the diaphragm. The causes of hepatic enlargement are very numerous, and only those which may be significant in dyspepsia and gastric diseases are considered in this section. Of most significance in this regard are secondary malignant disease and cirrhosis.

Enlargement by carcinomatous deposits may be of any degree and may even become obvious to the naked eye by widening of the costal arch; the edge is firm and irregular, while the surface has a hard nodular feel, showing a varying number of firm bosses in some of which umbilication may sometimes be detected. The liver is often tender, while ascites and jaundice are frequent but not invariable. Malignant

enlargement of the liver is not always general, for that portion of the organ adjacent to the primary lesion may be involved locally in a direct spread of growth, as from the stomach, gall-bladder or colon. It is then difficult before operation to define how much of the palpable swelling is due to liver enlargement and how much to the primary growth.

The liver of multilobular cirrhosis is not always enlarged, and even if so not always palpable. When it can be felt it has a sharp edge and firm consistence; in most cases the individual granulations are too small to be identified. Friction due to perihepatitis can sometimes be felt or heard over the anterior surface, and evidence of portal obstruction is present.

2. *Spleen.* The mode of palpation for enlargement of the spleen is the same as for the liver, but on the left side of the abdomen. In enlarging, the spleen extends downwards, forwards and inwards from under the left costal arch towards the umbilicus, immediately beneath the anterior abdominal wall, guided by the costo-colic ligament. It therefore descends in front of the splenic flexure of the colon. The hand cannot be passed between the swelling and the costal margin, a point of help in differentiation from a renal tumour. The surface is usually smooth and uniform and its sharp anterior border, with one or more easily felt notches, is retained. There is dullness on percussion, with a zone of resonance posteriorly between the swelling and the spine. The dullness of a renal tumour, on the other hand, is continuous behind with that of the vertebral column, and anteriorly is often traversed from above downwards by a band of resonance due to the descending colon.

Enlargement of the spleen of possible significance where the complaint is of dyspepsia occurs in splenic anæmia and alcoholic cirrhosis, in both of which a hæmatemesis may simulate peptic ulcer. The weight and pressure of the gross enlargement in chronic leukæmia often produces initial symptoms of dyspepsia. As over the liver, friction due to local peritonitis of the capsule may sometimes be felt or heard and is productive of pain.

3. *Kidney and Suprarenal.* Enlargement of the kidney is searched for by bimanual palpation of the loin on deep inspiration. Ptosis without enlargement is often found, especially in thin women, when either the rounded lower pole or the whole organ may be palpable. The kidney lies against the posterior abdominal wall and can, if displaced, always be

returned into the loin. Enlargements or tumours of the kidney, although they may become of great size, usually retain the shape and main features of the normal organ. Suprarenal tumours, on the other hand, while conforming in position and general features to renal swellings, are not so restricted in shape and may form very large swellings.

4. *Gall-bladder.* The gall-bladder is most readily felt when its walls are healthy and it is distended with bile. It then forms a tense globular swelling projecting from below the edge of the liver in the right nipple line downwards and forwards towards the umbilicus. It never extends in an outward direction and can thus be distinguished from other cystic swellings. Examination in the knee-elbow position will sometimes render palpable a gall-bladder not otherwise felt. These conditions are found at their best in obstructive jaundice caused by pressure from without on the common bile-duct as by a growth of the head of the pancreas. As the gall-bladder is not inflamed no tenderness is present on palpation. In chronic cholecystitis with gall-stones the gall-bladder can seldom be defined, as its walls are thickened and shrunken and often adherent to other structures. Tenderness and rigidity are often found over the tip of the ninth right costal cartilage, brought out in chronic cases by palpation on deep inspiration, when as the tender gall-bladder passes under the fingers a momentary catching of breathing (Murphy's sign) occurs. Even when deep jaundice is present from obstruction of the common bile-duct by a calculus, enlargement of the gall-bladder is seldom evident. Blockage of the cystic duct, however, may lead to a large tense elastic swelling—mucocoele of the gall-bladder.

A primary carcinoma of the gall-bladder is not often palpable but may cause a hard irregular mass continuous with the liver.

5. *Pancreas.* On account of their depth in the abdomen pancreatic tumours can seldom be felt, but usually declare themselves by other symptoms. A carcinoma, however, can sometimes be detected as a hard ill-defined immobile mass lying deep down across the abdomen above the level of the umbilicus. An intense degree of obstructive jaundice is present. A cyst of the pancreas is also fixed in position and usually extends forward as a rounded swelling between the stomach above and the transverse colon below.

6. *Colon.* Swellings arising from the colon, especially in its transverse course, frequently have to be diagnosed from gastric tumours.

Carcinoma of this region forms a hard, often tender, rounded or tubular swelling, superficial in the abdomen, readily moved about by the hand, but immobile on breathing. A carcinoma of the ascending or descending colon lies to the side along the normal course of the bowel and has a limited degree of lateral mobility. Growths of the flexures are usually obscured under cover of the costal arch. Seldom does a carcinoma of the large bowel attain the large size met with in gastric growths. With any doubtful swelling in the course of the colon the possibility of its being due to scybalous faecal accumulation must first be excluded by enemata. Such masses may sometimes be recognised to pit on pressure.

*Other abdominal swellings* felt in cases of dyspepsia include those arising from infiltration of glands in the mesentery or behind the peritoneum by growth, or less often by tuberculosis. In abdominal tuberculosis error may arise from contraction and rolling up of the omentum to form a sausage-shaped tumour lying transversely across the upper abdomen. The hyperplastic form of tuberculosis of the caecum is also readily mistaken for neoplasm.

*Auscultation* in abdominal conditions is sometimes of value in diagnosis. Friction may be heard by the rubbing together of roughened and inflamed peritoneal surfaces. The detection of brachyrgmi over an abdominal swelling proves its connection with the stomach or gut; rarely the hearing of a bruit over a swelling will suggest its aneurysmal nature. In the case of the oesophagus a delay in swallowing can sometimes be detected by listening with the stethoscope to the left of the trachea in the neck or along the side of the vertebral spines during deglutition, to the right in its upper half and to the left in its lower.

*Ascites.* The appearance of the abdomen when distended with free fluid has already been described. Its presence can be confirmed by palpation, when a sense of general resistance is felt. By a quick dipping movement a characteristic sense of displacement of fluid beneath the fingers is obtained and subjacent organs such as the liver felt beneath. A "fluid thrill" is felt on flicking the abdominal wall on one flank and palpating the opposite site of the abdomen with the other hand. On percussion, if the fluid is large in amount both flanks are stony dull and resonance remains only over the anterior abdominal wall in its upper part, where the stomach and intestines float up on their mesenteric attachments. When the patient turns on to one side, the opposite flank being uppermost becomes resonant on percussion,



returning to dullness when the side of recumbency is reversed (shifting dullness).

If small in amount, the presence of fluid is apparent only when examined for in the knee-elbow position. An area around the umbilicus, previously resonant, now becomes dull to percussion.

Clinically, the onset of ascites may be suggested by the filling with fluid of latent hernial sacs, especially in the groins—a primary hydrocele being first suspected. The main cause of confusion in the diagnosis of ascites is the sometimes difficult distinction from large encysted collections of fluid, especially ovarian cysts, a distended bladder, and occasionally a hydronephrosis or large hydatid cyst. The difficulty is greater when the ascitic fluid is loculated by adhesions within the peritoneal cavity, as is not infrequent in tuberculous peritonitis.

Further information is gained from removal by paracentesis of a specimen of the ascitic fluid for pathological examination, especially for cytology and protein content. Often, by further palpation immediately after the fluid has been partly or wholly drained and the abdominal wall is still relaxed, a more accurate indication of the subjacent abdominal condition can be obtained. By far the commonest cause of ascites in childhood and early adult life is a tuberculous peritonitis. In middle or later life cirrhosis of the liver, heart failure, nephritis, and involvement of the peritoneum by carcinoma are the common causes.

*Rectal examination* should never be omitted in the investigation of any abdominal case. The anal region is first inspected for external hæmorrhoids, fistula or anal fissure. On digital examination the presence is detected of faecal masses or of growth either of the rectum itself or pressing on it from without, and in the male any pathological enlargement of the prostate determined. If a tumour or inflammatory mass is detected in the pelvis an estimate of its position, size and mobility can be obtained. In the diagnosis of acute abdominal conditions the importance of tenderness of the pelvic peritoneum cannot be over-emphasized. In such a case the presence of pelvic peritonitis or intra-abdominal hæmorrhage can be detected by recognising a collection of fluid in the recto-vesical or recto-vaginal pouch, which becomes thickened and tender on pressure. The value of rectal examination in the diagnosis of appendicitis has already been mentioned.

In chronic cases where the possibility of malignancy is present, search should always be made for a deposit of growth in the peritoneum or lymphatic glands in this area. The presence of blood or mucus on

the examining finger after withdrawal should be noted. In some cases proctoscopic examination may be needed.

*Vaginal examination* will, in special cases, supplement the information obtained on rectal examination, in particular if there be the possibility of a primary growth in the uterus or adnexa. The not infrequent occurrence of secondary deposits in the ovary or pelvic peritoneum from a colloid carcinoma of the stomach should be remembered before a radical operation is carried out.

## CHAPTER IV

### BIOCHEMICAL INVESTIGATIONS

(1) Gastric Analysis.

(2) Occult Blood.

by

P. M. DEVILLE

(3) Examination of the Urine and Tests for Renal Efficiency.

(4) Wasserman and Kahn reactions for Syphilis.

(5) Complete Blood Count.

(6) Duodenal Intubation.

by

R. SLEIGH JOHNSON

#### (1) GASTRIC ANALYSIS

THE analysis of the gastric contents, together with a knowledge of the secretory power of the stomach, affords considerable aid in the diagnosis of a case of suspected gastric or duodenal disease. Its value is not, however, confined to primary diseases of these organs as it is of great assistance in the diagnosis of other conditions, such as pernicious anæmia and simple achlorhydric anæmia. The material vomited spontaneously by the patient should always be examined, but in order to obtain the maximum information resort is made to the use of test meals. The principle of the method is to give the patient an appropriate meal to stimulate gastric secretion, and to withdraw the contents of the stomach, for examination, by means of a tube during the process of digestion. The information obtained is not in itself diagnostic, except in those extremely rare cases where malignant cells are demonstrated in the material withdrawn; but when taken in conjunction with the history, clinical examination, and the results of the other means of investigation at our disposal, it gives considerable assistance.

## TUBES USED TO WITHDRAW GASTRIC CONTENTS

Before describing the various test meals and the information which they give, it is well to have some knowledge of the tubes that may be used to withdraw the gastric contents. A large number of these have been used from time to time, but they all have one feature in common, in that they are made of soft rubber with openings at their ends. They differ mainly in their diameter, and in the size and shape of the openings at the gastric ends, some being "weighted" with metal at their lower ends to facilitate swallowing. Only the more usual types will be briefly described here.

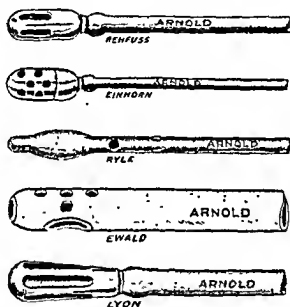


Fig. 40.—SOME VARIETIES OF STOMACH TUBES.

(1) *Ewald Tube*. This is a large diameter tube, with terminal and large lateral openings near its gastric end. It is not weighted. It may be used to withdraw the gastric contents in the Ewald One Hour Test Meal, and to wash out the stomach in adults, as in cases of poisoning. It should not be used in test meal work if there is much prostration, myocardial disease, aneurysm of the aorta, or a history of severe recent hæmatemesis. A case of perforation by the use of the tube has been reported.

(2) *Rehuss Tube*. This is a tube of much smaller diameter than the Ewald tube. It possesses a hollow metal terminal with vertical slots, as shown in figure 40; and the diameter of the slots is the same as

that of the tube, so that any material passing through the slots can also pass up the tube.

(3) *Ryle Tube.* This is the best tube to use for fractional test meal work and is the one usually employed in this country. Like the Rehfuß tube it is made of soft flexible rubber of small diameter, and is weighted. Just above its blind lower end it has a smooth oval lead weight inserted, above which are the rounded openings through which the gastric contents are aspirated. The lead weight cannot become detached as it is completely covered with rubber, which also forms a cushion and diminishes trauma to the gastric mucous membrane. The stem of the tube is marked by three sets of lines. The single line represents the average distance from the front teeth to the cardia; the double line the distance to the fundus, and the three-line mark the distance to the pylorus. In this way one is able to gauge with fair accuracy the position of the tube relative to the different parts of the stomach. Nevertheless, as variations occur in the size and position of the stomach, it follows that these marks only represent average distances, and that allowance may have to be made for these factors in individual cases. The position of the tube can, however, be accurately determined by means of X-rays. The metal end shows up well, and a slight amount of barium in the stomach suffices to outline it sufficiently well for this purpose. In practice, however, this is unnecessary, and all that is required is that the end of the tube should lie in the body of the stomach, and remain in the same position during the investigation; for the average case the marks indicate the position with sufficient accuracy.

Various other types of tubes are shown in the diagram (see fig. 40).

#### TECHNIQUE OF PASSING THE TUBES

The large tubes, such as Ewald's, must be passed by the physician. The patient should, if possible, be sitting up with the mouth wide open, the head in the normal position looking forwards, and not thrown backwards. All dentures must be removed. The tube, after boiling, is gripped in the right hand, near its gastric end, and directed through the patient's mouth into the pharynx. Any heaving on the patient's part may be controlled by asking him to take deep breaths through the nose. With a little more insertion the tube engages in the œsophagus, and by means of further gentle pushing rapidly enters the stomach. The tube is removed by traction. The small-bore tubes (Rehfuß, Ryle

tubes) are best left to the patients to swallow themselves. The patient may be in the same position as for passing the Ewald tube, or he may be standing. The procedure is first explained to the patient, and after lubricating the end of the tube with a little glycerine, he then places the tip on his tongue, and swallows in the ordinary way. As the tube passes the pharynx there may be some heaving, but this is counteracted by the taking of deep breaths through the nose. Patients differ greatly in the ease with which the tube is swallowed, and psychological factors play a large part, so that nervous or apprehensive patients must be reassured, and encouraged in their efforts. The majority swallow the tube quite easily, but others will need encouragement and perseverance before this is accomplished. Cocinisation of the throat and pharynx has been used, but it is unnecessary and is not to be recommended. The tube should be swallowed so that it lies in the body of the stomach, and is kept in place by means of a piece of strapping fixed on to the cheek.

#### TEST MEALS

Several different meals have been used, but the two main types are :

1. The Ewald One Hour Meal.
2. The Fractional Meal.

*The One Hour Meal.* This is performed in the early morning between eight and nine o'clock, and nothing must have been taken by mouth during the previous 12 hours. The test meal given consists of a pint of weak tea without sugar or milk and two small slices of toast (about two ounces). The patient takes the whole of this and, exactly 1 hour after commencing the meal, the Ewald tube is passed in the manner described ; then, by lowering the external end of the tube, the gastric contents are removed by syphonage and caught in a suitable receiver. A Rehfuß tube may be used instead and the contents aspirated with a syringe or Senoran evacuator.

*The Fractional Meal.* The patient takes his usual meal the previous evening at about eight o'clock with a charcoal biscuit or 2 oz. of powdered charcoal at the end of the meal. All other drugs are suspended. Nothing is taken after this. The following morning at eight or nine o'clock the patient swallows the Ryle tube, and when it is in position a Record syringe of 20 cc. capacity is attached to the end of the tube and the whole of the fasting contents of the stomach aspirated.

The whole of the contents so obtained are placed in a suitable receiver and kept for examination. When the stomach is empty the patient is given a pint of warm gruel to drink. This is prepared as follows: Two tablespoonfuls of fine breakfast oatmeal are mixed with two pints of water, and allowed to boil slowly until the volume is reduced to a pint. The gruel so obtained is strained through muslin, and when cool is given to the patient to drink. It is swallowed quite easily with the Ryle tube still in position. The time is noted. Every 15 minutes the syringe is attached to the tube and about 15 cc. of the gastric contents, or as much as can be obtained short of this amount, is aspirated and transferred to a series of test-tubes, each bearing a label corresponding with the time that the contents were withdrawn from the stomach. At the end of  $2\frac{1}{2}$  hours, if material can still be aspirated, it should all be withdrawn and its volume noted. The tube is then withdrawn by traction. Some pathologists are content to remove specimens at  $\frac{1}{2}$ -hourly intervals; a larger volume can then be obtained, and the shape of the curves found on analysis are in general agreement.

Normally the stomach is empty in  $2\frac{1}{2}$  hours. With a hypertonic active stomach it empties more rapidly, while in pyloric obstruction the rate of emptying is very slow. As it empties it becomes more difficult to aspirate the specimens, which may be frothy.

In the majority of cases there will be no difficulty in obtaining the full series of specimens, each of 15 cc. In cases of gastric hyposecretion however, much difficulty may be experienced in aspirating the later fractions, which may measure only 3 or 5 cc. in volume.

A little water may be injected down the tube to make sure the stomach is empty and, on aspiration a few moments later, it will be found to be free from gruel. Difficulty in aspirating may also be due to the tube becoming blocked; this may be remedied by injecting air down the tube with the syringe. The tube should, of course, be tested to make sure it is patent before it is swallowed. Kinking or twisting of the tube is an uncommon cause of difficulty, but trouble may arise owing to its passing into the pylorus; this may be remedied by withdrawing it for a short distance. Again, if the tip has not entered the stomach and is in the œsophagus, no gastric contents will be obtained.

While the test meal is being performed the patient should read or talk.

Where possible the fractional test meal should always be used in preference to the one hour meal, as it gives much greater information. In the latter, the meal becomes mixed with the fasting contents of the

stomach, which vary in amount in different individuals and even in the same individual from day to day, so that erroneous deductions may be made.

#### EXAMINATION OF RESTING JUICE, SAMPLES, AND CHARTING OF RESULTS

The contents of the stomach obtained by aspiration before the meal is taken are known as the resting juice, the examination of which is of great importance as it may yield valuable information :

(1) *Volume.* This is always measured, and, although in health it varies between wide limits, if over 60 cc. are obtained this should be regarded as either due to hypersecretion or to pyloric obstruction. In the latter condition large amounts, such as one or two pints, may be obtained.

(2) *Colour.* Normally the resting juice is clear, but may be bile-stained from regurgitation through the duodenum. In pyloric obstruction bile is absent. When blood is present, except for minute traces of red, the colour is dark reddish-brown. Small traces, seen as red streaks, may be due to trauma from the tube, but larger amounts usually indicate the presence of carcinoma or ulcer. If charcoal, taken the evening before, is recovered it indicates the presence of obstruction.

(3) *Odour.* Normally odourless, in pyloric obstruction and carcinoma there may be a foul or offensive odour, especially the rancid odour of lactic and butyric acids, owing to retention of the gastric contents with fermentation and decomposition.

(4) *Consistency.* The juice is normally quite fluid, but if excess of mucus be present it becomes thick and viscid. This usually indicates chronic gastritis or carcinoma.

(5) *Presence of Food.* Normally no food remains are found. In pyloric obstruction particles of food taken the previous day or earlier are present. The presence of charcoal taken the night before has already been mentioned.

(6) *Microscopic Examination.* Normally a few epithelial cells, leucocytes, and a little mucus are seen. Red blood cells are present if there has been bleeding due either to trauma or to disease (ulcer, carcinoma), in which case large numbers, some partly disintegrated, are seen. In chronic gastritis and carcinoma excess of mucus is found.



The presence of pus cells usually indicates carcinoma, if pus swallowed from the nose, throat, and chest be excluded. Yeast cells and sarcinae are evidence of stagnation and fermentation in the stomach, and in the former condition the Boas-Oppler bacillus may be found; it occurs in obstruction due to carcinoma, but is NOT pathognomonic of it. Starch granules also signify stasis and obstruction. The findings in the resting juice and their possible interpretation may be summarised as follows (modified after Rehfuess):

Determination.	Normally.	Abnormal.	Significance.
Volume.	50 cc.	Above 70 cc.	Hypersecretion, duodenal ulcer, pyloric obstruction and pylorospasm.
Odour.	None.	Offensive.	Carcinoma.
Blood.	None.	Present.	Traces may be due to trauma of tube, gastric ulcer, carcinoma, syphilis of stomach, blood diseases, portal obstruction.
Pus.	None.	Present.	Carcinoma (exclude swallowed pus).
Mucus	Traces.	Large amounts.	Chronic gastritis, carcinoma.
Food retention.	None.	Present.	Pyloric obstruction.
Yeast cells, sarcinae, and Boas-Oppler B.	Absent.	Present.	Stasis, fermentation, carcinoma, pyloric ulcer.
Free HCl.	0-30 cc. (NaOH)	1. Above 30. 2. Absent.	1. Duodenal ulcer. 2. Congenital, chronic gastritis, syphilis of stomach, carcinoma, pernicious anaemia, gall-stones, chr. appendicitis, simple achlorhydric anaemia, sprue, etc.
Total acidity.	10-50.	Above 50.	Duodenal ulcer, carcinoma.

The fractional specimens must be examined for total acidity, free HCl, mucus, bile, blood and starch, and the results recorded on a

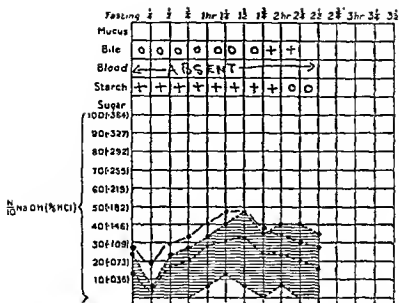


Fig. 41.—NORMAL CURVE. THE STOMACH IS EMPTY IN TWO HOURS.

chart as shown in figure 41. In this the shaded area represents the limits of free HCl found in 80 per cent of normal students by Bennett and Ryle. The fall that occurs in the first  $\frac{1}{4}$ -hour is due to the diluting effect of the meal, and after this there is a steady rise for about  $1\frac{1}{4}$  hours, followed by a fall due to the entrance of the alkaline duodenal contents; as the stomach empties the acidity gradually returns to its original level. The absence of starch indicates that the stomach is empty, and the time it takes to empty is shown by the time it takes for the starch

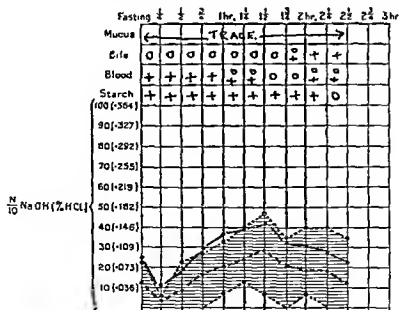


Fig. 42.—GASTRIC ULCER. THE CURVE PRESENTS NO GROSS ABNORMALITY, BUT THERE IS BLOOD IN MOST OF THE SPECIMENS.

to disappear. Normally the total acidity curve is about 10 cc. higher than the free acid curve, and roughly, but not absolutely, parallel to it.

In interpreting the results, attention must be paid to the curves of both free and total acidity, to the emptying time of the stomach, to the presence or absence of abnormal constituents, such as blood, and to the amount of mucus present.

From the surgical point of view, the results obtained are most useful in the diagnosis of ulcer and carcinoma of the stomach.

In *gastric ulcer* (see fig. 42) both the free and total HCl are within normal limits, and the curves are indistinguishable from those of a normal subject, but occasionally hyperchlorhydria is found. Blood is, however, frequently found in the specimens.

In *duodenal ulcer*, on the other hand, characteristic curves may be obtained, and are of two main types:

(1) *The Climbing Curve.* In this the resting juice is highly acid, and after a preliminary fall due to dilution, there is a gradual rise in acidity during the whole meal. The stomach empties slowly and little or no regurgitation takes place from the duodenum, so that bile is absent from the specimens. It is this lack of regurgitation, due to pylorospasm, that accounts for the continued rise in acidity.

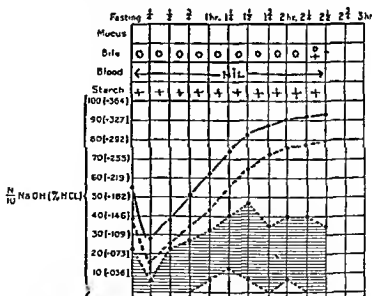


Fig. 43.—DUODENAL ULCER—CLIMBING CURVE. THE RESTING JUICE IS HIGHLY ACID AND THERE IS A CONTINUED RISE IN ACIDITY DURING THE WHOLE MEAL, WITH DELAYED EMPTYING OF THE STOMACH.

(2) *The Hurry Curve.* This type of curve is associated with a rapidly emptying stomach. The resting juice is again highly acid and

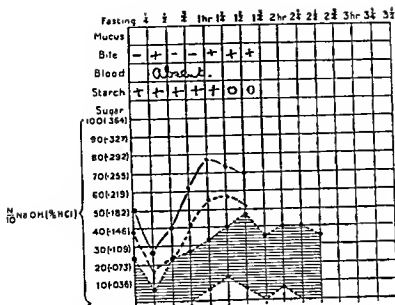


Fig. 44.—DUODENAL ULCER—THE HURRY CURVE. NOTE THE RAPID EMPTYING OF THE STOMACH, AND THE HIGHLY ACID RESTING JUICE.

after a preliminary fall the curve rises sharply, may form a plateau, and gradually falls to its original level. The stomach empties in an hour or so, as shown by the absence of starch.

Both these curves are hypersecretory in type and are characteristic of duodenal ulcer. A typical chart of each is shown in figures 43 and 44.

In *carcinoma* the typical finding is the absence of free HCl, together with the presence of blood in some or all of the specimens. This achlorhydria occurs in over 50 per cent of cases at the time when the patient first presents himself for examination, but the more advanced the growth the greater the likelihood of achlorhydria. The presence of free HCl, therefore, does not exclude a diagnosis of carcinoma, while, on the other hand, the absence of free acid is not in itself diagnostic of malignant disease, as it occurs in many other conditions, as well as in a small proportion of apparently healthy adults. The absence of HCl in cases of chronic gastritis, syphilis of the stomach, pernicious anaemia, simple achlorhydric anaemia, and sometimes gall-stones, chronic appendicitis, arthritis, asthma, etc., is well known. The surgeon is most likely to meet with it in carcinoma and chronic gastritis. In the latter, the excess of mucus fixes the free acid, but if the stomach be well washed out with hydrogen peroxide solution 3 per cent, as suggested by Hurst, before repeating the test meal, it will be found that some free acid is present. In carcinoma, the total acidity is usually also very low, but is sometimes found to be relatively high due to the presence of organic

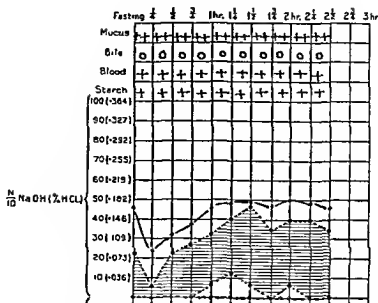


Fig. 45.—CARCINOMA OF STOMACH. THE TOTAL ACIDITY CURVE IS HIGH Owing TO THE PRESENCE OF ORGANIC ACIDS, BUT THERE IS ABSENCE OF FREE HCL. NOTE THE DELAYED EMPTYING OF THE STOMACH, AND THE PRESENCE OF BLOOD AND EXCESS OF MUCUS IN THE SPECIMENS.

acids such as lactic acid. The presence of lactic acid is not pathognomonic of malignant disease, but may occur when stasis and fermentation are present from any cause. Blood is present in some or all of the specimens, which may occasionally be foul-smelling. In a case presenting gastric symptoms the association of achlorhydria and blood in a test meal is highly suggestive of carcinoma. The special features

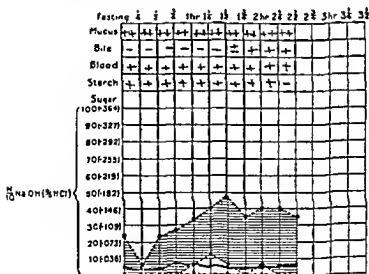


Fig. 46.—CARCINOMA OF THE STOMACH WITH ABSENCE OF FREE ACID AND LOW TOTAL ACIDITY. BLOOD AND EXCESS OF MUCUS IN ALL THE SPECIMENS.

found on gastric analysis in the rare condition of ulcer-cancer are described in a separate section (see page 562). It must be emphasised that the test meal is only one aspect of the investigation of a case in which gastric symptoms are prominent and that it is not meant to replace a careful history, clinical, X-ray and stool examination, but should be taken in conjunction with them.

## REFERENCES :

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2. BENNETT (I.). *The Stomach and Upper Alimentary Canal in Health and Disease*.
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## (2) OCCULT BLOOD

The examination of the stools for occult blood forms an important part of the investigation of gastric and duodenal disease, and should never be omitted. If bleeding takes place in the alimentary canal, or if blood is swallowed and it reaches the lower bowel, its presence will be demonstrated in the fæces. After a large hæmorrhage, the fæces will have the typical black, sticky and tarry appearance of melæna. On the other hand, lesser degrees of bleeding do not alter the macroscopic appearance of the fæces, and they appear normal to the naked eye, unless the bleeding is from low down in the alimentary canal—as bleeding from hæmorrhoids—when the blood may be seen as streaks on the surface of the fæces. To demonstrate the presence of a small amount of blood, special tests have to be applied. It is to this condition, namely small amounts of blood in the fæces, which needs special tests for its detection, that the term “occult blood” is applied.

When the bleeding is from the upper alimentary canal, the blood undergoes changes in its passage down the bowel. The hæmoglobin becomes converted into hæmatin and hæmatoporphyrin. The actual cause of this change is at present not understood, but it depends on the presence of bile in the intestines, and does not occur if the bile is unable to reach the alimentary canal. (Snapper ; Hurst and Stewart.)

*Preparation of the Patient.* Before collecting stools for examination, the bowels should be opened at least once a day during the three preceding days to overcome possible error due to constipation. If needed, and the case is otherwise suitable, a mild aperient may therefore be given at this stage, although no strong purgative is allowed as

this in itself might promote slight bleeding. To avoid minor bleeding from the gums, the patient should not use a tooth-brush during this period, though a mouth-wash may be used. It is necessary also for the patient to be on a hæmoglobin-free and chlorophyll-free diet during the previous three days, otherwise feebly positive results will be obtained with the tests. Meat, meat extracts, all green vegetables, whether cooked or raw, and coloured fruits are omitted. It is usual to advise omission of medicines containing iron, copper and iodides, although, contrary to what is often taught, preparations containing inorganic iron do not give a positive reaction. The diet given will largely consist of milk, milk puddings, eggs, fish, cheese, potatoes, bread and bananas. A dose of charcoal, or a charcoal bismit, is given during the first day of the diet, and its appearance in the fæces noted. A specimen of fæces passed subsequently when the fæces are again clear of charcoal is taken for examination. The motion is passed in a bed-pan. Special fæces pots, containing a small metal spoon, are available for collection of the specimen, and are most suitable. The bowels should be opened daily, giving enemata if necessary.

#### TESTS FOR OCCULT BLOOD

A. *Chemical.* Several tests have been described, but the following are the most satisfactory.

(1) *Guaiac Test.* A piece of fæces the size of a large pea is placed in a test-tube and mixed with a few cc. of water by means of a glass rod, and boiled. When cool, about an equal volume of glacial acetic acid is added and shaken. An equal volume of ether is further added, and again well stirred. On standing, the ethereal layer will, as a rule, separate. If it does not do so, water must be added by pouring down the side of the tube, when the ether layer will separate to the top on standing. This layer is carefully poured off into two clean test-tubes, one of which may be kept for spectroscopic examination. To the other tube two drops of tinct. guaiaci are added and shaken. Ozonic alcohol is now carefully poured down the side of the tube, when the formation of a blue ring will indicate the presence of blood.

(2) *Benzidine Test.* This test is considerably more delicate than the guaiac reaction, and will detect one part of blood in a million. It is essential that all glass apparatus used should be absolutely clean before use. A piece of fæces is mixed with water in a tube and boiled, as above. When cool, a few drops of a freshly prepared saturated

solution of benzidine in glacial acetic acid are added and mixed. Hydrogen peroxide 3 per cent is then poured down the side of the tube. A deep blue colour is produced if blood is present.

A convenient method has been described by Gregerson in which the test may be carried out on a glass slide. Powders are prepared containing 0.2 gramme of barium peroxide and 0.025 gramme of pure benzidine. If wrapped up in waxed paper these will remain satisfactory indefinitely.

When it is required to carry out a test one of these powders is dissolved in 5 cc. of freshly prepared 50 per cent solution of acetic acid. A small fragment from the centre of the stool is smeared on to a clean glass slide by means of a glass rod, and a few drops of the solution run on to the smear. If the reaction is positive a blue or bluish-green colour develops within a minute. The strength of the reaction may be graded according to the depth of colour and the time taken for its development. It is worth noting, moreover, that if a well-marked blue colour develops within half a minute the presence of occult blood in the stool may be inferred, whatever diet the patient has been taking.

*B. Spectroscopic Examination.* The ether extract, prepared as described under the guaiac test, is examined with the direct vision spectroscope. If hydrochloric acid be added to the extract, any alkaline hæmatoporphyrin will be converted into acid hæmatoporphyrin. This is more easily identified, and shows a narrow band in the orange and a wider band in the green. Acid hæmatin must be looked for, and shows two broad bands, one in the red and the other in the green.

The chemical tests are more sensitive than the spectroscopic test, and are easier to perform. Considerable experience is needed to interpret correctly the various absorption bands that may be seen with the spectroscope. On the other hand, iron-free derivatives, such as hæmatoporphyrin, give negative results with the chemical tests, but are identified with the spectroscope. Both chemical and spectroscopic methods should be used.

#### INTERPRETATION OF RESULTS

Provided the rules laid down as to diet are observed, a positive result indicates the presence of blood in the fæces. The chemical tests give no clue as to the source of the bleeding, but the spectroscopic test may help, the presence of hæmatoporphyrin indicating as a rule that the bleeding is from the upper parts of the alimentary canal.

Confining ourselves to diseases of the stomach and duodenum, the presence of occult blood is of great value in distinguishing between



organic disease, such as ulcer or carcinoma, on the one hand, and functional conditions on the other. Care must be taken to eliminate any possible source of bleeding from the mouth, gums, naso-pharynx, and hæmorrhoids.

In *carcinoma* the presence of occult blood in the fæces is the rule. The guaiac test is positive, and the spectroscope will, in the majority of cases, demonstrate the presence of hæmatoporphyrin. Hurst states that he has never seen a case of carcinoma of the stomach in which the fæces were free from occult blood. Negative tests are therefore strong evidence against a new growth.

In *peptic ulcer* during the active stage occult blood is present in the majority of cases. If the first result is negative, it is essential that the tests should be repeatedly performed. A negative result simply rules out the presence of blood in the particular specimen of fæces examined, but as an ulcer bleeds irregularly, the tests must be repeated on subsequent motions. With active medical treatment, it may be shown that the blood disappears from the fæces *pari passu* with the clinical and radiographical evidence of healing. The disappearance of occult blood, therefore, gives valuable evidence as to the progress of a case. Should the blood persist for more than a few weeks while under medical treatment, the possibility of the ulcer being primarily malignant or undergoing malignant transformation should be kept in mind, and the case carefully reviewed. If the patient has been kept in bed, the treatment has been really thorough, and all other sources of bleeding have been eliminated, the possibility becomes a probability, and is an indication for laparotomy. In such an instance Knott considers that the burden of proof should not be laid solely on the chemical test, but a positive result be required also from the less highly sensitive spectroscopic method.

In *gastric syphilis* occult blood is usually present in the stools, while in *chronic gastritis* it is usually absent. In various intestinal conditions where small hæmorrhages occur, as in ulcerative colitis, neoplasm, and typhoid or tuberculous ulceration, the test is of value in diagnosis.

In summary it may be said that the chief value of the test is in exclusion. Many factors may lead to a positive result; the value of a negative test is of much greater significance. It may confidently be said that, if a series of these negative results be obtained on alternate days, any hæmorrhage from the gastro-intestinal tract may be safely excluded.

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### (3) EXAMINATION OF THE URINE AND TESTS FOR RENAL EFFICIENCY

In its relation to cases of dyspepsia an attempt to determine the degree of functional efficiency of the kidneys is of importance from a double aspect. In the first place, primary renal disease may clinically be most evident by gastro-intestinal symptoms and so mislead diagnosis. Secondly, in weighing the operative risks of an established gastric case, in later life in particular, it is imperative among other considerations to have full knowledge of any latent cardio-renal defect.

The simplest and most obvious indication of renal efficiency is afforded by routine examination of the urine, described fully in the medical text-books. In many instances all the needed information can be obtained from a record of the total volume, naked-eye appearance, smell, colour, specific gravity and reaction of the urine, and the macroscopic appearance of any deposit, together with simple ward tests for albumen and sugar, and for bile, acetone bodies, blood and pus if indicated. Before any operation inspection under the low power of the microscope of the urinary deposit on standing should be made as a routine in patients over middle age; such a procedure is easily carried out in the ward or consulting room. If there be any suspicion of nephritis, cardiac failure or urinary tract infection, detailed search is made for red blood cells, excess of leucocytes or frank pus, casts of various kinds, epithelial cells and crystals, supplemented by bacteriological examination where necessary.

The investigation of a primarily surgical disorder of the urinary tract, especially prior to any operative procedure, demands, of course, special tests of more detailed or differential character, such as the excretion by each kidney of various dyes and foreign substances opaque to X-rays, with which aspect we are not here concerned.

Where from a preliminary examination of the urine renal damage is suspected, confirmation can be obtained by blood analysis, since defective elimination of breakdown products in the urine is reflected by their undue storage in the blood. In employing such tests the great reserve of the kidney must be remembered; probably not until two-thirds of the total secretory tissue is rendered inactive is a functional defect so revealed. The substances which have from time to time been so estimated in the blood and the resulting tests propounded are innumerable, but that most helpful and generally applicable as a routine

in surgical cases is estimation of the *blood-urea*, which serves as an index of the retention of various waste products in the body. This is a simpler procedure than estimation of the total non-protein nitrogen with which the figures usually run parallel, and gives equally satisfactory results. In most cases, therefore, the fuller test can be omitted, although occasionally both are helpful.

The normal range of blood-urea, which remains constant in health, is between 20 and 40 milligrammes per 100 cc. of blood, increasing slightly with age. The figure is raised in those cases of chronic interstitial nephritis or primary renal sclerosis of the azotæmic type, where nitrogen retention rather than œdema is the main feature. In uræmic states a reading of over 200 mgms. is obtained. Direct renal damage, e.g. by a stone or urinary obstruction, causes an even more rapid rise of the blood-urea than do degenerative changes.

It must be borne in mind that any concentration of the blood from loss of fluid, prolonged vomiting (as in intestinal obstruction) or profuse sweating, brings about a corresponding though moderate rise of the blood-urea. The diminished output of urine in cardiac failure has a similar effect. The vomiting of intestinal obstruction or cardiac failure may raise the blood-urea to say 80 or even 100 mgms. per 100 cc., but there is seldom doubt as to a possible uræmic origin, the high-level figures of which are not reached.

Aid in differentiation on this point is obtained also by estimating the percentage of urea in a specimen of urine taken at the same time that the blood is withdrawn. If the vomiting is due to a renal cause, the urea percentage in the urine is low—less than 2 per cent—the kidney being unable to concentrate more highly. If the cause be circulatory or mechanical, the urine is scanty and high-coloured with a high concentration of urea, well above 2 per cent.

If non-renal causes can be excluded, it may be said that a blood-urea estimation of over 40 mgms. per 100 cc. indicates renal damage, the degree of the rise being roughly proportional thereto. It is in the case awaiting operation with a high blood-pressure and hypertrophied heart that estimation of the blood-urea is particularly of value in determining to what degree, if any, renal damage is a contributory factor.

Details of the technique of estimation are omitted, being laboratory procedures. In brief it may be said that two main methods are employed. One requires 5 cc. of blood collected from a vein in an oxalated tube; after precipitation of proteins with trichloroacetic acid, the urea is decomposed by sodium hypobromite and the resulting

nitrogen evolved is measured. In the alternative micro-method, described by Archer (1), only 0.2 cc. of blood is needed, collected from a finger-prick, the urea in this case being converted by the urease ferment of the soya bean into ammonium carbonate, which is estimated colorimetrically with Nessler's reagent against a standard solution. With either method a delay up to 24 hours between collection and estimation is of no consequence.

Many other substances besides urea have been estimated in the blood as tests for renal function, e.g. creatinine, cholesterol, chlorides, diastase, calcium, phosphorus, etc. In the ordinary case their employment is of little or no value. Of the group, the *cholesterol content* of the blood is occasionally of prognostic aid. This in health is normally remarkably constant in the same individual, i.e. between 0.13 and 0.20 mgms. per 100 cc., averaging 0.17 mgms. Becoming raised in old age with arteriosclerosis, it is, nevertheless, found to be low in cases liable to sepsis. A low figure is therefore a danger sign of possible infection in surgical, especially prostatic, cases, a later rise of the cholesterol content to normal corresponding with a rise in the general resistance (Maxwell, 2).

In distinction to blood examination, defective elimination by the kidney of nitrogenous waste products is readily demonstrated in the urine by giving a meal of urea by mouth under test conditions and measuring its rate of excretion, the widely-used *urea concentration test*. This, again, affords only a rough index of renal ability and is of value in medical rather than surgical cases.

The test is carried out in the following way. The patient takes nothing by mouth after 9 p.m. except, if he so wishes, tea and toast at 6 a.m. on the following morning. The bladder is emptied overnight. The tests are begun at any convenient time, usually 8.30 or 9 a.m., when the patient first passes urine, the specimen being saved; he is then given to drink 15 grammes of urea dissolved in 3 ounces of water, flavoured because of its bitter taste with tincture of orange. Subsequent specimens of urine are collected in separate bottles at 1, 2 and 3 hours after the urea is taken, the bladder being completely emptied on each occasion. The volume of each specimen is measured and its urea percentage estimated by the hypobromite method.

It is generally stated that with healthy kidneys the concentration of urea in the first and second hourly specimens after administration should be a minimum of 2 per cent; often a reading up to 3.5 per cent or higher is found, the normal subject rapidly excreting such a dose without storage. Allowance must, however, be made for the possible diuretic action of the urea during the first hour, when a large volume of

urine may be passed of relatively low concentration, below 2 per cent. It is then found that the percentage of urea is higher and the volume of urine smaller in the next hourly specimen. The average volume of each specimen passed works out at 67 cc., with say 2 per cent of urea. It is clear, therefore, that passage in the hour of 130 cc. with only 1 per cent of urea would have the same good significance as regards excretory power of the kidney. A volume, however, of 60 cc. or less, with say only 1 per cent urea, shows badly damaged kidneys. For this reason it is essential in evaluating the result of the test to consider the volume of each specimen passed. A more accurate method of recording which would overcome this difficulty would be to state in actual grammes the total weight of urea excreted in each hour. On the above standard this should be a minimum of 1.4 grammes if renal efficiency is to be accepted as normal. Probably it would be safer only to consider satisfactory an average concentration of 2.5 per cent or higher (apart from diuresis) with a corresponding total output of 1.7 grammes of urea per hour. No general ill-effects are found from giving the urea, even in nephritic or uræmic patients.

A combination of the two urea tests described above is advocated by Archer and Robb (1) in the form of a *urea tolerance test* on the analogy of the familiar glucose tolerance test, and is of more exact value in judging renal efficiency than either test alone. The technique of giving the urea in no way differs from that of the urea concentration test. A sample of blood is taken at the time this is drunk, and subsequent blood specimens removed at  $\frac{1}{2}$  hour, 1, 2 and 4 hours later, the urea content of each being estimated by the micro-method. As only the finger is pricked no more discomfort is caused than in a sugar tolerance test. Specimens of urine are collected and examined throughout at the usual intervals as already described.

It is found that with a dose of 15 grammes of urea by mouth very rapid excretion occurs in the normal subject, the rise of blood-urea not exceeding 5 to 15 mgms. in the first  $\frac{1}{2}$  to 1 hour, with a complete recovery to resting level by 4 hours or earlier. In the majority of healthy individuals indeed the peak is reached in  $\frac{1}{2}$ -hour with return to normal within 1 hour.

With gross renal damage, not only is the resting level of blood-urea high (above 40 mgms.), but the subsequent rise enormous with a very gradual subsidence, incomplete in 4 hours. The test is most delicate and, therefore, of greatest value with milder degrees of defective function, where although the resting figure is often normal and the rise only slightly excessive (i.e. greater than 15 mgms.), yet recovery is

definitely very slow and incomplete by the full period of 4 hours.

S. E. King (3) has extended these observations by following the urea curve in pathological cases to its resting level, and adjusting the test dose of urea according to the body weight. The patient is given 1 gramme of urea by mouth for every 10 lbs. of body weight, and estimation of the blood-urea made before, 2 hours and 14 hours after the test-dose. The intake of fluid during this period is limited to 500 cc., and the urine passed over the period of the test measured, being normally not more than 750 cc. Where, apart from an excessive diuresis above this figure, the blood-urea has failed to return completely to the control level at the end of 14 hours, defective excretory function is present.

With the aim of greater precision, the use has been advocated from time to time of a mathematical formula such as Ambard and Weill's co-efficient, depending on the ratio of blood- to urinary-urea. The majority of these tests have suffered the drawback of too great a complexity and have been rendered of doubtful value in diagnosis by the many variable and uncontrollable factors introduced, such as age, weight and relation to food. An exception must be made, however, in the test described in 1928 by Möller, McIntosh and Van Slyke (4) under the term of *Blood-Urea Clearance Test*, which does afford a more accurate and delicate measure of renal function than the majority of tests in general use, and yet remains simple of performance and estimation. The blood-urea clearance is a record of the number of cubic centimetres of blood cleared or rendered completely free of urea by the kidneys per minute of time, so linking up (as in the urea tolerance test already described) the two factors of concentration of urea in the blood and its excretion in the urine. The test is carried out in the following way, no special preparation being needed. The period most convenient for the test is that between breakfast and lunch. The patient is allowed an ordinary breakfast, except that no tea or coffee may be taken. At exactly 10 a.m. the bladder is emptied completely, if necessary by passing a catheter, and the specimen of urine discarded, not being required for the test. At 11 a.m. and 12 noon precisely the bladder is again completely emptied in the same way, the separate specimens collected being kept for examination. It is of the greatest importance in avoiding fallacies and erroneous results, both that the whole of the urinary output shall be obtained and that the length of time between collection of specimens shall be exactly 60 minutes in each case. The volume of urine passed in each hourly period is then

accurately measured and its urea-percentage subsequently estimated. At about midway between the collection of the two urinary specimens, the exact time in this instance not being important, 0.2 cc. of blood is taken from the finger for estimation of the blood-urea, which may be assumed to be constant over the time of the test. The Blood-Urea Clearance for each hourly specimen is then calculated as described below, and the average of the two readings taken as the result.

The volume of urine passed during the one-hour period concerned divided by 60 gives the output of urine from the kidneys per minute, expressed as "V". The urea content of the urine-specimen is estimated, termed "U". "V"  $\times$  "U" then represents the amount of urea excreted in the urine, and therefore got rid of from the blood, per minute. From estimation also of the blood-urea, "B," the volume of blood completely cleared of urea, or "*Blood-Urea Clearance*" can be easily determined, being equal to  $\frac{U \times V}{B}$ .

The calculation actually employed varies with the volume of urine passed. It has been shown that the clearance of the blood from urea is at its maximum when the output of urine per minute reaches 2 cc., any more rapid secretion of urine not being accompanied by a correspondingly higher rate of clearance. For all urine volumes over 2 cc. per minute the B.U.C. is thus independent of the volume, and is hence termed the *Maximum Clearance* or  $C_m$ , being calculated from the formula  $C_m = \frac{U \times V}{B}$ .

With a slower rate of secretion of urine, i.e. when the volume falls below 2 cc. per minute, the rate of excretion of urea from the blood is also diminished, falling approximately in proportion to the square root of the volume of urine passed per minute. If, for example, this volume is reduced to  $\frac{1}{4}$ , the resulting clearance of the blood is diminished to  $\frac{1}{2}$ . The blood-urea clearance in any case, therefore, where less than 2 cc. of urine is passed per minute is expressed as a *Standard B.U.C.* or  $C_s$ , calculated on a standard urine volume of 1 cc. per minute, from

the formula  $C_s = \frac{U}{B} \times \sqrt{V}$ . In the case of children a correction of volume is necessary by multiplying V by a factor  $\frac{1.7}{\text{Surface area}}$ , the latter being calculated from a standard height-weight formula.

In actual practice the test is very simple. It has been found that in normal healthy adults, taking average results, the Maximum B.U.C.

is 75 cc. and the Standard B.U.C. 54 cc. of blood per minute. By taking each of these figures as 100 per cent respectively for maximum and standard clearance, results of clinical tests are expressed in percentages, e.g. 27 cc. in a Standard Clearance is recorded as 50 per cent.

The blood-urea clearance test is a more delicate and discriminating measure of minor degrees of renal inefficiency than either the simple blood-urea or urea concentration test alone, revealing degrees of damage otherwise latent. In this way it is of prognostic value, especially in determining what grade of reserve function is available to withstand the trauma of operative measures, while a series of observations on the same patient is similarly a more accurate guide of progress.

It has the chief drawback of a rather wide variation, by as much as 20 per cent in either direction, in the results obtained in the healthy subject clinically free from renal abnormality. In the presence, however, of renal disease this zone of variation is considerably narrower, and the readings obtained a more constant and localising standard of the degree of renal damage. By Van Slyke any reading below 70 per cent is considered pathological.

A modification of the test has been suggested, in an attempt to make it still more selective and delicate, by artificially increasing the work thrown upon the kidneys, the blood-urea clearance being determined directly before and one and two hours after giving 15 grammes of urea by mouth, and the results compared. Fowweather (5) finds that estimation of the blood-urea clearance in the second hour after urea gives a more constant reading in the normal subject and affords a more exact measure of renal function in the presence of disease than the test as ordinarily carried out, and suggests, therefore, that estimation before giving urea may be dispensed with. Further confirmation as to the value of this modification is needed, in view of the added complexity of the test, before its general adoption is recommended.

Of the group of renal tests depending on the excretion of a foreign substance, the most reliable is the *phenol-red* or *phenol-sulphone-phthalein* test. Given intramuscularly this substance begins to be excreted in the urine (only) within 15 minutes. To promote a good flow of urine 300 to 400 cc. of water is first given to drink. A quarter of an hour later, the bladder is emptied and 6 mgms. of the dye injected intramuscularly. The amount excreted in specimens of urine passed after 1 and 2 hours is estimated quantitatively in alkaline solution by comparison with a standard in a colorimeter. Normally about 60 per cent of the total injected is excreted during the first and the remainder



during the second hour. Anything below 70 per cent of total elimination within 2 hours points to defective renal function.

This last test together with urea tolerance and clearance tests form the triad of choice in surgical cases upon which a fair estimate of renal efficiency can usually be based.

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#### (4) WASSERMANN AND KAHN REACTIONS FOR SYPHILIS

The mode in which chronic syphilitic lesions of the nervous or vascular systems may simulate abdominal conditions has already been briefly described. Apart from these special considerations, the unearthing by a blood test of latent syphilitic infection is not without value, as its possible harmful effect cannot be definitely estimated and specific treatment by increasing the general resistance often aids recovery from other infections. Indeed, the routine use of these tests in hospital has been advocated and is not without advantage. Moreover, the possible gummatous origin of any tumour in the upper abdomen has to be considered. Gumma of the stomach itself is very rare, of the liver less uncommon.

#### (5) COMPLETE BLOOD COUNT

The value of a blood count in diagnosis of apparent dyspeptic states may be briefly reviewed.

The clinical observation of anæmia can be confirmed and exact information gained as to its degree and type, pernicious anæmia, for example, being distinguished from the secondary type, say, of gastric carcinoma. Acute secondary anæmia due to blood loss is again confirmed by blood examination, with its falling colour index and subsequent response of granular leucocytes and perhaps nucleated red cells. Of inestimable value in acute infections is the determination of

leucocytosis, both in diagnosis of the likelihood of suppuration and in prognosis as to response and possible recovery.

Light may also be thrown on chronic parasitic infection, e.g. bydatid cyst, or metallic poisoning, e.g. lead colic, by the presence of characteristic eosinophilia or punctate basophilia respectively. Primary blood conditions such as leukaemia or splenic anaemia will also be revealed.

Special blood investigations will often supplement the information of an initial blood count in diagnosis; for example, an increase of red cell fragility confirms the view that a splenomegaly is due to acbolic jaundice, while in some haemorrhagic conditions, such as purpura or haemophilia, investigations of the blood-platelets and of clotting and bleeding time are of diagnostic value. In conditions of jaundice also, with its increased liability to haemorrhage, they are again of service.

#### (6) DUODENAL INTUBATION

Examination of the gastric contents aspirated through a tube has long become a commonplace method of pathological investigation, and has proved of the greatest value. Similar study of the contents of the duodenum, though presenting no marked increase of difficulty in technique, has not, however, achieved popularity in this country, and in the writer's opinion has met with inadequate recognition both as a method of diagnosis and of treatment. The fullest investigation of the possibilities of the method has been carried out by Vincent Lyon (1). Its main indication is in the investigation of suspected lesions of the gall-bladder and biliary passages.

The underlying physiological reaction on which the test is based is the law of contrary innervation of the biliary tract, and on the related observation that magnesium sulphate in concentrated solution brought in contact with the region of the sphincter of Oddi causes relaxation of the sphincter together with powerful contraction of the gall-bladder.

The test is carried out in the following way. The patient takes no food for 12 hours previously. The mouth and fauces are cleansed as far as possible by thorough gargling and mouth washes. The patient then swallows a fine duodenal tube, either of the Ryle or better of the Einhorn type, which is paid out to the 60 cm. mark, when it should be in the body of the stomach. The gastric contents are then aspirated and saved, and the stomach gently washed out with water until clear, leaving a little water behind. The patient now turns over on to his

right side and very slowly swallows the tube further until the 75 cm. mark is reached. The tube should now be in the duodenum. Confirmation of its passage can be obtained by noting the alkaline reaction and bile-stained appearance of the fluid withdrawn, by the fact that water injected into the duodenum cannot be recovered, and by X-ray screening. A specimen of the duodenal contents is now aspirated and kept for examination.

50 cc. of a warmed 25 per cent solution of magnesium sulphate are slowly injected into the duodenum, and five minutes later the contents withdrawn by aspiration or syphonage. This first specimen after the injection in normal cases consists of clear, golden-yellow bile derived from the bile-ducts. Withdrawal of fluid is repeated at intervals of a few minutes. It is usually found that the later specimens consist firstly of dark brown viscid bile, being expelled gall-bladder contents, and subsequently of thin watery bile from the liver itself. These fractions are kept separately for examination.

At the end of the examination, which usually occupies from 2 to 4 hours, the duodenum is washed out with normal saline and a drachm of sodium sulphate given to clear the intestinal contents of any infected bile. The chief drawback to the investigation is the length of time involved. It can, if need be, be performed on out-patients and no greater discomfort is experienced than with a fractional test meal.

The volume of each specimen removed is measured, and its naked-eye appearance noted with regard to viscosity, colour, clarity or otherwise, and the amount and character of deposit on standing, these observations being of equal importance to microscopic tests. Wet films are then made from the deposit of each sample and examined for mucus, crystals, and the number and type of cells present. Bacteriological investigation is also made by stained films and cultures of the deposit.

The procedure of duodenal intubation is of value both for diagnosis and treatment. The information of a cholecystogram can be confirmed and extended. In chronic cholecystitis and cholelithiasis, for example, the presence of excess of pus cells and cholesterol crystals may be demonstrated in some cases before X-ray findings are positive. Bacteriological tests have shown the infecting organism, in some cases the typhoid bacillus. In organic obstructive jaundice the failure of the gall-bladder to react to magnesium sulphate will be evident.

As a mode of treatment the method is of most value in conditions of cholangitis, catarrhal jaundice and cases of acute or chronic cholecystitis where for one or another reason operation is not indicated. In

such instances by repeated duodenal drainage a freer flow of bile has been obtained and toxic symptoms and degree of general illness materially reduced. With advance in technique it is possible that some chronic affections of the pancreas also may fall within the scope of the investigation.

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SECTION 2  
STOMACH AND DUODENUM

by  
RODNEY MAINGOT

CHAPTER I  
*Infantile Pyloric Stenosis*

CHAPTER II  
*Acute Dilatation of the Stomach*

- CHAPTER III
- A. *Acute Phlegmonous Gastritis*
  - B. *Injuries of the Stomach*
  - C. *Foreign Bodies in the Stomach*

CHAPTER IV  
*Peptic Ulcer*

CHAPTER V  
*Complications of Peptic Ulcer*

CHAPTER VI  
*Treatment of Peptic Ulcer*

CHAPTER VII  
*New Growths of the Stomach*

CHAPTER VIII  
*The Péan-Billroth I Operation*

by  
ENRIQUE FINOCHIETTO

## SECTION 2

### STOMACH AND DUODENUM

#### CHAPTER I

##### INFANTILE PYLORIC STENOSIS

ALTHOUGH other titles have been given to this disease, such as congenital hypertrophic stenosis of the pylorus, that given above is sufficiently descriptive and uncompromising.

The disease is four times commoner in males than in females ; 50 per cent of those affected are first-born children, and the condition is said to be more prevalent in breast-fed than in bottle-fed babies. As infantile pyloric stenosis has been observed to occur in twins or in more than one member of the same family, there may be a definite hereditary tendency.

The true ætiology of the condition is unknown, but there are two principal views held to explain the condition : (1) That the pyloric hypertrophy is due to spasm (Thomson); and (2) That there is a primary hyperplasia of the muscle (Hirschsprung).

The following explanations have, at one time or another, been given to account for the spasm :

- (a) That it is due to an inco-ordination between the sympathetic and parasympathetic fibres supplying the area (Thomson; Cameron).
- (b) That it is the result of hyperacidity (Engel).
- (c) That there is hyperæsthesia of the mucous membrane (Shattock).
- (d) That there is hyperadrenalism (Tyrrell-Gray and Pirie).

None of these theories adequately explains the true condition, but Thomson's view that it is due to an inco-ordination of the muscle-

fibres, secondary to lack of balance between the sympathetic and parasympathetic nerves controlling the pyloric region of the stomach, is the generally accepted one.

#### PATHOLOGICAL ANATOMY

The characteristic feature is the pyloric tumour, which is produced by an abnormal overgrowth of the circular muscle. It is felt as a hard, cartilaginous tumour, varying from 1-1½ inches in length. On viewing the stomach externally the peritoneal coat appears unaltered, and blood-vessels can be seen coursing over it. There is no evidence of peritonitis, although occasionally the area occupied by the tumour appears somewhat lighter in colour than the adjacent stomach wall.

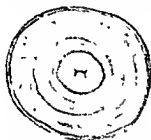


Fig. 47.—INFANTILE PYLORIC STENOSIS. TRANSVERSE SECTION THROUGH PYLORIC CANAL.  
(After Horsley.)



Fig. 48.—INFANTILE PYLORIC STENOSIS, SHOWING INCREASED CALIBRE OF PYLORIC CANAL AND BULGING MUCOUS MEMBRANE AFTER DIVISION OF THE HYPERTROPHIED MUSCLE.  
(After Horsley.)

In individual cases the tumour may be fairly pronounced, forming a bobbin-like mass in the pyloric region; in others very little can be seen from without to indicate the presence of a tumour.

When a transverse section is made through the pyloric canal it will be seen that the bulk of the tumour consists of the hypertrophied, inner, circular muscular coat (fig. 47). There is an increase in the number and size of the muscle-fibres. The longitudinal layer of muscle is likewise thickened, though to a slighter extent, and there is also some increase in the fibrous tissue. The mucous membrane is crammed into a narrow canal and tightly packed in folds. Although firmly compressed in this way within the narrow limits of the pyloric canal, the mucous membrane undergoes little change apart from a mild degree of fibrosis. When the muscular tumour is divided at operation its walls rapidly spring apart and allow the mucous membrane to expand to its fullest extent (fig. 48).

When a longitudinal section is made through a hardened specimen it will be seen that the body of the stomach is dilated, that its walls are hypertrophied and stretched, that the folds of mucous membrane are partly flattened over the greater portion of the stomach area, and that they converge in longitudinal ridges towards the inlet of the pyloric canal, where, although severely compressed, at least one prominent fold can usually be discerned (fig. 49).

The great thickening of the pylorus is now evident, and the maximum degree of thickening will be seen just proximal to the pyloric opening. The muscular hypertrophy gradually decreases as it is traced upwards along the stomach wall. It ends abruptly below where the sphincter bulges into the duodenum, forming a projection similar to that made by the cervix into the vagina. This projection of the pyloric tumour into the duodenum of necessity forms a fornix which is well shown in the illustration (see fig. 49). It is this fornix which may be inadvertently opened in the performance of Rammstedt's operation. The duodenum in cases of infantile pyloric stenosis is normal in every respect.



Fig. 49.—INFANTILE PYLORIC STENOSIS. LONGITUDINAL SECTION THROUGH PYLORIC TUMOUR AND STOMACH. (Museum, Hosp. for Sick Children, 61, Ormond St. By kind permission of the Board of Management.)

#### SIGNS AND SYMPTOMS

The child at birth is usually quite healthy and of normal weight. The first symptoms often appear about the third week of life, although they have been noticed in infants a few days old or even as late as the sixteenth week.

*Vomiting* is in nearly every instance the first and most important of all symptoms. In the early stages it may be described as slight, and follows each feed. It is more in the nature of regurgitation than true vomiting. After the feed a little fluid is seen to well up and dribble from the mouth. It may give the mother or the nurse the impression that the child is being overfed. Later the regurgitation becomes very pronounced and there is an accompanying decrease in weight. This is a cause for some anxiety and leads to an alteration in the infant's diet or in the method of feeding, often with temporary amelioration. It is surprising that a temporary improvement practically always results



when these methods are adopted. The vomiting, however, soon returns, fresh advice is sought, and the diet is altered a second or even a third time. Meanwhile the loss of weight continues, and eventually vomiting of much larger amounts occurs at longer intervals, until it becomes projectile in nature. The contents of the stomach are voided with great force through the mouth, and may even pour through the nostrils. Bile is rarely, if ever, found in the vomited material. In the terminal stages of the disease, when the stomach becomes atonic and grossly dilated, the fluid appears to overflow and spill into the mouth, and is passed in effortless gushes.

As a result of the persistent vomiting there is *loss of weight*. In some cases such loss may occur that the skin becomes dried, thrown into folds, and assumes a waxen appearance. In the late stages of the disease the face is cadaveric, the cheek bones are prominent, and wrinkles around the eyes, forehead, corners of the mouth and neck give the child the wizened, shrivelled mien of a very aged person. Marked loss of weight usually implies that the symptoms have been in progress for some time, and the greater the loss of weight, the worse is the prognosis.

*Constipation* is a very common symptom of pyloric stenosis; in fact, it usually occurs in any obstructive lesion of the gut. In infants with pyloric stenosis constipation is present in over 80 per cent of cases. Occasionally the bowel movements are normal, or there may even be diarrhoea.

Maizels and McArthur (*Lancet*, I, 286, 1930), working at the Infants' Hospital, have confirmed that *alkalosis*, not acidosis, is found in a large majority of cases. An estimation of the blood chloride and plasma bicarbonate will reveal the former to be well below the normal, while the latter will be in excess. The recognition of this fact has had an important bearing on pre-operative treatment, and has done much to lower the mortality.

David Levi (*Practit*, p. 67, Jan., 1933) writes as follows on this subject: "Maizel has shown that the frequent vomiting rids the system of a corresponding amount of acid radicals, and that, although the child is starving and vomiting, it is not in a condition of acidosis but of an alkalosis. There is an increase in the plasma bicarbonate and a decrease in the plasma cell chloride in 88 per cent of the infants examined by him. This change from the normal in the blood chemical picture is of great importance, and has even assumed diagnostic significance. Alkalosis is of very rare occurrence in a non-obstructive vomiting of infancy. The picture of an alkalemic child is typical. The child is pale, ashen and sunken, and tends to be cyanosed. Such children were among the bad operative risks of the past. It must be remembered that many still advocate that the stomach should be washed with sodium bicarbonate (Macleod, 1931), and that glucose and sodium bicarbonate should be administered to these

infants. Such treatment aggravates the alkalosis and renders the risk of operative interference greater. Since we have abolished the use of sodium bicarbonate, and have used water with which to wash the stomach and have left a little normal saline in the stomach, we have not seen the pale, ashen-grey patient with whom all workers in this field are familiar."

In some late cases *tetany* has been observed, and in a few instances *jaundice* of a very mild type.

### EXAMINATION

On *examination* the distension of the epigastrium is in marked contrast to the retraction of the lower half of the abdomen. Visible peristalsis may be seen in a well-established case, particularly after the child has been given a feed. The peristaltic waves course along the line of the stomach from left to right in characteristic manner. Sometimes the stomach can quite clearly be seen to be tensely blown out and ballooning the epigastrium and left hypochondrium. After a feed has been given or the abdomen kneaded, the tenseness in this region disappears and is replaced by vigorous, visible peristaltic waves which sweep across from the left costal margin to the umbilicus.

Before attempting the examination the child should be quietened and appeased by giving him some sweetened sterile water from a bottle. It will at once be evident that he is ravenously hungry, and the fluid is gulped down eagerly to appease an insatiable appetite.

On making the examination the child should be disturbed as little as possible, and it is quite unnecessary to remove all the garments and to expose the abdomen. The surgeon should, for preference, stand on the left side of the patient, and feel for the pyloric tumour with the left hand after it has been well warmed. The tumour will most often be felt about 1 inch to the right and above the umbilicus, but sometimes it is more internally placed, or lies further out. It may be inaccessible to the examining fingers when it is tucked away under the recesses of the right lobe of the liver. The fingers of the left hand should be placed a little below the right costal margin, and firm steady pressure should be exerted in a backward, upward, and inward direction, in an attempt to compress the pylorus against the vertebral column.

The examination may be negative, and in suspected cases it should be repeated at varying intervals. In doubtful cases where it is most urgent that an immediate diagnosis should be made, it may even be expedient to examine the patient under an anæsthetic. Such a measure, however, is rarely necessary or desirable in elinching a diagnosis. The

tumour when once felt in a case is unmistakable. It has been described as "acorn-shaped," "hazel-nut in size," "hard as a lymphatic gland," "like the tip of a finger." It will be found to change its position at successive examinations, but it is usually deeply placed, above the umbilicus, and to the right of the middle line in the epigastric region.

#### TREATMENT

In a diagnosed case Rammstedt's operation followed by medical treatment is now the rule. Medical treatment by dieting, gastric lavage, etc., is reserved for the doubtful cases and for the very few and exceptional instances in which there is rapid and successful response to medical measures.

It is no exaggeration to state that prior to Rammstedt's description of his operation practically every case was treated medically. The mortality was very high, probably about 80 per cent, and the complicated and unsatisfactory operations that were performed with damning mortality strengthened the claims of medical treatment. All these operations lacked the simplicity, the ease, and the security of Rammstedt's method. Pylorotomy had a mortality of 100 per cent; pyloroplasty 80 per cent; gastro-jejunostomy certainly not less than 50 per cent; while Loretta's operation of division of the sphincter was a very fatal procedure except in the expert hands of Burghard. Fredet obtained better results by dividing the muscle, but he prolonged, complicated, and rendered the operation more lethal by a transverse suture of the severed muscle of the pyloric canal. Rammstedt (*Med. Klin.*, 8, 1702, 1912, and *Zentralb. f. Chir.*, 39, 1741, 1912), in attempting Fredet's operation on a case, was unable to complete it owing to collapse of the patient. The child made a good recovery, and Rammstedt had, to the lasting benefit of mankind, the sense to appreciate the fact that simple division of the pyloric tumour, allowing full expansion of the mucous membrane of the pyloric canal, was all that was necessary, and he had the courage to repeat this method in subsequent cases with successful results.

It is very difficult indeed to assess correctly the mortality of Rammstedt's operation at the present time. The numerous statistics published in England and America show how widely varied are the results. They are, in many cases, also misleading, as no specification is made as to whether the results concern early or late cases. "The point that emerges forcibly in my maturer experience is that one's mortality depends almost entirely upon nursing and segregation. In

my private cases it is under 2 per cent, but I consider that the over-all mortality ought not to exceed 5 per cent." (Twistington Higgins.)

In America, Ladd (*Boston Med. and Surg. Jl.*, 211, Feb., 1927) published statistics of one hundred and ninety-seven cases, and of these one hundred and eighty-six were cured and eleven died, making a mortality of 5.5 per cent. Strachauer (*Ann. Surg.*, 67, Jan., 1927) had the remarkable figures of forty-eight cases with forty-seven cured and only one death, a mortality of 2 per cent.

Lanman and Mahoney (*Surg., Gynec., and Obst.*, 205, Feb., 1933) give the following summary of 425 of their cases treated by pyloromyotomy (Rammstedt's operation):

TABLE—SUMMARY OF CASES

<i>Years.</i>	<i>Serial number.</i>	<i>Number of Cases.</i>	<i>Deaths.</i>	<i>Mortality per cent.</i>
1915-1923	1-125	125	13	10.4
1923-1928	126-275	150	11	7.0
1928-1931	276-425	150	3	2.0

In my series of 22 cases 2 have died, a mortality of 9 per cent, but at one period there were ten successful cases in succession. One case that died—the thirteenth—lived for nearly six weeks after Rammstedt's operation, and then developed diarrhoea which eventually proved fatal. The post-mortem examination of this case revealed a patent pylorus, almost complete disappearance of the pyloric tumour, and a healed scar. It is possible for a surgeon to have a large number of *early* cases in succession with a low mortality, and it is usually operation upon *delayed* cases, where there has been a great loss of weight and inanition, and where surgery has had to be undertaken as a last resource, that raises the death-rate.

#### FACTORS WHICH HAVE LOWERED THE DEATH-RATE

- (1) The general recognition of the fact that *early* operation is the treatment of choice.

(2) Careful, intensive, routine pre-operative treatment. This includes :

(a) Regular stomach wash-outs with sterile saline solution, the final irrigation of the stomach being performed shortly before the child is taken to the operating theatre.

As the contents of the stomach are fluid, a Ryle tube should be used for aspirating the stomach contents and for the irrigations. Under no condition should sodium bicarbonate be employed for washing out the stomach, or for rectal or subcutaneous infusions, as it increases alkalosis.

(b) Subcutaneous salines. During the last 12 hours before operation is performed, 10-12 oz. of normal saline should be injected. The saline should be run very slowly into the pectoral region and groins until small tumours form. It is important to see that the injected fluid is not too hot, and that the tumours are not rendered too tense, as otherwise sloughing of the skin or of the subcutaneous tissues may result. Glucose 2 per cent may be added to the infusions.

(c) Small frequent fluid feeds up to 2 hours before operation.

(3) The maintenance of the child's body heat prior to, during, and after the operation. Before the patient is taken to the operating theatre the arms and legs should be swathed in cotton wool; the temperature of the theatre should not be less than 75 degrees Fahrenheit, and the operating table may, with advantage, be heated.

(4) The choice of anæsthetic. Chloroform is now never given to these patients; the anæsthetic of choice is gas and oxygen (with a little ether), open ether, or a local in desperate cases.

(5) The universal acceptance of Rammstedt's operation.

(6) A well-planned scheme of post-operative treatment. After operation these patients are now handed over as a routine to the physician for dieting, etc., as operation is only an incident, although a very important one, in their treatment. The services of a good nurse, who has had previous experience in the management of such cases, is indispensable. A great deal will depend upon her skill, attentiveness, and cleanliness, and she should devote her whole time unremittingly to the one case under her care.

It has been found that private cases fare better than hospital cases. This is probably due to the fact that private cases are diagnosed earlier and that they are isolated. It is undoubted that there is a tendency for hospital patients to contract infectious diseases and otherwise fare worse when nursed in a large infants' ward, or even, as has been

attempted, in an adult ward. It has long been recognised that in some of the up-to-date infants' hospitals, where special self-contained suites have been set aside so that patient, nurse, and mother can be isolated while the post-operative regime is being conducted, results far exceeding those that have ever been obtained in the past are now possible.

There are numerous methods and schemes adopted for post-operative feeding, many of which will be found to be reliable, but the one on which I have myself placed most reliance is that here given.

# FEEDING OF A CASE OF PYLORIC STENOSIS AFTER OPERATION

(Infants' Hospital)

*Feeding begins six hours after operation.*

The first feed consists of	Glucose 7.5%
	in saline, dram 1.
Then at $\frac{1}{2}$ hour	interval give Glucose 7.5%
	in saline, dram 1.
" $\frac{1}{2}$ hour	" Glucose 2.5%
	in saline, dram $\frac{1}{2}$ }
	Breast mk., dram $\frac{1}{2}$ } dram 1.
" $\frac{1}{2}$ hour	" " dram 1.
" 1 hour	" " dram 1 $\frac{1}{2}$ .
" 1 hour	" " drams 2.
" 1 hr. 15 mins.	" " drams 2 $\frac{1}{2}$ .
" 1 hr. 30 mins.	" " drams 3.
" 1 hr. 45 mins.	" " drams 4.
" 2 hrs.	" " drams 5.
" 2 hrs. 5 mins.	" " drams 6.
" 2 hrs. 10 mins.	" " drams 6.
" 2 hrs. 15 mins.	" " ounce 1.
" 2 hrs. 20 mins.	" " ounce 1 + dram 1.
" 2 hrs. 25 mins.	" " ounce 1 + drams 2.
" 2 hrs. 30 mins.	" " ounce 1 + drams 3.
" 2 hrs. 35 mins.	" " ounce 1 + drams 4.
" 2 hrs. 40 mins.	" " ounce 1 + drams 5.
" 2 hrs. 45 mins.	" " ounce 1 + drams 6.
" 2 hrs. 50 mins.	" " ounce 1 + drams 7.
" 3 hours	" " ounces 2.

Continue with two-ounce feeds three-hourly, giving six feeds in the 24 hours for three or four days.

If there is no breast milk, substitute the following mixture: Cow's milk, ounces 2; lactose, dram 1; water, ounce 1: peptonize for 2 $\frac{1}{2}$  hours.

*Ramstedt's Operation.* The child is first bandaged to a padded wooden cross, and a small sand-bag or air-cushion is placed under the

lower ribs to render the epigastric region more prominent and accessible. The whole of the abdomen and the lower half of the chest are painted with absolute alcohol before a large abdominal towel, with a central hole, is placed in position.

There is a choice of three incisions: (1) mid-line; (2) paramedian; and (3) right transrectus muscle-split, whereby the inner fibres of the muscle are divided in a longitudinal plane. Each of these incisions has its advocates, but there is little to choose between them. When it is known that the tumour is centrally situated in the epigastrium, the mid-line incision will probably be the one of choice. On the other hand, if the pyloric tumour has been palpated rather further afield, more ready access will be gained by making an incision through the rectus muscle. This incision is small, commencing high up in the region of the xiphi-sternum and extending downwards for about 2 inches. Tetra-cloths are affixed to the skin edges before the peritoneal cavity is opened. The liver presents in the wound and hides the stomach from view. After the liver has been gently retracted upwards with the finger, the pyloric region of the stomach is seized and coaxed through the wound. It is well at this stage to place a small pack in the lower portion of the wound to prevent the ubiquitous omentum from prolapsing and interfering with the manipulations. The surgeon then fixes the pyloric tumour between the index finger and the thumb of the left hand, and so rotates it that its anterior aspect, the commencement of the duodenum, and a small portion of the pyloric vestibule are clearly visible.

An incision should then be made in the hypertrophied area at the junction of the middle and upper thirds of its anterior aspect where the blood-vessels seem to be scarce (fig. 50). It is slightly curved with

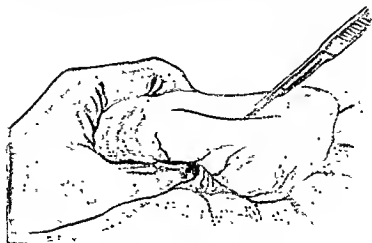


Fig. 50.—RAMMSTEDT'S OPERATION, THE INCISION.

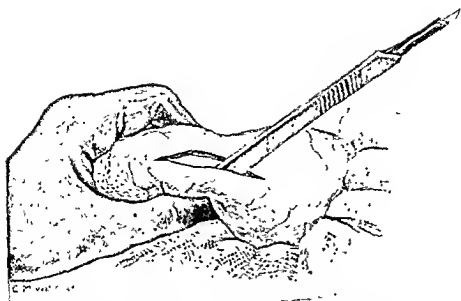


Fig. 51.—RAMMSTEDT'S OPERATION. THE INCISION HAS BEEN CARRIED DOWN TO THE SUBMUCOSA OF THE PYLORIC CANAL. THE HANDLE OF THE KNIFE COMPLETES THE SEPARATION OF ANY REMAINING MUSCULAR STRANDS.

its convexity downwards, and starting just proximal to the gastroduodenal junction it is carried through the peritoneal and superficial muscular coats, over the pyloric tumour, and a little beyond it into the body of the stomach. Using a dissector or the handle of a knife, the muscle-fibres are gently separated throughout the length of the incision, until the glistening white mucous membrane is visible and eventually bulges through the gap (fig. 51).

This part of the dissection is simple as the thickened circular coat rapidly springs apart. There is now an oval wound in the pyloric region of the stomach, the base of which is formed by the tough mucous

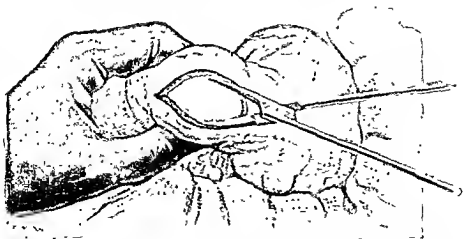


Fig. 52.—RAMMSTEDT'S OPERATION. THE EDGES OF THE WOUND ARE STRETCHED WITH A HEMOSTAT, ALLOWING THE MUCOUS MEMBRANE TO BULGE FREELY THROUGH THE GAP.



membrane of the pyloric canal. A little gentle stretching of the fibres with a hæmostat, as is well depicted in figure 52, and a careful dissection here and there, may be necessary to make absolutely sure that all the constricting fibres, and particularly those at the gastro-duodenal junction, have been divided, and that an efficient patency is assured (fig. 52).

When this is completed it is a wise manoeuvre to compress the body of the stomach and to force some air through into the duodenum. This ensures that the mucous membrane at the fornix has not been inadvertently perforated. If the mucous membrane has been punctured this compression will produce a hissing sound at the site of the perforation, and so lead to its detection. If a perforation is discovered it should be closed at once by one or two interrupted Lembert stitches, and reinforced with a tag of omentum. This mishap, if at once attended to in the method described, in no way detracts from the success of the operation.

A little bleeding may occur from the cut edges of the wound, but this does not, as a rule, call for any special attention and soon abates. If, however, a bleeding point is troublesome it should be underrun with a fine atraumatic intestinal needle, and ligatured.

When the stomach is replaced into the abdomen the operation is completed by closure of the wound. As these patients are very debilitated and mild sepsis of the wound is not uncommon, steps should be taken to prevent burst abdomen or post-incisional hernia by closing the wound securely, employing one of the many approved methods.

#### POST-OPERATIVE COMPLICATIONS

These may be briefly summarised as follows :

(1) Hyperpyrexia. This is rarely seen nowadays, and is a very infrequent cause of death. It is not peculiar to the operation as it has been observed prior to it. It is probably secondary to an enteritis.

(2) Innation. A few infants who have successfully withstood the operation seem incapable of recovery, and in spite of meticulous care in post-operative dieting and treatment they go steadily downhill, waste, and eventually die.

(3) Vomiting. There may be a little post-anæsthetic vomiting; if persistent, however, it is a real cause for anxiety. It usually implies that the operation has been incomplete, and that not all the constricting fibres of the pyloric tumour have been severed. A number of these cases have consequently to be submitted to a second operation.

(4) Diarrhœa. This is the most dreaded post-operative complication, and is the cause of most of the deaths which occur. It is usually delayed in its onset, not arising until the second post-operative week.

(5) Intra-peritoneal hæmorrhage and peritonitis. Both these complications are due to errors in technique, and rarely occur.

(6) Sepsis of the wound. Burst abdomen, suppuration of the wound, and post-operative ventral hernia also imply faulty operative technique, and are therefore avoidable complications. They should be treated on the generally accepted lines.

(7) Chest complications. Pneumonia and other chest complications do not occur in more than 1-2 per cent of cases.

## CHAPTER II

### ACUTE DILATATION OF THE STOMACH

ACUTE dilatation of the stomach is a condition in which the stomach rapidly becomes distended with gas and fluid, and this is followed by severe collapse of the patient. In the majority of cases it is associated with intractable vomiting. It is most important to recognise the condition in its early stages, as, if undetected, death will almost invariably ensue.

It is often stated that the condition is rare, but with this I cannot agree. The reason for this statement is that very few cases are recorded and that quite a number pass unrecognised. Recently I have seen and treated no fewer than five cases, four of which occurred in the practices of other surgeons. Three of the patients recovered and two died. None of these cases was recorded. My own patient was a man of 54, who had had a nephrectomy performed for calculous pyonephrosis. All appeared to be going well until 36 hours after the operation when he developed all the signs and symptoms of acute dilatation. He died five days later in spite of intensive treatment on the lines which I shall later indicate.

Rokitansky (1842) was the first to describe dilatation and obstruction of the stomach by compression of the third part of the duodenum by the superior mesenteric artery. Brinton (1859) is credited with recording the first case. Hilton Fagge gave a lucid description of the symptomatology and pathology of the condition in the *Guy's Hospital Reports* (1872-3). Albrecht (1899) was able to collect nineteen cases up to that date, while Campbell Thomson (1902) published a paper and gave the records of forty-four cases. Box and Wallace (1911) published cases and gave results of some interesting experimental work. Borchgrevink, in a paper which must be regarded as a classic and which laid the foundations of the modern-day treatment of the condition, investigated the records of one hundred and forty-four cases from 1895-1913, and drew some very important inferences, specially with regard to treatment. Doolin (1918), in an authoritative paper dealing with the subject, skilfully assesses the value of the various methods of treatment, and lays down certain rules in the management of these cases which have not been bettered even to this day.

## ÆTIOLOGY

The true cause of acute dilatation of the stomach is unknown, but explanations offered by various authors may be briefly summarised as follows :

- (1) That the dilatation is due primarily to an obstruction of the third part of the duodenum by the superior mesenteric artery. (Rokitansky; Albrecht.)
- (2) That there is an excessive and abnormal secretion of gastric juice. (The "gastro-succorrhœa" of Morris; Fagge.)
- (3) That there is a primary paralysis of the stomach wall, this being followed by dilatation. When duodenal dilatation and obstruction are present it is a secondary phenomenon. (Campbell Thomson.)
- (4) That there is compression of the third part of the duodenum by the loaded and dilated stomach. (Box and Wallace.)
- (5) That the condition is due to septic intoxication.
- (6) That the primary factor is spasm of the pylorus.

None of these theories by itself fully explains the condition which is probably a combination of several factors. If there is a paralytic dilatation of the stomach it is conceivable that this may be maintained or complicated by mesenteric compression. Again, the paralysed and dilated stomach may, by its bulk, press upon and further block the third portion of the duodenum. Probably one of the most important ætiological factors is the compression of the third portion of the duodenum by the superior mesenteric blood-vessels, as this is evident in about one third to one half of all cases. Although the ætiology of the condition may perhaps be said to be found in the sympathetic nervous control of the stomach, the true primary activating factor in the disturbance is at present inexplicable.

Cope, in the Lettsomian Lectures (1933), writes : "The most reasonable explanation to account for the hæmorrhagic vomit and the gastric atony is retrograde venous embolism of the smaller gastric veins. This form of embolism has been well described by Payr and by Wilkie, and their experimental findings show that venous emboli can be carried along the omental veins to the veins on the greater curvature of the stomach, thereby leading to an infarction of the small veins of the gastric mucosa. If this took place on any considerable scale, it would certainly for a time interfere with the normal functions of the stomach, leading to bleeding into the viscus, and furnish a reason for the inability of the stomach contractions to overcome even a slight resistance to the overflow of the contents."

Both sexes are equally affected, and no age is exempt as the condition has been noticed in an infant of nine months old and in a patient of seventy-five years of age.

Acute dilatation of the stomach may occur :

(1) *During abdominal operations.* "Richardson and Lee have both recorded interesting cases where the dilatation commenced with startling suddenness during the course of an operation. Richardson's case was one of perforated duodenal ulcer which was operated upon and the perforation closed. At the commencement of the operation the stomach was small and collapsed, but while the abdominal wall was being closed it became so rapidly distended that this portion of the operation was difficult. The stomach was found to be drum-like and tense and as the distension increased it rolled upwards. On the passage of a stomach tube vast quantities of gas were passed, the stomach rapidly collapsed and the patient recovered. Lee's case also followed an operation upon the stomach. There was a chronic pyloric ulcer associated with stenosis for which a posterior gastro-enterostomy was performed. The dilatation developed during the course of the operation and the patient died. As Lee points out, this case was of special interest in proving that an acute dilatation could occur in spite of the fact that a posterior gastro-enterostomy had been performed." (Walton, *Surgical Dyspepsias*, p. 305, 1930.)

(2) *After abdominal operations.* About 70 per cent of all cases have been recorded as following abdominal operations. The condition is commoner after operations upon the pelvic organs, and then, in order of frequency, after operations upon the gall-bladder and biliary passages, appendix, herniæ and stomach. Not more than 5-10 per cent of cases are seen after operations upon the stomach. As has been stressed by Walton, acute dilatation may occur even after gastro-enterostomy.

(3) *After operations upon the extremities.*

(4) *Following an abdominal injury or fracture of one of the limbs.* I have seen two cases follow the setting of a severe fracture of the femur.

(5) *After child-birth.*

(6) *During an acute illness.* Borchgrevink (*Surg., Gynec., and Obstet.*, 662, June, 1913) and others have described cases occurring after the crisis of pneumonia and during typhoid fever. I have also known acute dilatation to occur in a patient recovering from acute septicæmia.

(7) *During chronic illnesses and debilitating diseases such as diabetes, phthisis, chronic nephritis, etc.*

(8) *Immediately following the rapid ingestion of a large meal or after an excessive bout of drinking.*

(9) *After genito-urinary operations.* The condition is not infrequently seen after operations upon the prostate and kidney.

(10) *In patients with deformities and diseases of the spine, e.g. spondylitis deformans, tuberculous disease of the spine, syphilitic myelitis, etc.*

As acute dilatation of the stomach may occur where no operation has been performed, it is quite obvious that intra-abdominal handling of the viscera is not the direct cause of the condition. Again, inhalation anæsthetics cannot be regarded as a contributory factor, as the condition has been seen after the administration of a local anæsthetic, or even where no anæsthetic has been given.

#### PATHOLOGICAL ANATOMY

The post-mortem appearances are quite typical (fig. 53). The enormously dilated stomach will be seen to occupy practically the

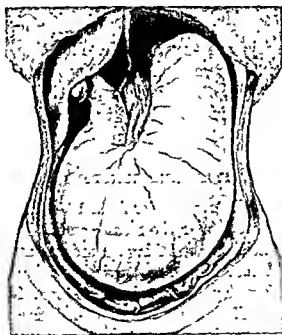


Fig. 53.—ACUTE DILATATION OF THE STOMACH.

whole of the abdominal cavity. The intestines are crowded into the pelvis or crammed behind the dilated viscus. The stomach is blown out and drum-like, and has a large descending left limb, which may extend into the pelvis, and a smaller ascending right limb. The stomach is U-shaped or V-shaped, and where the limbs join there is usually a deep indentation in the region of the incisura. In nearly

all the autopsy records the fact that there has been little or no peritonitis has been emphasised. When the outer surface of the stomach is carefully examined, numerous white striæ will be discerned. These are probably the result of the stretching. The walls are very thin and in some instances diaphanous, so that the watery contents can be seen. When the stomach is opened it will at once be evident that its walls are stretched to maximum capacity, that they are very thin and friable, that the mucous membrane has been compressed and flattened out, that the rugæ are indiscernible, and that scattered here and there are numerous small erosions. It is these superficial abrasions in the mucous membrane which account for the dark-brown or black material that is vomited during the late phases of the disease.

In some instances the dilatation does not extend beyond the pylorus. In the majority, however, it will be seen to involve the duodenum and to end abruptly where the mesenteric blood-vessels course over it. As noted above, this is the appearance presented in fully a third to a half of the cases described. In a case recorded by Baunier the constriction in this portion was very tight, and even led to necrosis of the mucous membrane. In a few cases, however, the upper coils of the jejunum shared in the dilatation, and proved conclusively that mesenteric compression could not have been an important causative factor. Again, other cases are seen in which the dilatation does not extend into or involve the duodenum, but ends abruptly at the pylorus.

#### SIGNS AND SYMPTOMS

The condition usually commences from 12-48 hours after operation but may be delayed as late as the second or third week. In a case I saw recently, the patient was making an uninterrupted recovery after an abdominal hysterectomy when, two and a half weeks after the operation and a few days before her discharge from hospital, she developed an acute dilatation of the stomach from which she died three days later. Cases have been known to occur *during* or *immediately after* an abdominal operation, but in the majority of cases the complication ensues about the second post-operative day. The most outstanding symptoms are vomiting, distension of the abdomen, and collapse.

(1) The vomiting may at first be indistinguishable from post-anæsthetic sickness, but there is usually first a calm before the storm, when the patient appears to be progressing favourably. The vomiting

is frequent, effortless, and unaccompanied by pain. With each vomit large quantities of gas may be belched up. The constant welling up of the stomach contents, attended by involuntary gushes through the mouth, is most distressing to the patient. The mouth becomes painful and sore, the patient is conscious of an acid burning sensation, and the eroding constituents in the voided material may sear the cheeks and the corners of the mouth. The vomiting is never projectile. In a few cases (about 10 per cent) it may be absent, and this is most noticeable where acute dilatation occurs after operations upon the bladder or prostate.

The vomited material is at first colourless, and later bile-stained, eventually becoming yellow-brown, brown, or even black. In the latter stages it is invariably dark brown or "coffee-grounds" in appearance, owing to the presence of altered blood. It is never feculent, but it may possess a peculiar musty odour. The amount vomited during the course of a day may be as much as several pints.

(2) *The abdominal distension* is preceded and accompanied by discomfort, pain, tenderness, tightness, and a sense of fulness. The epigastric pain is never very severe in character, but is persistent and obstinate. The distension is most marked in the epigastrium, particularly on the left side, and rapidly extends downwards, filling the left flank, bulging into the hypogastrium, and, when well established, producing a universal inflation of the abdomen. If the stomach is grossly distended with gas there will be a resonance in the epigastrium and over the lower half of the left side of the chest on percussion. The heart will be displaced upwards and will be embarrassed in its action. In other cases, when the stomach is full to overflowing with fluid, there will be a dull note on percussion of the epigastric region. Splashing noises will be clearly audible on palpation, but owing to generalised abdominal tenderness and exhaustion the patient will often resent anything more than a superficial examination.

(3) The signs of *collapse* are evident. The patient is pale, ashen-grey or lemon-tinted in colour. The eyes are hollow, but bright, active and attentive. Although exhausted almost to breaking point, feeble through continuous vomiting, and suffering great distress, the mind remains alert to the end, and the eyes will follow every movement and study each action of those around with the keenest interest. The hearing is acute, and the memory retentive. On recovery patients will describe most accurately all the details connected with the examinations performed upon them, the treatments they received, and the



particulars of even whispered conversations which took place in their presence. The pulse is rapid and weak, and in a severe case may be imperceptible at the wrist. The temperature is normal or sub-normal, and the respirations are quickened and thoracic in nature. The patient feels cold and shivery, the extremities are numbed and death-like, and the whole body is drenched in sweat. Thirst is a most distressing feature and is unquenchable. Hiccough, although present in a certain number of mild cases, is rarely seen in the more severe ones. The bowels are usually constipated, although in a few instances they may be normal or there may even be incontinence of fæces. The urine is scanty, highly coloured, of high specific gravity, and loaded with urates. There may be diminution in the blood chlorides, and other features of alkalosis may be in evidence in a certain proportion of cases.

#### DIAGNOSIS

If the condition is borne in mind very few cases will pass unrecognised. The following are sometimes mistaken for acute dilatation of the stomach :

- (1) Post-anæsthetic vomiting.
- (2) Acidosis.
- (3) Acute peritonitis.
- (4) Paralytic ileus.
- (5) High intestinal obstruction.
- (6) Intra-peritoneal hæmorrhage.
- (7) Acute poisoning by drugs.

Acute dilatation presents a picture which should be very easily recognised. The persistent vomiting, the dilated abdomen, the splashing sounds, the marked collapse, the enormous quantities of fluid and gas which can be withdrawn from the stomach by means of a stomach tube, the rapidly rising pulse-rate, should all readily indicate the true state of affairs.

#### PROGNOSIS

As the chances of a spontaneous cure are very slight, the results in undiagnosed cases are almost invariably fatal. Connor (1907) found on analysing all the recorded cases up to that date that the mortality was 72 per cent; Laffer's figures (1908) showed the death-rate to be 63 per cent; Borchgrevink (1913) recorded one hundred

and forty-four cases, and of these 54 per cent died. Doolin (*Br. Jl. Surg.*, No. 21, 125, 1918) writes :

"Within the past ten years there is an improving prognosis, largely depending upon more widespread recognition of the condition and better methods of treatment. We get a truer perception of the patient's chances if we analyse the mortality of the various methods of treatment adopted :

- (1) Of 31 cases treated medically, 29 died, a mortality of 93 per cent.
- (2) Of 29 cases treated by operation, 21 died, a mortality of 72 per cent.
- (3) Of 54 cases treated by lavage, 25 died, a mortality of nearly 50 per cent.
- (4) Of 30 cases treated by posture, 2 died, a mortality of nearly 7 per cent.

Thus, of 114 cases where postural treatment was not carried out, 75 died, giving a combined mortality of 65 per cent ; *whereas in the 30 cases where postural treatment was adopted, the mortality is reduced with a jump to below 7 per cent.*"

#### TREATMENT

There are various points which should be emphasised as far as treatment is concerned :

(1) *That under no condition whatsoever should an operation be performed.* As Doolin shows, the mortality of such operations as gastrostomy or gastro-jejunostomy is prohibitively high, and as the condition has been known to arise immediately after the performance of gastro-jejunostomy it is clear that this operation will be purposeless and unavailing.

(2) Postural treatment, aspiration and lavage of the stomach, intravenous and subcutaneous salines, and the administration of certain drugs constitute the method of successful treatment, and these will now be dealt with in detail.

(a) *Gastric aspiration and lavage.* As the stomach contents are of a watery nature a small-bore stomach tube should be used for aspiration and irrigation. The patient should be in the Trendelenburg position when the stomach tube is passed ; otherwise he may choke or even drown himself in his own vomit, as the irritation produced by

the passage of the tube may result in the sudden and involuntary welling up and flooding through the mouth of enormous quantities of fluid. It is quite a common experience to find that as soon as the tube is passed into the stomach the belching of a large amount of gas precedes the gushing stream of fluid. The tube, which is passed through the mouth or nostril, should be strapped to the side of the face with elastoplast, and aspiration and irrigation begun (figs. 54 and 55). All



Fig. 54.—ACUTE DILATATION OF THE STOMACH. A SMALL STOMACH TUBE HAS BEEN PASSED THROUGH THE MOUTH, AND THE CONTENTS OF THE STOMACH ARE ABOUT TO BE ASPIRATED.



Fig. 55.—ACUTE DILATATION OF THE STOMACH. A RYLE TUBE HAS BEEN PASSED THROUGH A NOSTRIL, AND IS FASTENED TO THE FACE WITH A STRIP OF ELASTOPLAST.

the fluid in the stomach should be aspirated, and irrigation with normal saline performed every half hour unless the patient is asleep. This aspiration and irrigation must be continued and persevered with until it is quite evident that the stomach is regaining its tone and is returning to its normal condition. It may even be necessary in certain cases to leave the tube *in situ* for 46 hours or possibly longer. With patients to whom the continued presence of this tube is intolerable it may be necessary to pass it at half-hourly and gradually lengthening intervals.

(b) *Posture.* As soon as it is recognised that the patient is suffering from an acute dilatation of the stomach the end of the bed should

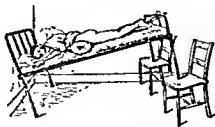


Fig. 56.—ACUTE DILATATION OF THE STOMACH. POSTURAL TREATMENT.

be raised at least 18 inches upon blocks or chairs. The patient should then be turned over, face downwards, with a bolster or pillow under

the pelvis (fig. 56). The head should be turned to the right or to the left side, while the arms should be slightly extended above the head. To prevent the patient from slipping down the feet should be tied with a towel or a thick cotton bandage to the transverse bar at the end of the bed. The patient is thus in the Trendelenburg position, but lying on his stomach. This position is a very uncomfortable one, and much patience and persuasion are often needed to induce the patient to adopt and maintain it, as it is very difficult to convince him or the nurses of the benefits that will follow the adoption of what must appear to them to be a most unnatural and undesirable position. The abdominal distension and tenderness, the painful pressure on the wound, and the grip of the bandage holding the feet, together with the inevitable embarrassment in respiration and in the action of the heart, the exhausted state of the patient, and the many difficulties of nursing all seem to combine to contra-indicate it. But in spite of everything postural treatment should be persevered with until the patient is well on the road to recovery, as it is one of the most influencing factors in the successful management of such cases. Relief may be afforded at intervals by changing the patient from the prone to the right or left lateral position, or even by allowing him to lie upon his back for a while, still maintaining the elevation of the feet, when the other positions appear to be taxing his strength and endurance unduly.

Postural treatment should be maintained for at least 2 days after all signs and symptoms have subsided. The blocks or chairs at the end of the bed are then removed, and the patient is made to lie flat on his back without a pillow until recovery is assured and there is no fear of relapse. The object of postural treatment is clear. The fluid which has collected in the stomach is displaced upwards by gravity, and can be more easily aspirated. The downward drag of the overlaid stomach on the intestine and the mechanical obstruction of the duodenum are relieved.

(c) *The administration of fluids.* Owing to the amount of fluid lost by vomiting and sweating, it is important to replace this by giving large quantities of saline. By this means the blood-pressure is rapidly restored, the failing circulation is re-established by the re-filling of the vascular system, and alkalosis is combated. It is usual to inject 100 cc. of 6 per cent saline intravenously as soon as the treatment is commenced, and 250–500 cc. of normal saline with 5 per cent glucose at varying intervals subsequently, or to introduce the salines and glucose into the circulation at a slow but steady rate by the continuous

drip method. No useful purpose is served by giving fluids by the mouth.

(d) *Drugs.* There are no specific drugs used in the treatment of acute dilatation of the stomach. 1 cc. of pituitrin may be injected every 3 hours for six doses, and morphia,  $\frac{1}{4}$  gr., to relieve pain and to induce sleep.

## CHAPTER III

- (A) ACUTE PHLEGMONOUS GASTRITIS
- (B) INJURIES OF THE STOMACH
- (C) FOREIGN BODIES IN THE STOMACH

### (A) ACUTE PHLEGMONOUS GASTRITIS

THIS condition, occasionally termed acute suppurative cellulitis of the stomach or gastric phlegmon, is extremely rare, less than 300 cases being reported in literature. Watson (*Amer. Jl. Surg.*, xviii, 113, 1932) in a review of the subject, stated that 266 cases had been reported up to 1932. It is due in most cases to a spreading streptococcal infection of the submucous coat of the stomach.

*Ætiology.* The disease may occur at any age and has been known to affect a child of 11 and a patient of 70. As a rule, however, those affected are young adults, the majority being in the third decade. Males are more liable to the infection than females, Adams stating that the proportion is approximately 5 to 1.

The following are predisposing factors :

- (1) Injury to the stomach.
- (2) Gastric operations.
- (3) Peptic ulcer—acute or chronic.
- (4) Cancer of the stomach.
- (5) Acute specific fevers ; septicæmia ; erysipelas.
- (6) Alcoholic excess.
- (7) Constant dietetic indiscretions.

The disease is said to be *primary* when it arises spontaneously in an apparently healthy individual, and *secondary* when it follows an acute specific fever or some local lesion in the stomach. The majority of cases, however, are of metastatic origin and associated with some generalised infection. It is due to an infection of the submucous coat of the stomach with micro-organisms. These organisms, which may include

streptococci (present in 70 per cent of cases), *B. Welchii*, *B. coli*, *B. proteus*, and pneumococci, may gain access to the submucous tissues either through the blood stream, or directly through a breach in the mucous membrane such as might be caused by an acute erosion, a chronic gastric ulcer, or a breaking-down malignant growth.

*Pathology.* Two forms of the condition are recognised: the *diffuse* and the *circumscribed*, the diffuse form occurring about twice as frequently as the circumscribed. The organisms having gained a foothold in the submucosa multiply and rapidly infect this tissue, which is so richly supplied with lymphatics. The infection may remain localised to one portion of the stomach, leading to the production of a circumscribed abscess. In the majority of cases, however, the whole or the greater part of the stomach becomes affected. There is an intense inflammation of the submucous coat of the stomach, the profuse exudate being composed of serum, fibrin, pus, and organisms. The mucous membrane itself may be normal in appearance or it may be smooth, flattened out, and devoid of rugæ. Nevertheless, cases have been recorded where the mucosa has been very congested and hæmorrhagic, œdematous, and markedly rugose, diffusely and superficially ulcerated, locally necrotic, or sloughing away in strips over a wide area. The muscular coat becomes water-logged, soft and boggy, being also affected by the inflammatory process. When the serosa becomes involved acute peritonitis, either generalised or localised to the region of the stomach, develops with incredible rapidity. In such cases the serosa becomes reddened and covered with adherent flakes of yellowish fibrin.

The whole stomach wall may become markedly thickened and friable, and this thickening may be increased to as much as six times the normal width. When an incision is made through the serous and muscular coats of the organ into the submucosa in the infected area, a considerable amount of pus and blood-stained watery fluid will pour through the wound.

In the diffuse form, although the whole stomach is usually affected, the changes are most marked in the pyloric portion.

*Symptoms.* The onset is sudden, with cramp-like epigastric pain, vomiting, and immediate prostration. The temperature is raised and the pulse is rapid. The face is flushed, pinched and drawn, and the eyes are glistening and hollow. The tongue is dry and thickly coated, later becoming deeply fissured. Thirst is insatiable, and although in certain cases there may be diarrhœa, the bowels are usually constipated.

At the very inception of the disease the patient becomes gravely ill, toxæmic, and collapsed. Symptoms of peritonitis develop early. The epigastrium, which may be distended, is tender on palpation, and there is localised rigidity which becomes generalised when general peritonitis supervenes. In the majority of cases there is a marked leucocytosis. The vomit at first consists of normal stomach contents, and later of bile; if a large localised abscess forms and bursts through the mucous membrane it may contain pus. In the circumscribed form it may be possible to feel a mass in the epigastrium, and more often than not a diagnosis of perforated gall-bladder with localised abscess will be made.

The disease is not invariably fatal, but in the majority of cases death will occur from acute toxæmia or from septic peritonitis after five or six days.

*Diagnosis.* A correct diagnosis is rarely possible without an exploratory operation, although in one of Watson's cases thin turbid slightly reddish fluid was obtained on abdominal puncture, and the presence of streptococci in the aspirated fluid confirmed a diagnosis of phlegmonous gastritis. The symptoms in most cases are indistinguishable from those of an acute abdominal catastrophe such as a perforated peptic ulcer, acute hæmorrhagic pancreatitis, or acute fulminating cholecystitis.

*Treatment.* The localised form of the disease, and especially where a circumscribed abscess is found in the stomach wall, should be easily recognised at operation. Additional proof may be gained by aspirating pus from the fluctuating or sodden stomach wall at various points.

In certain cases, simple drainage of the abscess may be indicated. If, however, the disease is localised to the pyloric portion of the stomach partial gastrectomy may be performed, and the results, as confirmed by Gerster, are satisfactory. When the generalised variety is present little can be done apart from a palliative jejunostomy and drainage of the peritoneum. But Weinstein, Klein, and Hamilton Bailey recommend delivering the stomach through the abdominal wound, and maintaining it there by a glass rod, as in Paul's operation of colostomy, after making a number of small vertical incisions through the sero-muscular coats of both walls of the stomach down to the purulent submucosal layer.

The lesser sac is drained with a small rubber tube and the upper and



lower extremities of the wound are sutured without unnecessarily constricting the exteriorised stomach. At the completion of this operation a jejunostomy is performed through a small separate incision, whereby the patient may be fed.

The prolapsed stomach is covered with several large hot moist packs which are frequently changed. If progress is satisfactory, and it is evident that the acute inflammatory process in the stomach is subsiding, the glass rod is removed, allowing the stomach to sink back into the abdominal cavity. Secondary suture of the middle part of the epigastric wound will usually be necessary.

In the immediate post-operative period large doses of anti-streptococcal serum should be given; intravenous antiseptics, such as mercurochrome, may also be tried. Saline, to which glucose may be added, should be given intravenously, subcutaneously, or per rectum, as dehydration and toxæmia are always very marked in these cases. Pain must be relieved by liberal doses of morphia.

## (B) INJURIES OF THE STOMACH

### *Causation.*

#### (1) Violence from without.

- (a) *Indirect*, such as may be caused by blows to the epigastrium, often with subcutaneous contusions.
- (b) *Direct*, with external wound, the result of gunshot wounds, stabs, etc.

#### (2) Violence from within.

- (a) The passage of instruments into the stomach, e.g. gastro-scope.
- (b) The swallowing of large quantities of corrosive fluids, or of sharp foreign bodies which cause mechanical injury.
- (c) Over-distension of the stomach with gas or fluids.

### *Varieties.*

#### (1) Complete rupture of the stomach.

#### (2) Incomplete rupture of the stomach:

- (a) Of the serous coat.
- (b) Of the muscular coat.
- (c) Of the mucous membrane.
- (d) A combination of any two of the above.

*Spontaneous Rupture of the Stomach.* This is an extremely rare condition, and all the reported cases have proved fatal. The rupture occurs in the region of the lesser curvature, and is due to a spontaneous over-distension which may be occasioned by some severe muscular effort, such as vomiting or lifting a very heavy weight.

As soon as the rupture occurs the patient is seized with a violent attack of abdominal pain, and all the clinical phenomena are indistinguishable from those of an acute perforated peptic ulcer, with profound collapse, demanding immediate exploratory operation.

*Traumatic Rupture.* It is most exceptional to find a rupture of the stomach due to injury apart from some accompanying damage to other viscera. The commonest single cause of rupture of the stomach is a run-over accident or a fall from a height, e.g. aeroplane crash.

Rupture from external violence usually occurs in the region of the greater curvature, and owing to the severity and extent of the laceration of the stomach and also of the associated injuries, such as rupture of the liver, spleen, intestines, etc., such an event is almost invariably fatal.

A complete or partial rupture of the stomach may follow distension with fluid used for purposes of lavage or from deliberate distension with gas as a means of diagnosis. In cases of rupture of the stomach due to over-distension the rent usually occurs in the lesser curvature and nearer the cardia than the pylorus. When, on the other hand, rupture occurs as a result of the employment of the above-mentioned methods in the treatment or diagnosis of a gastric ulcer or malignant growth, the rupture generally takes place in the immediate vicinity of the existing lesion.

In cases of penetrating wounds, e.g. gunshot wounds, the abdomen must in all cases be explored without delay. The abdominal wound should be excised and the abdomen explored through an ample incision. The entire stomach must then be examined, and this is particularly essential where there has been a wound of the anterior wall, for in such cases there is frequently injury to the posterior wall also which might otherwise be overlooked.

Such wounds of the stomach should be closed with a three-tier suture, but if the edges are jagged and have an unhealthy appearance the wound should be excised and carefully sutured.

In cases of rupture of the stomach due to indirect violence, if operation is performed within twenty-four hours of the infliction of the injury the prognosis is tolerably good. If, on the other hand, the injury is due

to gunshot wounds, the prognosis will in any case be very grave, the mortality being about 50 per cent, whilst where there are associated injuries to neighbouring viscera it may be even higher than 70 per cent.

### (C) FOREIGN BODIES IN THE STOMACH

Foreign bodies of many varied shapes and sizes have from time to time found their way into the stomach. Some of these are of such a shape that it is remarkable that they should have travelled down the œsophagus and passed the sphincter at the cardiac end of the stomach. Some, of course, do become impacted in the œsophagus, and may cause ulceration with its attendant dangers, such as perforation, mediastinitis, or subsequent stenosis.

Foreign bodies are found chiefly in the stomachs of children and of those of unsound mind, although they are not infrequently swallowed by normal adults, either accidentally or as a means of escaping punishment, e.g. convicts; some, on the other hand, may do it out of sheer bravado or to excite sympathy. It is impossible to give a complete list of objects which have entered the stomach, but these include knives, forks, coins, buttons, stones, keys, whistles, etc. Pins, safety-pins open or closed, and nails are frequently swallowed by dressmakers and boot-makers owing to their habit of holding these objects in their mouths. The article most frequently swallowed is probably the denture, and if large it may become impacted in the œsophagus. In the case of operation, before an anæsthetic is administered it is important to ascertain that all dentures have been removed, as these may not only be swallowed, but, of greater importance, they may enter and obstruct the air-passages with possibly fatal results.

Another and more rare foreign body is a hair ball or trichobezoar. This consists of a collection of human hair, horse hair from mattresses, or of twine or similar material, matted together so as to form a rounded mass. When large it may fill the whole cavity of the stomach and form a cast of that organ.

A mass similar to a hair ball may occur in workers in the cotton trade through the habit acquired by some of them of frequently swallowing pieces of cotton. Large quantities of undigested food composed of vegetable fibres sometimes form a solid tumour in the stomach, which may be so large as not only to fill the whole stomach but to project into the duodenum and œsophagus, and contain in its meshes foul decomposing food residues.

*Symptoms.* Foreign bodies in the stomach do not necessarily give rise to symptoms, and many objects may be present for some time, remaining unsuspected until discovered on X-ray or post-mortem examination. This is especially the case with lunatics, the classical example being that recorded by Vandervert and Mills, who found in the stomach of a lunatic who died of renal disease no fewer than 1446 objects, including nails of all sizes, safety-pins, needles, buttons, and spoons. These had given rise to no obvious symptoms during life. Usually objects such as buttons, small coins, marbles, etc., pass through the pylorus and travel the length of the alimentary canal without mishap, eventually being evacuated per anum with the faeces. The time taken on this journey varies and may be as long as 48 hours or more, since the object may be held up in the stomach for some time. Sharp bodies may wound the gastric mucosa and cause ulceration which may be followed by perforation, but needles have been known to penetrate the stomach walls without giving rise to any symptoms. Sharp objects may also travel the whole length of the alimentary canal and be passed with the faeces, or they may become impacted in the anal canal giving rise to an abscess. As has been stated, large objects may remain in the stomach without causing symptoms, although the danger of perforation is constantly present, and the longer they remain in the stomach the greater the danger. If perforation occurs the symptoms differ in no way from those due to perforation of a peptic ulcer with general or local peritonitis. In other cases vague epigastric discomfort, with sudden bouts of colicky pain due to the object becoming wedged across the stomach, is complained of by the patient. Again, the foreign body may pass out of the stomach and become impacted lower down in the intestine, producing symptoms of intestinal obstruction or of perforation and peritonitis.

A hair ball may be felt in the epigastrium as a hard mass, painless on palpation, and freely movable. This, associated with discomfort or pain after meals, vomiting, flatulence, and loss of weight due to the difficulty of food percolating through the mass when it is large, will in some cases lead to a diagnosis of carcinoma which may even appear to be confirmed by X-rays, the true condition being discovered only at operation. The majority of cases, however, can be correctly diagnosed nowadays by an X-ray examination of the stomach.

*Treatment.* All cases should first be X-rayed in order to confirm that a foreign body has actually been swallowed, and, if present, to determine its position. As the majority of foreign bodies swallowed

are opaque to X-rays, the diagnosis may at once be confirmed or negatived by these means. In children it is not uncommon for the parents to allege that an object has been swallowed just because it is missing, or because the child was recently playing with it and since that time it has disappeared.

In all cases the chest should be included in the X-ray examination in order to verify that the foreign body has not passed into the trachea or bronchus or remained in the œsophagus, although when this is so the history and symptoms will usually indicate such an occurrence. The possibility of error through a hasty screening examination with incomplete removal of clothing should not be overlooked; dentures in a patient's pocket have been thought to be in the abdomen.

The course of a small foreign body can be followed by screen examination until it is passed per anum. The stools should be voided into a chamber containing water, and examined until the object is recovered. Nothing further need be done if small smooth objects are seen to be travelling down the intestines. No purgatives must in any case be given. With small sharp objects, such as pins or nibs, which are likely to pass the pylorus, a large quantity of soft pulstaceous food should be given so as to form a kind of cushion around the object and protect the mucous membrane. Suitable food includes the soft part of bread, porridge, potatoes, rice, and tapioca. Large doses of normacol may be given at frequent intervals to embed the foreign body in a gelatinous mass, thus aiding its downward passage and protecting the walls of the stomach and intestine.

Larger objects, such as knives, forks, and any smaller foreign body which is still retained and appears to be impacted in the stomach, demand surgical interference. An attempt may be made to remove them with a gastroscope, but this is a difficult procedure which should only be undertaken by one expert in the use of the instrument, and often ends in failure as the foreign body frequently lies hidden between the folds of mucous membrane or is submerged in a pool of gastric juice. The best method is to open the abdomen and remove the object by *gastrotomy*. Hair balls should be dealt with in the same way, and any underlying neurotic condition subsequently treated. It should be stressed that immediately before operation is undertaken a final X-ray examination should be performed to make quite sure that the object has not, at the last moment, slipped through the pylorus.

## CHAPTER IV

### PEPTIC ULCER

PEPTIC ulcer is a common disease with an incidence which indicates that its prevalence is increasing, as is well shown by the results of radiological investigations, abdominal operations, and post-mortem examinations. Hart (*Mitteil. u.d. Grenzgeb. d. Med. u. Chir.*, xxxi, 350, 1918-19) found open peptic ulcers or scars of healed ulcers in 12 per cent of the 3058 autopsies of all diseases (adults only) investigated by him. Stewart (*B.M.J.*, ii, 1021, 1923) found healed scars or a chronic gastric or duodenal ulcer present in about 10 per cent of 4000 post-mortems conducted at Leeds.

"Hargis and Robertson in a routine study of material from 2000 necropsies found evidence of healed or active duodenal ulcer in 237 instances, that is, in about 12 per cent of all necropsies. In addition to this 141 gastric ulcers were found. In 7 per cent of all these cases both gastric and duodenal ulcers were present. It is interesting to note that 21 per cent of the duodenal ulcers and 40 per cent of the gastric ulcers were healed. Of still greater importance is the fact that in 6.4 per cent of these cases ulcer was not even suspected." (A. B. Rivers, *Coll. Papers Mayo Clinic*, xix, p. 83, 1927.)

It may therefore be estimated that approximately 10 per cent of all individuals at some period of their existence suffer from peptic ulceration. Chronic gastric ulcer is rarer than chronic duodenal ulcer, although the incidence of the two diseases was formerly thought to be about the same. All available statistics tend to prove that, as the result of the exigencies of modern methods of living, duodenal ulcer is yearly increasing in frequency. The rarity of chronic gastric ulcer is also emphasised by Balfour, who states that only 1.7 per cent of operations for intra-abdominal disease, performed at the Mayo Clinic, are for this condition, whereas over 10 per cent are for chronic duodenal ulcer.

### ACUTE ULCER

Acute ulcer may be defined as the digestion of a circumscribed portion of the stomach wall by the gastric juice. Before ulceration can occur the mucous membrane must be subjected to some form of damage. In some instances this damage may be evident as

a patch of necrosis, a submucous hæmatoma, or as an abscess of a lymphoid follicle. In others its origin is unrecognised. Acute ulcers are sometimes termed acute erosions, hæmorrhagic erosions, or follicular ulcers, depending upon the visible lesion from which they arise. For instance, hæmorrhagic erosion is a superficial loss of substance in the mucous membrane, resulting from a small hæmorrhage. As the distinctions are very fine it would be preferable simply to apply the term "acute ulcer" to all these lesions.

One of the main factors in the production of damage to, or devitalisation of, the mucous membrane is infection. This is proved by the fact that oral sepsis, general peritonitis, septicæmia, burns, and other septic conditions are so often associated with acute peptic ulceration. The devitalised and necrotic areas in the mucosa become digested by the gastric juice, leading to the formation of acute ulcers. In the majority of cases these heal spontaneously in the course of a few days or weeks, but in others

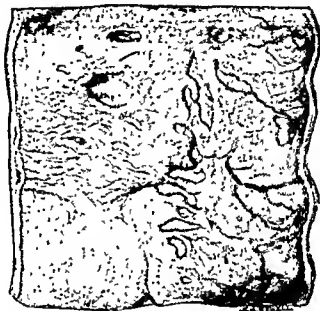


Fig. 57.—SUB-ACUTE GASTRIC ULCER AND SEVERAL ACUTE ULCERS. PORTION OF A STOMACH ALONG THE LESSER CURVATURE OF WHICH THESE ARE SEVERAL SMALL CIRCULAR, SLIT LIKE, OVAL, AND SHARPLY DEFINED ULCERS IN THE MUCOSA.  
(Museum, "Royal College of Surgeons.")

they persist and eventually a sub-acute or chronic ulcer results. Chronicity is maintained by the action of the acid gastric juice, infection, and other factors which will be considered in due course.

In the proportion of 3 to 1 acute ulcer is commoner in females than in males, and in the stomach than in the duodenum. No age is exempt; although occasionally seen in infants it is rare before puberty, most frequent between the ages of 15 and 30, and has a rising incidence later in life, after the age of 50, owing to its frequent association with malignant disease. It is seen in conjunction with a separate chronic ulcer in 4 per cent of autopsies, but in life the incidence may be as high as 10 per cent. Acute ulcers are more frequently associated with chronic ulcers in the duodenum than in the stomach. They are frequently observed in cancer of the stomach, and may be the primary cause of severe hæmatemesis. They are usually multiple, but in individual cases there is great variation in the number. Single acute ulcers are very much more frequent in the duodenum than in the stomach. The size and shape of acute ulcers

vary considerably (fig. 57). They may be oval, round, polyhedral, triangular, crescentic, or linear. The majority are minute, just visible to the naked eye, while others may be as large as an inch or more in diameter. They may be found in any part of the stomach, but show a preference for the lesser curvature and the pyloric portion. It is exceptional for acute ulcers of the duodenum and stomach to co-exist, as has been shown by Hurst and Stewart (*Gastric and Duodenal Ulcer*, 1929), who found that in a total series of 151 cases ulcers were present in the stomach alone in 111, in the duodenum alone in 34, and in both organs in only 6.

On section an acute ulcer appears to be punched out of the mucous membrane, has sharp, clear-cut edges, and a base which is covered with black altered blood, while in duodenal ulcer the base may be green from staining by bile. Usually there is no inflammatory reaction to be seen around the ulcer, although œdema and mild congestion of the margins may be observed in some cases. A characteristic feature of acute ulcers is that the mucous membrane is healthy right up to the margin. There may be penetration of the submucosa and even of the superficial fibres of the muscularis. But even in those cases where there is a breach of the muscle layer there is no evidence of any appreciable reactionary inflammatory change. The submucosa is not thickened, nor is there any evidence of granulation tissue in the floor of the crater. The majority of these ulcers heal without leaving a scar. Where, however, there has been destruction of the muscularis, on healing there will be a small puckered scar, which is visible only from the mucous aspect of the stomach, is stellate, and has fine lines radiating towards the centre. Instead of healing the ulcerative process may extend superficially with a wide destruction of mucous membrane, or may deeply penetrate or completely breach the muscular coat, and produce the so-called "terraced" or "cone-shaped" ulcer. In very exceptional instances an acute ulcer may erode all the coats of the stomach and give rise to a perforation, but such an occurrence must be exceedingly rare. Melena is frequent, while hæmatemesis in a mild or severe form is a well-known complication.

*The factors concerned in the prevention of the healing of acute ulcers are persistence of sepsis, increase in the acidity of the gastric juice, and spasm or stenosis of the pylorus.*

Acute ulcers give rise to no typical diagnostic signs and symptoms during life, except when such complications as perforation or hæmatemesis occur. Acute gastric ulcer may occasionally occur in association with chlorosis, often accompanied by hæmatemesis (so-called *gastrostaxis*), epigastric pain, and nausea; but chlorosis has practically disappeared owing to improved modern hygienic developments. In such cases the appearance has been noted at operation of a widespread or generalised oozing of the gastric mucosa rather than of discrete ulceration.

The treatment of acute ulcer is medical, and except in the presence of perforation, operation is never advised.

### SUB-ACUTE ULCER

Sub-acute ulcers are usually symptomless and of little clinical importance. They may, however, give rise to severe hæmorrhage or even perforate. It is the chronic lesion which is chiefly responsible for the symptomatology of ulcer. Sub-acute ulcer cannot, however, be ignored as a pathological entity, as it has certain well-marked and distinctive characteristics. The life history of these lesions is short, but they mark the intermediate stage between the acute and chronic forms. Those seen at operation or at autopsy are certainly not less than two months old. An acute ulcer may be said to have become sub-acute when it has persisted for some weeks, and when instead of healing there has been a definite continuation and extension of the ulcerative process. This causes such alterations in the characteristics of the lesion that finally



the differentiation between a sub-acute and a chronic ulcer becomes almost impossible except with the aid of a microscope.

Sub-acute ulcers may be single or multiple, and they are frequently associated with healed or healing acute ulcers. (See fig. 57.) They vary in size from 5 to 25 mm. in diameter, but the average single sub-acute ulcer, although larger than an acute, is smaller than a chronic. They are found mostly in the region of the lesser curvature, and occasionally in the pyloric canal. They are irregular in outline, deeply excavated, and have elevated margins. The edges are undermined and overhanging, but flatten out when healing is in progress, and eventually merge into the floor of the ulcer. They may be funnel-shaped or terraced, but rarely penetrate deeply into neighbouring viscera. The floor is usually formed by the muscularis, but sometimes by the subserosa. The base of the ulcer is covered by a thin layer of necrotic material which may be black with altered blood or stained green with bile. When situated in the duodenum it is not possible to differentiate between a sub-acute and a chronic ulcer. Sub-acute ulcers, when healed, leave a visible, puckered scar. The complications to which they give rise will vary according to the site of the lesion; when situated on the anterior wall they may perforate, when on the posterior, hæmatemesis may occur, whilst those involving the pyloric canal and associated with a considerable degree of œdema may produce obstruction. It is the large shallow type of sub-acute ulcer which bleeds, whereas the cone-shaped and funnel-shaped types perforate.

The treatment of sub-acute ulcer is medical except where such complications as pyloric stenosis, perforation, and sometimes hæmorrhage, demand operative measures.

### CHRONIC PEPTIC ULCER

#### (A) *Ætiology.*

Acute ulcers are more common in women, chronic ulcers in men. Cases of duodenal and of gastric ulcer are more frequent amongst males than among females in the proportion of at least 3 to 1. Duodenal ulcer is becoming commoner, gastric ulcer rarer; the former occurs more frequently in both sexes, whilst coincident duodenal and gastric ulcers are by no means uncommon. Probably more than 80 per cent of all chronic peptic ulcers occur in the first part of the duodenum.

Chronic peptic ulcer is very seldom seen before the age of 15, but a number of cases have been operated upon for this condition before this age. Many patients give a history dating back 5, 10, 15, or more years, and on careful inquiry a story of attacks of indigestion and other intestinal symptoms in adolescence, suggestive of the presence of an ulcer, can be elicited.

In 1933 I performed partial gastrectomy upon two patients for chronic gastric ulcer, one a man of 72, and the other a woman of 75. In the case of the man typical symptoms had been present from the age of 28, that is for 44 years. The woman had been treated on and off for "ulcers of the stomach" from the age of 17, that is for 58 years, having been operated upon at the age of 46, when a lesion was found in the

stomach which was considered to be an inoperable carcinoma. At operation I found a large ulcer penetrating the pancreas and producing hour-glass constriction. In both cases recovery was uneventful.

Balfour (*Coll. P. Mayo Clin.*, xix, p. 129, 1927) found the average age of patients operated upon for gastric ulcer to be 47 years, for duodenal ulcer 43, whilst the average age for the onset of symptoms was 39 years for gastric ulcer and 32 years for duodenal ulcer.

No particular occupation appears necessarily to predispose to chronic peptic ulceration, although a greater incidence has been noticed amongst the professional classes, the Services, athletes, and office workers. Amongst doctors duodenal ulcer seems to be specially prevalent.

Hurst (*Guy's Hosp. Rep.*, lxxi, 450, 1921) has described two constitutional types of stomach and individual—the gastric ulcer type, and the duodenal ulcer type. These he terms *the hyposthenic gastric diathesis* and *the hypersthenic gastric diathesis*. A definite family history is obtained in about one-third of ulcer cases.

Other ætiological factors to be considered are over-smoking, excessive consumption of alcohol, undue fatigue of mind or body over prolonged periods, worry, insufficient mastication of food, unsuitable diet, hasty or irregular meals, vitamin deficiency, exposure to extremes of temperature, seasonal influence, and racial characteristics. The disease is more common in the northern countries than in the southern, and appears to be particularly prevalent in England, other European countries, and America. It is rare in China—a nation of vegetarians—and in certain parts of India where rice constitutes the staple form of diet.

### (B) Pathogenesis.

(1) *The Ulcer Diathesis.* There is good reason to agree with Hurst that there are two diatheses in connection with chronic peptic ulceration—(a) a general, and (b) a local or special. In the former there is an abnormal acidity of the tissues, the excess of which is excreted by the stomach which, under certain circumstances, would predispose to the production of peptic ulcer. Hurst's well-supported assertion is that the local diathesis is determined by the shape, size, and motility of the stomach, the constituents of the gastric juice, and by general physique. For instance, in the hypersthenic gastric diathesis (duodenal diathesis) certain factors are constant; the patients are usually men of the vigorous, intellectual and emotional type, in whom the stomach

is small, transversely placed, steer-horn in shape, and associated with rapid emptying and an excess of hydrochloric acid (fig. 58). After the consumption of food this eager stomach deals rapidly and efficiently with its contents, so that it is empty and ready for more food in a very short space of time. Gastric juice with its excess of hydrochloric acid is produced in great quantity in the empty stomach, and by the expulsion of its undiluted contents for many hours during the day with great force into the duodenal bulb—the annexe of the stomach—the process of neutralisation of the acid in the duodenal bulb is outstripped.

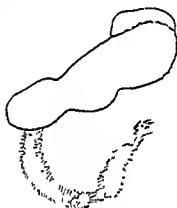


Fig. 58.—"STEER HORN" STOMACH. THE DUODENAL CAP LIES BEHIND THE PYLORIC END OF THE STOMACH. (H. Cecil Bell.)

Although this is unproductive of symptoms in and quite compatible with perfect health, where there is impairment of the vitality of the mucous membrane such as would result from a patch of necrosis, the supreme factor in the production of an ulcer is ready to hand. Only where these conditions are present can a duodenal ulcer occur. A gastric ulcer is never seen where the stomach is of the "steer-horn" type unless certain complicating factors are present such as pyloric stenosis or chronic duodenal ileus.

Hurst admits that it is much less easy to define what are the predisposing factors to gastric ulcer—what, in fact, constitutes the gastric ulcer diathesis. Patients with the hyposthenic gastric ulcer diathesis are listless and feeble, in comparatively poor health, have long J-shaped

stomachs reaching to the pelvis, and in comparison with the duodenal type there is diminished motility and gastric secretion. In such stomachs there is often a pull on the lesser curvature, which would influence the production of an ulcer at this site.

(2) *The Acid Factor.* The importance of hydrochloric acid in the production and perpetuation of peptic ulceration is well recognised. Peptic ulceration can only occur in those portions of the gastrointestinal tract which are regularly bathed with acid gastric chyme.

The presence of heterotopic gastric mucous membrane would account for the production of peptic ulcer in the œsophagus and in a Meckel's diverticulum. Almost the only circumstances in which a peptic ulcer can occur in the jejunum is where this portion of the gut is anastomosed to the stomach, as in the operation of gastro-jejuno-stomy which exposes the jejunum to the influence of the gastric juice to which it is unaccustomed.

I have, however, seen one case of a *chronic jejunal ulcer* which had perforated, producing generalised peritonitis. The lesion was situated about six inches from the duodeno-jejunal flexure on the anti-mesenteric border of the gut, and in appearance simulated in almost every respect the perforation of a chronic duodenal ulcer. The ulcer was excised and the aperture closed with a three-tier suture. Examination of the stomach and duodenum showed these organs to be perfectly normal. The patient, who was a man of 32, made a good recovery.

A gastric ulcer cannot develop in the absence of acid. It has been found difficult, therefore, to explain those rare cases where an ulcer is associated with achlorhydria, but they are thought to be due to the chronic gastritis which accompanies chronic ulceration of the stomach. The theory that the mucus in these cases may prevent gastric secretion by blocking the mouths of the ducts, or by rendering the acid excreted inert by neutralising it or by combining with it, appeals to me as being reasonable and sound. After gastric lavage the gastritis will subside, and fresh samples of gastric juice will again show the presence of acid. In diseases such as pernicious anæmia, where there is an acidity, gastric ulcer has never been known to occur.

Although acid gastric juice is the most important factor in the production of peptic ulceration, it is not the only one. It has, of itself, no "elective affinity" for any particular portion of normal gastric mucous membrane. Before ulceration can develop there must be a devitalised or damaged area produced by some other agency, such as bacteria or their toxins, which paves the way for the digestion of the necrotic tissue by the pepsin of the gastric juice which can only act in the presence of free hydrochloric acid. The appreciation of the acid factor in the

pathogenesis of ulcer is of great importance in treatment, as both medical and surgical measures aim at neutralising, diminishing, or controlling its production.

(3) *The Toxic Factor.* There is abundant evidence, both clinical and experimental, to show that in a large number of cases the initial lesion in the gastric or duodenal mucous membrane is caused by toxins which may be of bacterial or non-bacterial origin. It is well known that the toxæmia of acute infections may give rise to acute ulcers, and that acute ulceration is frequently seen in cases of severe sepsis, such as that accompanying general peritonitis, acute appendicitis, septicæmia, acute infection of the gall-bladder, burns, etc. The infection in the majority of cases is blood-borne, although in some instances the swallowed bacteria from infected teeth or tonsils may play a small part in perpetuating ulceration.

Rosenow (*Jl. Inf. Dis.*, xix, 333, 1916, and xxxiii, 248, 1923) isolated non-hæmolytic streptococci from the walls of chronic gastric ulcers, from the neighbouring infected lymph glands, and from septic teeth and tonsils, in patients suffering from ulcers. The inoculation of animals with these organisms led to the production of both gastric and duodenal ulcers in a significantly high proportion of cases. He claimed that these bacteria were specific and had an "elective affinity" for the mucous membrane of the stomach and duodenum, as, when streptococci were used from other foci of infection in patients without ulceration, a peptic ulcer resulted in only a very few instances. Although his views are not generally accepted, his experiments have been repeated with confirmatory findings. It would seem, therefore, that bacteria originating in a septic focus may pass into the blood stream and be excreted by the mucous membrane of the stomach or duodenum, and that they may produce necrosis of the mucous membrane which subsequently becomes digested by the gastric juice, leading to the production of an ulcer.

The natural tendency for acute ulcers is to heal rapidly, but new ulcers will continue to develop if the primary cause of the infection is not eradicated. The removal, therefore, of all septic foci is of great importance, both with regard to the prevention of peptic ulceration and in its treatment, as is amply shown by the results following such measures.

(4) *The Theory of Neurogenic Causation.* J. Shelton Horsley, in his monograph, *Surgery of the Stomach and Duodenum* (Kimpton, 1933),

discusses this theory and gives a summary of the views now generally held on this subject. He writes:

"Harvey Cushing emphasises the relationship between peptic ulcers and stimulation of the interbrain. His study was inspired by the loss of three patients after operations involving the base of the brain. One patient died of perforation of the œsophagus, another of perforation of the stomach, and the third of perforation of the duodenum, all of them within a few days after the operation. He expounds the theory of the neurogenic causation of peptic ulcer, particularly as described by Carl Rokitsansky, and after a careful review of the literature and from his own experience he concludes that, while there may be different causes for peptic ulcers, many of them are probably of neurogenic origin. Cushing calls attention to the important part that the interbrain plays in connection with the parasympathetic system. It seems probable that there is a parasympathetic centre in the tuber cinereum in the diencephalon. From this centre, according to Cushing, fibres go backward relaying with the cranial autonomic system of the midbrain and of the medulla of which the nucleus of the vagus nerves is the most important. Experimentally, lesions in the intracranial course of these fibres from the anterior hypothalamus to the vagal centre seem to cause gastric erosions, perforations, or ulcers. This is apparently due to parasympathetic stimulation, though possibly it may be from the suppression of the sympathetic stimuli. A satisfactory nerve balance between the parasympathetic or vagotonic impulses on the one side, and the sympathetic on the other, leads to normal conditions. Excessive stimulation of the parasympathetic nerves, or suppression of the sympathetic impulse which makes the parasympathetic dominant, may result in vagotonic impulses that induce hypersecretion of the stomach, hyperchlorhydria, hypermotility and hypertonicity, particularly in the pyloric segment of the stomach. These are the phenomena that accompany peptic ulcer and may, by the contraction of the gastric and duodenal muscles and spasm of blood-vessels, cause erosions which in turn are subject to the action of the highly acid gastric juice.

Cushing states that intraventricular injections of pituitrin produce in man these symptoms of hypersecretion and hypermotility of the stomach. This is associated with patches of hyperemia of the gastric mucosa as shown by Beattie to follow direct electrical excitation of the tuber cinereum in animals. Pituitrin, the active principle of the neurohypophysis, may find its way through the infundibular stalk to the region of the tuber. It has been shown that this secretion also is under the control of autonomic fibres that go from the supra-optic nucleus into the posterior lobe. The posterior lobe extract of pituitrin seems, then, to have a stimulative effect on the local vegetative nerve centres when applied directly to them, even though intravenous or intramuscular injections of pituitrin produce no such effect.

It is probable, from the work of Pavlov and others, that the parasympathetic apparatus under normal conditions is strongly affected by cortical or psychic influences. Cushing does not claim that the theory of the neurogenic origin of peptic ulcer explains all peptic ulcers; but the probability of a centre in the tuber for parasympathetic impulses as well as the accepted sympathetic nucleus in the posterior hypothalamus, the proximity of the parasympathetic centre to the hypophysis, the control of the hypophyseal secretion by cortical impulses, the action of this hypophyseal secretion when applied directly to the vegetative nerve centres, seems to support this neurogenic theory of peptic ulcer in at least some cases.

(5) *The Vascular Theory.* The old view of Virchow that peptic ulcer is due primarily to embolism of the small vessels of the mucous

membrane is difficult to substantiate, for although ulcers may be reproduced artificially by the occlusion of the blood-vessels of the stomach, peptic ulceration is rarely, if ever, seen in cases where there are multiple emboli of such organs as the kidney and spleen, unless these emboli are associated with frank sepsis. Likewise, although thrombosis of the gastric blood-vessels may be followed by ulceration, its incidence does not appear to be greater in patients suffering from general arterial degeneration or peripheral thrombosis. We may infer, therefore, that both embolism and thrombosis of the blood-vessels of the stomach and duodenum are rarely the primary factor in the production of ulceration, unless there is a concomitant septic element.

(6) *The Gastritis Theory.* Chronic gastric ulcer is always associated with some degree of chronic gastritis, as has been confirmed during recent years by examination of large numbers of gastrectomy specimens. This has led Konjetzny (*Arch. f. klin. Chir.*, cxxix, 139, 1924, and with H. Puhl, *Med. Klin.*, xxiii, 986, 1925, and 1063, 1927) to express the view that the erosions which frequently accompany chronic gastritis develop into chronic ulcers owing to the lowered resistance of the mucous membrane of the stomach. He would therefore regard a gastric ulcer as an incidence in the course of an ulcerative gastritis. Hurst, however, takes the opposite view, that the chronic gastritis is secondary to the ulcerative condition.

(7) *The Site-determination of Ulcers.* Chronic gastric and duodenal ulcers occur in the "ulcer-bearing area," and are rarely found elsewhere. This area is confined to the lesser curvature of the stomach, to a variable strip, about one inch wide, on the anterior and posterior walls on either side of it, and to the duodenal bulb. There has been much speculation in seeking to account for the constant position of these ulcers, and the following theories as to the determination of the site of the ulceration deserve consideration:

(a) *The lymphoid follicle theory.* The lymphoid follicles are situated in the mucous membrane at the base of the glandular crypts, and are therefore superficial to the muscularis mucosæ. They are most abundant along the lesser curvature, the pyloric vestibule, the pyloric canal, and in the duodenal bulb. These lymphoid follicles, whose functions, as with lymphoid tissue in general, include resistance to bacterial invasion, may become infected by organisms and give rise to minute abscesses, which, on rupture, will result in an acute erosion.

It is conceivable that such an ulcer, when once formed, may develop into a chronic ulcer.

(b) *The traumatic theory.* The mucous membrane along the lesser curvature is more firmly adherent to the underlying coats of the stomach than elsewhere. It is therefore less able to adapt itself to the variations in the size and shape of the stomach, and as the main traffic route—*magenstrasse*—for food is along the lesser curvature it will be more liable to be subjected to trauma. Again, the anterior and posterior walls of the duodenal bulb—the “ulcer-bearing area” of the duodenum—receive the brunt of the viscid acid gastric chyme which is forcibly ejected through the pylorus.

(c) *The ischæmia theory.* It has been shown by Wilkie and Reeves that the anterior wall of the duodenum and the lesser curvature of the stomach have a relatively precarious blood supply. Mayo has demonstrated during operations that if the anterior wall of the duodenum is gently pulled downwards and held in this position for a comparatively short time, a white, anæmic area will appear on the anterior wall, about half an inch distal to the pylorus.

#### (8) *Accessory Factors.*

(a) *Chronic duodenal ileus.* Hurst and Wilkie have drawn attention to the frequent association of chronic duodenal ileus with chronic gastric and duodenal ulcers.

(b) *Tobacco.* In patients with the hypersthenic gastric diathesis over-smoking increases the liability to the formation of a duodenal ulcer. It has, however, but little bearing upon the production of gastric ulcer. Excessive absorption of nicotine, by augmenting the secretion of acid in the gastric juice, will necessarily hinder the process of healing and may, in addition, create or increase “hunger pain.”

(c) *Mental and physical fatigue.* Worry, mental fatigue, hard work and anxiety appear to be important factors in the production of duodenal ulcers, but it is doubtful whether they play any significant part in the production of gastric ulcers.

(d) *Food.* Unsuitable food, irregular or hurried meals, imperfect mastication, and a diet deficient in vitamins may predispose to peptic ulceration.

*We may sum up this difficult problem by stating that according to our present limited knowledge all available clinical and experimental*



evidence shows that two combined factors are simultaneously at work in the production of ulcers: (i) local trauma of the mucous membrane, whether mechanical, septic, or from infective emboli, bacterial or non-bacterial toxins, or neurotrophic disorders; and (ii) the eroding, digestive, and irritant powers of the acid gastric juice.

(C) *Pathology.*

(1) *Number.* In the stomach chronic peptic ulcers are almost invariably single, although the scars of healed ulcers or of acute ulcers are frequently seen in those portions of the stomach removed at operation by partial gastrectomy. It is rare to find two or more chronic ulcers in a state of activity at the same time (Hurst). In the duodenum, however, two chronic ulcers—one anterior and one posterior—co-exist in over 30 per cent of cases. In certain countries, such as Germany, multiple chronic peptic ulcers are more common than in England or America. Whereas in the latter countries the incidence is comparatively low, Graves (*Ann. Surg.*, 488, p. 197, Aug., 1933), as the result of intensive research into the statistics of central European hospitals, has shown that dual or multiple lesions are present in not less than 30 per cent of cases.

(2) *Associated Gastric and Duodenal Lesions.* The co-existence of gastric and duodenal ulcer has received emphasis from Wilkie (*B.M.J.*, p. 771, May 6, 1933), who writes: "I have operated upon 81 cases in which there were coincident ulcers in stomach and duodenum. These occurred in a total of 780 ulcer cases. In my series, out of a total of 695 patients with duodenal ulcer, 81, or 11.6 per cent, had a coincident gastric ulcer, and out of 166 patients with gastric ulcer, 81, or 49 per cent, had a coincident duodenal ulcer. The two lessons I draw from these observations are, that the aetiology of the two types of ulcers must be the same and that assessment of the total pathology should invariably be the first step of an abdominal operation."

Hurst and Stewart, however, find it difficult to reconcile their findings with those given by Wilkie. They found, in 4000 consecutive autopsies, 173 cases of chronic gastric and 230 cases of chronic duodenal ulceration or scarring. There were 21 (5.5 per cent) of this group in which lesions of both organs co-existed. "In other words, 12 per cent of chronic gastric and 9 per cent of chronic duodenal lesions were accompanied by chronic lesions of the other organ, but if open chronic ulcers only are taken into account—89 gastric and 153 duodenal, with

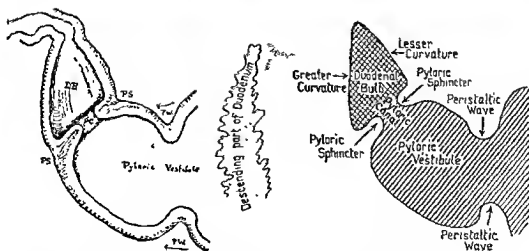


Fig. 59.—RADIOGRAM OF NORMAL PYLORIC VESTIBULE, PYLORIC CANAL, AND DUODENAL BULB. BELOW: LEFT, OUTLINE DRAWING OF RADIOGRAM; RIGHT, DIAGRAM OF SECTION THROUGH THE CORRESPONDING PARTS. DB, DUODENAL BULB; PS, PYLORIC SPHINCTER; PC, PYLORIC CANAL; PW, PERISTALTIC WAVE MOVING TOWARDS THE PYLORUS.

(Hurt and Stewart, "Gastric and Duodenal Ulcer." By kind permission of the Oxford Medical Publications.)

4 in which both organs were affected—the figures are only 4.5 and 2.6 per cent respectively.”

(3) *Position.* Ninety-six per cent of chronic gastric ulcers are situated between the cardia and the incisura, either on or within one inch of the lesser curvature. Ulcers of the pyloric segment are rare, not more than 4 per cent occurring in this situation. Ulcers on the greater curvature should always be considered as malignant. About 80 per cent of all chronic peptic ulcers occur in the first part of the duodenum. In females gastric ulcers tend, on the whole, to be situated somewhat nearer to the cardia than to the pylorus.

Chronic ulcers involve the posterior wall of the stomach much more frequently than the anterior, and the majority straddle the lesser curvature and involve both walls to a greater or less degree. Double or triple chronic gastric ulcers are nearly always close together. Chronic duodenal ulcer is almost invariably confined to the bulb, although I have operated upon two patients in which the lesion was situated on the posterior wall of the second part of the duodenum, just above the papilla. When single they are situated on the anterior or posterior wall; when multiple they often face one another. An anterior or a posterior ulcer may, by a process of extension, partly encircle the duodenal bulb. In cases of duodenal ulcer the anterior position is commoner than the posterior. The most frequent site for duodenal ulcers is three-quarters of an inch away from the pylorus (fig. 59), and they never extend through the pylorus into the pyloric canal, although where there has been much scarring and distortion it is sometimes difficult at operation to be sure whether the ulcer is situated in the duodenum or in the pylorus (fig. 60).

(4) *Macroscopic Characteristics.* A chronic gastric ulcer is larger than an acute or a sub-acute ulcer; it is also larger than a chronic duodenal ulcer. Stewart states that 93 per cent measure less than 2.5 cm. An anteriorly-placed duodenal ulcer is smaller than one situated on the posterior wall, where fixation to or penetration of the pancreas complicates and hinders the process of healing.

Anterior-wall ulcers show a greater tendency either to heal or to perforate.

Chronic gastric ulcers are round or oval in shape, or may be saddle-shaped when they lie astride the lesser curvature (figs. 61 and 62). Duodenal ulcers, on the other hand, are many-shaped. Although usually oval they may be crescentic, triangular, quadrilateral, or pear-shaped

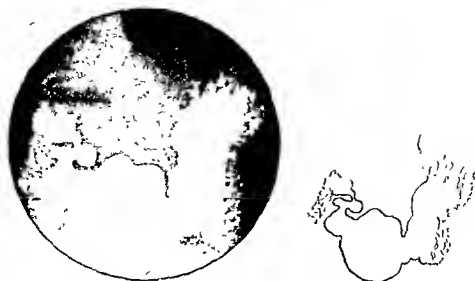


Fig. 60.—ULCER OF THE DUODENUM SHOWING DEFORMITY OF THE CAP.  
(H. Cecil Bull.)



Fig. 61.—CHRONIC GASTRIC ULCER ERODING THE PANCREAS.  
(Museum, St. Bartholomew's Hospital.)

(fig. 63). Irregularity in outline in the case of duodenal ulcers is of little importance, whereas in chronic gastric ulcers such irregularity would suggest the possibility of malignant degeneration in the margin.

Peptic ulcers present a punched-out appearance. They have overhanging or rounded margins which, during the active phases of inflammation, are red, œdematous, and eurved over into the crater. The walls are vertical, and their depth will depend upon the degree of penetration that has occurred (fig. 64). The base is formed of thickened

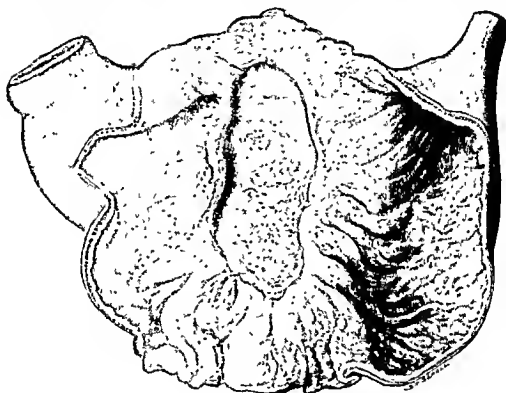


Fig. 63.—SADDLE-SHAPED GASTRIC ULCER.  
(Museum, Royal College of Surgeons.)

fibrotic subserosa and peritoneal coat, except in cases of deep penetration where the floor will consist of the viscus which the ulcer has eroded (fig. 65). The walls and the base of the ulcer are lined with granulation tissue, which in turn is coated with a yellowish muco- or fibrino-purulent exudate. Complete penetration, with consequent breaching of the muscular coats, is the most important characteristic of a chronic ulcer. Other evidences of chronicity are fibrosis and chronic inflammatory changes in the base and sides of the ulcer, adhesion of the muscularis mucosæ to the muscularis at the margin of the crater, adhesion to or penetration of neighbouring tissues, and the extent of deformity resulting from cicatrisation.

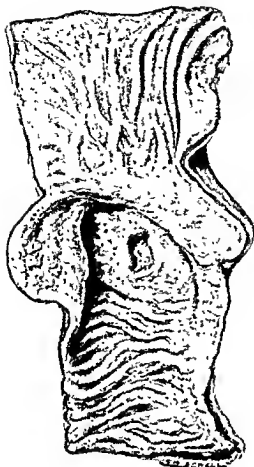


Fig. 63.—CHRONIC DUODENAL ULCER SITUATED ON THE POSTERIOR WALL, JUST DISTAL TO THE PYLORIC OUTLET.

(Museum, Royal College of Surgeons.)

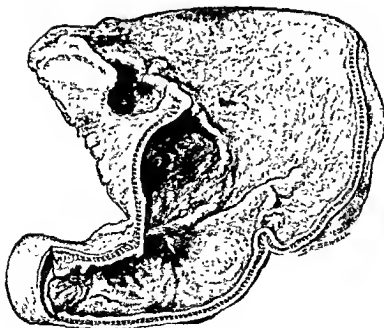


Fig. 64.—CHRONIC GASTRIC ULCER.

(Museum, Royal College of Surgeons.)

At operation a chronic peptic ulcer, whether situated in the stomach or the duodenum, is easily palpable as a localised, indurated mass, and by invagination of the walls of the gut the crater can often be felt. The tumour mass may resemble a carcinoma, and it is sometimes exceedingly difficult, in spite of the closest scrutiny during the course of an operation or after the ulcer has been removed, to decide by naked eye whether the ulcer is simple or malignant, whilst in certain instances,

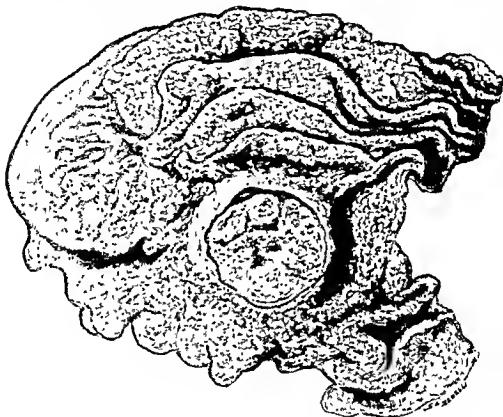


Fig 63. —CHRONIC GASTRIC ULCER DEEPLY EXCAVATING THE PANCREAS. PARTIAL GASTRECTOMY SPECIMEN.  
(Museum, Royal College of Surgeons.)

even with the aid of a microscope, doubt may still exist as to the true nature of the lesion.

*As stated before, a chronic ulcer situated in the stomach or duodenum is always palpable at operation, and for purposes of inquiry an incision through the gut to verify the presence of an ulcer should never be necessary.*

An acute ulcer cannot be felt or in any way identified at operation except in the rare cases of perforation. The serosal surface in the neighbourhood of a chronic ulcer is thickened, opaque, whitish, and leathery. Filmy bands and adhesions may be present. If at operation the surface is gently rubbed with a swab, numerous fine red points will appear. This stippling is due to the rupture of several thin-

walled vessels which are present in the adhesions. In ulcers that have healed, lines radiating towards a central point are produced by the contraction of the scar tissue. The surface will still be indurated, but there is no evidence now of congestion or stippling. The adhesions have become devascularised, but the cicatrisation and deformity persist. This stippled appearance over an ulcer, produced by friction, is important from a diagnostic point of view, as in cases of cancer it is absent. In cancer the outer surface of the gut may be pale, or be replaced by a white, disc-like plaque. Little peripheral nodules may be made out, as well as stiffened strands of lymphatics permeated by growth. Frequently the outlying nodules will indicate the margins or the extent to which the underlying growth has spread. The demarcation to the naked eye between growth and normal stomach wall may be clearly defined, whereas in simple ulcer the thickened and fibrotic margins gradually fade away into the neighbouring stomach wall. The adjacent lymph glands in cases of ulcer will be found to be enlarged and elastic. On section they are greyish or pink in colour. A lymph gland containing secondaries will be hard and irregular if extensively involved. On section it will show white circumscribed neoplastic areas which are clearly visible to the naked eye.

(5) *Microscopic Characteristics.* On microscopical examination the appearance of an ulcer will vary according to whether it is active or healing. In the active stage there will be a considerable amount of œdema, infiltration of the base and margin with leucocytes, and evidence of an increased proliferation of fibroblasts. The margins of the ulcer will be seen curling over towards the base, the muscular coats are entirely breached, and the granulation tissue lining the walls of the crater shows engorgement with blood. When healing is in progress, owing to the contraction which takes place in the scar tissue the size of the ulcer is diminished, and there is up-turning of the breached muscle ends. The purulent exudate which covers the floor of the ulcer is absorbed, the granulation tissue assumes a fresh, healthy, and active appearance, and there is subsidence in the venous and lymphatic engorgement around the margins, leading to a flattening out of the mucous membrane, which creeps across the now rejuvenated ulcer bed. Healing is always precarious, as the floor of the ulcer is covered in the first instance by a single layer of epithelium. When conditions are favourable, downgrowths of this layer of epithelium, and other changes, occur, until an inferior mucous membrane, somewhat approximating the original but thinner and less convoluted, is reproduced.



It is not surprising that, on account of the fixation of this new mucous membrane to its sclerotic base with its poor blood supply—the result of endarteritis—and its loss of flexibility, recrudescence of ulceration at the same site is not infrequent.

There are probably many chronic ulcers which heal completely and break down again many times during their life history. The final result of the cicatrization of a chronic ulcer depends upon the situation, the chronicity, and the size of the ulcer. For instance, a chronic peptic oesophageal ulcer, on healing, may produce oesophageal obstruction. A saddle-shaped ulcer on the lesser curvature of the stomach may give rise to hour-glass stomach. A large oval ulcer, or multiple ulcers on the lesser curvature, will lead to shortening and contraction of the lesser curvature itself—"purse-bag" stomach. A chronic ulcer situated in the pyloric canal or in the duodenum produces pyloric stenosis. The commonest cause of pyloric stenosis is a cicatrizing duodenal ulcer.

#### (D) *Diagnosis.*

(1) *Symptoms.* The majority of acute ulcers are unrecognised during life, as they give rise to no symptoms unless hæmorrhage or perforation occurs. Chronic indigestion, abdominal discomfort, nausea, and other symptoms may be associated with an acute ulcer, but in these cases the dyspeptic symptoms are secondary to lesions of the abdominal viscera, and especially of the gall-bladder and appendix.

In cases of acute ulcer nothing abnormal is found on abdominal examination. There is no muscular guarding, no tenderness, no distension; in fact, no physical signs of any importance, except perhaps those caused by an associated condition such as appendicitis, which might be held to account for the symptoms of indigestion.

During the active phases of acute ulceration occult blood tests will be positive, but an X-ray examination of the stomach after the administration of a barium meal will yield negative results.

The most important symptoms of a chronic gastric ulcer are pain, vomiting and hæmatemesis, and by far the most outstanding single symptom is pain. The pain is usually very severe, and arises shortly after the intake of food. The nearer the ulcer is to the cardia the earlier will be the onset of the pain, although this is not invariably the case. It is usually felt about one to one and a half hours after meals, and the more indigestible the food the more severe the resulting pain. Patients with gastric ulcers rarely, if ever, experience pain during the

night, and seldom on an empty stomach in the morning before breakfast. The attacks of pain last for a variable time—an hour or two, sometimes more, sometimes less. They may be severe and cramp-like, or dull, aching, gnawing, and piercing through to the back. The pain is partially relieved by taking alkalis or milk, but instantaneous and complete relief is afforded by vomiting, which is commoner in gastric than in duodenal ulcer. This vomiting rarely occurs when pain is absent, and seldom unless it is very acute. On discovering that vomiting relieves the pain, patients will often resort to self-induced vomiting. The periods of freedom from pain are usually short-lived.

During an attack the pain will follow every meal, and may be clock-like in its regularity, and undiminished in its severity unless rest in bed, diet, and medicines are prescribed. In a typical case of gastric ulcer the patient will give a history of these attacks of dyspeptic pain, each lasting a few days or weeks, with intervals of complete freedom for weeks or months. It is important to inquire carefully into this question of periodicity, as it is very characteristic of the early stage of chronic peptic ulcer. The periodicity of the attacks is more constant and more clearly defined in duodenal than in gastric ulcer. The complete disappearance of pain after severe hæmatemesis or melaena is an interesting feature in cases of chronic peptic ulceration.

The pain is felt in the epigastric region, is usually centrally placed, but will occasionally be to the right or to the left of the mid-line, or even higher up in the region of the xiphisternum. Unlike the diffuse epigastric pain in cases of chronic gastritis, growth, and nervous indigestion, the pain is often localised to a small area. The cause of the pain in chronic ulcer of the stomach and duodenum is difficult to explain, but it has been thought to be muscular in origin, and to be produced by tension or stretching of the muscular fibres and nerves in the vicinity of the ulcer, or by involvement of the parietal peritoneum. Most gastric symptoms are due to motor disturbances. When chronicity is established the attacks will increase in frequency and will last longer, the intervals will shorten, and the symptoms during the attacks will become more acute.

Appetite and nutrition are usually good in cases of gastric ulcer, except where the disease is advanced, when, owing to fear of the pain that will follow the intake of food, there is an enforced abstinence, resulting in loss of weight and malnutrition. With the onset of pyloric stenosis or hour-glass stomach, loss of weight, increased vomiting, and anorexia will result.

Chronic peptic ulceration is frequently associated with pylorospasm, which by interfering with the emptying of the stomach will produce gastric distension, belching, regurgitation, and heart-burn. Whereas severe hæmatemesis may occur in cancer of the stomach, cirrhosis of the liver, splenic anæmia, and in certain blood dyscrasias, and is present in approximately 20 per cent of all ulcer cases, chronic ulcer is by far the commonest single cause. It is more often associated with gastric than with duodenal ulcer, although when occurring with duodenal ulcer it is more lethal. Melæna, on the other hand, is more frequently seen in the latter condition. The vomited blood is partially clotted, acid in reaction, and may be mixed with food, whilst if it has been in the stomach for some time, it may be dark brown or black, and may resemble coffee grounds.

Sudden hæmatemesis may be the first and only symptom of a peptic ulcer, although in a number of cases it is preceded by an exacerbation of symptoms, and may follow indiscretions in diet or undue physical exertion. In such cases melæna will occur some hours later, dark, tarry motions being passed.

In *duodenal ulcer* a correct diagnosis can be made on the symptoms alone in about 90 per cent of all cases; in gastric ulcer in about 50 per cent. The pain in duodenal ulcer comes on at a longer interval after the intake of food. *The bigger the meal the greater the interval, but the worse the pain.* The average interval is about three hours. After breakfast (a small meal) the interval will be less, about two hours; after dinner (the largest meal of the day) it may be as long as three and a half to six hours. Certain articles of diet or the intake of alcohol will aggravate the pain.

*Hunger pain* is a sensation of combined pain and craving for food, which is appeased by food, alkalis, or vomiting. It is often relieved by rest in bed and warmth. It may waken the patient out of his sleep, usually early in the morning about 2 a.m., and after a little while—one to two hours—slowly subside and disappear. Patients with chronic duodenal ulcer who suffer from this nocturnal pain will often be found to adopt the habit of placing biscuits or a glass of milk beside their bed on retiring for the night, realising that swift relief is afforded by taking a small amount of such nourishment. The patient will then be comfortable for the remainder of the night, and will awaken in the morning free from pain, and remain so until some two hours after breakfast when the symptoms will return. Pain which is severe enough to arouse a patient from sleep is likely to be organic in nature. On the other hand, pain which increases in severity when

the patient is in bed and prevents him from sleeping is suggestive rather of functional disorder.

The attacks seem to have a seasonal occurrence and to be more common in the autumn and spring. Each lasts for a few days or weeks, and is then followed by a period of freedom from all symptoms and apparent good health.

In an advanced case pain is rarely absent, even between the more acute attacks. It may be very severe, burning and exhausting in character. As stated before, over-smoking or the excessive consumption of alcohol may precipitate an attack, whilst worry or anxiety may intensify it.

It should be noted that hunger pain occurs in many other conditions besides peptic ulcer, such as chronic appendicitis, cancer of the stomach, chronic diseases of the gall-bladder, hyperchlorhydria, excessive nicotine absorption, and in certain nervous disorders. Vomiting is very rare in uncomplicated duodenal ulcer. When pyloric obstruction occurs, vomiting is a prominent and characteristic feature.

Water-brash is especially frequent in cases of chronic duodenal ulcer, as has been observed by Ryle. He considers that the abundant production of this watery alkaline saliva is a natural reaction on the part of the body to neutralise the excess of acid in the stomach. In simple cases the appetite is good, and patients will often gain weight and appear fit and well. With the onset of stasis a loss of weight will ensue as the result of vomiting and a distaste for food. As in cases of gastric ulcer, constipation is present and will be aggravated by the onset of pyloric stenosis. Melæna is common. In a severe attack *large quantities of blood rapidly pass into the stomach and small intestine, accompanied by fainting attacks and increasing pallor, whilst tarry stools will proclaim the source from which the blood originates.*

(2) *Physical Signs.* In suspected cases of ulcer a careful physical examination of the abdomen should be performed as a routine, not only to verify as far as possible the presence of an ulcer, but also to detect any concomitant diseases which may be present in other organs. It is often taught that a physical examination will be negative in the majority of ulcer cases, and consequently this method of investigation is likely to be perfunctorily or carelessly conducted. It is true that *tenderness and rigidity* may be absent during the quiescent period, but during an attack these signs are generally present. It is well, therefore, to examine these patients at various times, preferably both during an attack of pain and when pain is absent. By this method useful

information will be obtained as to the site of tenderness and as to the extent and degree of muscular guarding.

In cases of *gastric ulcer* tenderness will often be found in the mid-epigastric region or at a variable distance to the left. It may be higher up, below the left costal cartilages, or, where the ulcer is in the pyloric portion, the site of tenderness may be located to the right of the mid-line in the region of the duodenum. Marked muscular rigidity would suggest the possibility of a grave complication such as sub-acute perforation. Deep palpation should be performed when testing for visceral tenderness, and if a localised tender spot is discovered it is advantageous to watch the repetition of this examination under an X-ray screen after the administration of a barium meal, in order to ascertain whether the tender area corresponds with the crater of a chronic gastric ulcer.

In *duodenal ulcer* the site of tenderness is usually situated in the epigastrium on the outer border of the right rectus muscle. It is fairly constant in position and does not shift perceptibly on respiration. It may be confused with gall-bladder tenderness, but the latter is placed more laterally and is intensified by palpation during deep respiratory movements.

It should be remembered that the earliest sign of response in an ulcer to medical treatment is the disappearance of pain, which is followed successively by a loss of tenderness and of muscular guarding.

Occasionally a gastric or duodenal ulcer may form a palpable tumour. It is very rare, however, to feel such a tumour unless the patient is very thin and has a flaccid abdominal wall. Its palpation is also more likely when the stomach is empty. It is often impossible to determine whether the tumour which is felt is a growth or an inflammatory mass. On palpation it is tender, and if fixed to the pancreas will not move on respiration. A cancer of the stomach is less tender than a peptic ulcer "tumour."

When *dilatation of the stomach* occurs as the result of stenosis there is distension of the epigastrium, visible peristalsis, and splashing noises. By ordinary physical methods of examination it is difficult to discriminate between atonic dilatation of the stomach and stasis produced by organic stenosis, and the diagnosis can only be clinched by X-ray investigation.

In the general examination of the patient particular attention should be paid to any evidence of septic teeth, gums, or tonsils. Pallor and anaemia are obvious and may suggest haemorrhage, growth, or some blood disorder. A rectal examination should never be omitted.



Fig. 66.—CHRONIC GASTRIC ULCER. LARGE GASTRIC ULCER ON LESSER CURVATURE OF THE STOMACH. THERE IS CONSIDERABLE SPASM OF THE GREATER CURVATURE. OPERATION—PARTIAL GASTRECTOMY. BENIGN ULCER. (Author's case.)



Fig. 67.—ULCER OF THE DUODENUM. THE DUODENAL CAP IS TOTALLY DEFORMED. (H. Cecil Bull.)

(3) *Investigations.* The following additional investigations will be necessary to arrive at an accurate diagnosis :

- (a) X-ray examination.
- (b) Gastric analysis.
- (c) Occult blood examination.
- (d) Wassermann reaction.
- (e) Complete blood examination.

Of all investigations a well-taken history is probably the most important, but X-ray examination is indispensable, and no method of inquiry is complete and no diagnosis final until this has been undertaken. X-rays are now outstanding in their reliability as a method in the diagnosis of chronic peptic ulcers, but a diagnosis should not be based *solely* upon the report of the radiologist. Although cases have at times been treated as chronic gastric or duodenal ulcer apart from X-ray confirmation, it is now generally accepted that the diagnosis must be confirmed by definite and irrefutable X-ray evidence before any systematic treatment is instituted. Such an examination should in all cases be undertaken only by a *skilled radiologist*.

It is very rare for a chronic gastric or duodenal ulcer to be overlooked when the examination is conducted by a competent radiologist, who can in over 95 per cent of suspected cases with accuracy demonstrate the presence of an ulcer (figs. 66 and 67).

#### (E) *Differential Diagnosis.*

Chronic gastric and duodenal ulcer must be distinguished from :

##### (1) *Nervous gastric disorders.*

- (a) Functional.
- (b) Gastric crises of tabes.
- (c) Migraine.

##### (2) *Reflex dyspepsias.*

- (a) Appendix dyspepsia.
- (b) Gall-bladder dyspepsia.
- (c) Carcinoma of the colon, and other intestinal conditions.
- (d) Epigastric hernia.
- (e) Visceroptosis.
- (f) Pancreatic disease.
- (g) Ileo-cæcal tuberculosis.

(3) *Other organic gastric and duodenal disorders.*

- (a) Gastritis and duodenitis.
- (b) Syphilis of the stomach.
- (c) Cancer of the stomach.
- (d) Tuberculous ulceration of the stomach.
- (e) Duodenal diverticula.
- (f) Duodenal ileus.
- (g) Duodenal bands.
- (h) Innocent new growths of the stomach.

*Every patient with chronic indigestion has a potential gastric or duodenal ulcer until it is proved otherwise.* It is surprising the number of diseases, functional or organic, that will closely mimic chronic peptic ulceration, as a consideration of the following will clearly show.

(1) *Nervous Gastric Disorders*

(a) *Functional conditions.* There are many functional disorders, such as nervous dyspepsia and neurasthenia, in which there are symptoms of chronic indigestion; but a diagnosis of "functional dyspepsia" should not be made until a complete investigation has ruled out the possibility of organic disease. If this were done as a routine many definite cases of gastric or duodenal lesion would be detected at an early stage, permitting of radical operation in cases of cancer, and early treatment in cases of peptic ulcer.

In cases of "functional dyspepsia" there is a multiplicity of vague symptoms which will be found to vary each time the history of the case is taken. On close interrogation it is frequently found that abdominal discomfort of one form or another, rather than true pain, is the main complaint. The attacks appear to arise without any regular periodicity, and the dyspeptic symptoms are not relieved by rest or by the administration of alkalis. The "pain" is increased when the patient is confined to bed, as opportunity is then afforded for him to dwell upon and magnify his troubles.

On physical examination there is, as a rule, no localised area of tenderness. When tenderness is present it is often diffuse, inconsistent, vague, and readily subject to suggestion. Occult blood tests and a barium meal examination will prove the absence of a chronic peptic ulcer.



(b) *Gastric crises.* The symptoms in the gastric crisis of *tabes* may closely resemble those of chronic gastric or duodenal ulcer; when severe they may be mistaken for those of a perforated ulcer, acute obstructive cholecystitis, or acute appendicitis, even leading the unwary surgeon to undertake an unnecessary operation, many such instances being on record. The possibility of chronic peptic ulcer being associated with *tabes* should, however, be borne in mind. It should be remembered that visceral analgesia may result from *tabes*, and that an acute abdominal catastrophe may occur without any pain, tenderness, or rigidity. Although tabetic crises are a late syphilitic manifestation heralding the onset of ataxia, the evidences of organic nervous disease may be singularly slight, even on the most careful physical examination. In a typical case of gastric crises the symptoms are ushered in with abruptness. There is epigastric pain, and lightning attacks of girdle-like pain radiating round the chest and abdomen, producing a sensation of constriction or strangling which lasts for a variable time, and which may vanish with equally dramatic suddenness, only to recur again without premonitory symptoms. Vomiting is a distressing symptom, and is more severe and recalcitrant than in cases of chronic ulcer. It may be prolonged and exhausting, but the amount vomited is usually small, consisting of frothy gastric contents, stained with bile. The vomiting does not assuage the pain as it does in cases of gastric ulcer; in fact, nothing short of a full dose of morphia will bring relief.

In a suspected case of *tabes* an examination of the ankle jerks should always be undertaken, as these will often be found to disappear sooner than the knee jerks. A diagnosis of gastric crises will here be rendered certain if occult blood tests are negative, if radiology definitely excludes the presence of ulcer, and if signs and symptoms of *tabes dorsalis* are present with confirmatory serological tests.

(c) *Migraine.* In cases of migraine the initial symptom is severe headache accompanied by ocular disturbances. This is followed by a sensation of nausea, and frequently bilious vomiting. Abdominal pain, however, is absent. It should be noted that bouts of vomiting, when unassociated with abdominal pain, are seldom connected with gastric diseases. The symptoms—headache, followed by nausea and vomiting, in the absence of abdominal pain—will suggest a migrainous attack, and there will usually be a history of similar short attacks in the past, quickly followed by complete relief and absence of all abdominal symptoms.

## (2) *Reflex Dyspepsias*

(a) *Appendix dyspepsia.* Chronic appendicitis may be associated with pain in the right iliac fossa or in the epigastric region, or both. The term appendix dyspepsia is only applicable to those cases in which chronic appendicitis is accompanied by epigastric pain. Appendix dyspepsia should not be diagnosed as such until a thorough investigation definitely incriminates the appendix.

When a careful history is taken in these cases, the resemblance of the symptoms to those of chronic peptic ulcer is found to be very superficial. The dyspepsia is more or less continuous, and though there is no typical periodicity, there are irregular short attacks of sharper pain. In the intervals between these attacks the patient is never quite free from symptoms. Nausea, which is rare in chronic gastric and duodenal ulcer, is fairly common in appendix dyspepsia. Vomiting in chronic gastric ulcer gives instantaneous and complete relief from pain, whereas in appendix dyspepsia the relief is incomplete. Pain, when present, is made worse by exertion, constipation, diarrhoea, or the administration of purgatives. It appears to be unrelated to the ingestion of food. It may arise at any time before or after a meal. Neither diet nor alkalis will relieve it completely, although some mitigation of the symptoms may result from such measures. As in duodenal ulcer, there may be nocturnal pain. The pain is mid-epigastric in position, but often radiates to the right iliac fossa, and may, at times, remain localised to the lower abdomen for a considerable period without producing any epigastric disturbance.

When the pain in the epigastrium is at its height, pressure over the appendix will be resented. Similarly, pressure on the epigastrium will sometimes produce pain which is referred to the right iliac fossa. If a history of a previous attack of acute appendicitis is given, i.e. pain in the right iliac fossa lasting for some hours or days, and associated with pyrexia and other symptoms, very valuable information will be obtained as to the seat of the trouble; but unfortunately such a history is rarely elicited.

Even without the aid of radiology, a provisional diagnosis can be made with more confidence in young patients than in those over the age of 30. The best method of differentiating between appendix dyspepsia and chronic peptic ulcer is by means of X-ray examination, especially when no ulcer crater is demonstrated on screening.

(b) *Gall-bladder dyspepsia.* Chronic diseases of the gall-bladder often produce reflex gastric symptoms. A typical case of cholecystitis

should cause no difficulty in diagnosis, nor should the symptoms be confused with those of chronic peptic ulceration. In some cases, however, there are definite attacks of pain which come on at intervals, these attacks being associated with hunger pain which is relieved by food. The attacks in cholecystitis tend to be irregular, and during the intervals there is always a certain amount of discomfort. Nausea and flatulence may be marked symptoms.

The epigastric upset and pain in diseases of the gall-bladder are, on the whole, less regular in their onset, and less influenced by rest in bed, diet, and the administration of alkalis than is the case with ulcer. The appetite may be good, but there is distaste for certain articles of food such as fats and oils. Chronic cholecystitis is the commonest chronic intra-abdominal lesion, and is more frequent in women than men. Duodenal ulcer, on the other hand, preponderates in men by about eight to one. The site of gall-bladder tenderness is placed further out than in the case of duodenal ulcer, and may be present in the intervals when the patient is free from pain. Murphy's sign is often positive.

Although duodenal intubation may give useful information in distinguishing between chronic diseases of the gall-bladder and duodenal ulcer, the chief reliance will have to be placed upon cholecystography by Graham's method and a barium meal examination, and particularly so when these two tests are carried out simultaneously. The two diseases may co-exist, but only X-rays will afford proof of the presence of a double lesion. Not infrequently the abdominal triad of Wilkie—chronic cholecystitis, chronic duodenal ulcer, and chronic appendicitis—is present.

(c) *Carcinoma of the colon and other intestinal diseases.* These may produce reflex gastric symptoms. In carcinoma of the colon, and particularly where there is no obstruction of the gut, the early symptoms are mostly referable to the stomach. There is nausea, flatulence, epigastric discomfort, distaste for food, and possibly vomiting. There may be constipation or diarrhoea, or diarrhoea alternating with constipation. Mucus and blood may be found in the motions, but the blood in cases of cancer of the large gut is rarely tarry. It is usually brown or red, and is intimately mixed with the motions. In addition to the epigastric tenderness there will be tenderness over the whole course of the colon. A palpable tumour may be detected in the line of the colon, away from the stomach region. In suspected cases an X-ray examination after the administration of a barium enema

will indicate the presence or absence of an organic stricture or filling defect of the colon.

Other intestinal conditions, such as chronic colitis, diverticulitis, etc., may produce pylorospasm and reflex gastric symptoms. They are, however, easily differentiated from chronic ulcer by their other characteristics.

(d) *Epigastric hernia.* Epigastric hernia is a protrusion of fat (pro-peritoneal lipoma) through an aperture in the supra-umbilical portion of the linea alba. These herniæ vary considerably in size. They may be as small as a pea or as large as an orange. The larger varieties situated near the umbilicus are termed *para-umbilical herniæ*. Occasionally the fatty tumour may form a small hernial sac by dragging a funnel-shaped process of peritoneum with it through the linea alba. These tumours arise from the fat in the falciform ligament, and carry small blood-vessels with them in their extra-abdominal excursion. The symptoms they produce are caused by the dragging on the fat and peritoneum of the falciform ligament. This irritation of the parietal peritoneum causes a reflex pylorospasm which Ryle considers to be akin to the pylorospasm encountered in some cases of appendicular and gall-bladder diseases. The symptoms complained of—eructations, nausea, vague abdominal discomfort, epigastric pain which may be aggravated or relieved by changes of posture, and vomiting which may be the only symptom, are dependent upon this pylorospasm.

The severity of the symptoms is not wholly dependent upon the size of the hernia; it is more the outcome of the peritoneal drag or of any concomitant complications.

*Quite small uncomplicated epigastric herniæ may produce very severe symptoms and yet pass undetected for many years, particularly in obese patients.*

Such complications as inflammation or gangrene may arise owing to the twisting of the tumour pedicle. Under such circumstances there will be acute localised epigastric pain, marked superficial tenderness, and muscular rigidity, suggesting the possibility of an upper abdominal inflammatory lesion, or even a sub-acute perforation. It is possible, too, for a Richter's hernia to occur in the sac of an epigastric hernia, causing very severe symptoms of acute intestinal obstruction, which may even resemble those of acute perforation, acute obstructive cholecystitis, or acute pancreatitis. The protrusion is easier to detect when the patient is examined standing than when he is lying down. It is often irreducible and very tender. This tenderness is the most

important diagnostic feature. If a patient has an epigastric hernia with symptoms, and X-rays exclude the presence of ulcer, the tumour should be dissected out and the breach in the linea alba repaired.

(c) *Visceroptosis*. In this condition there is ptosis of the abdominal viscera. Two types are described—*virginal* and *maternal*.

In *virginal* ptosis displacement of the viscera is due to congenital causes. On examination the build of the patient may stamp her as of this type. The patients are usually young, thin, asthenic females, with drooping shoulders, flat chests, and narrow waists. The abdomen is elongated, compressed in the narrow epigastric region and protuberant in the hypogastrum.

Any condition which leads to weakness or loss of tone in the abdominal wall or mechanical support of the organs, such as (i) rapid loss of weight due to debilitating disease, (ii) repeated pregnancies, or (iii) excessive dragging on or stretching of the peritoneal processes and suspensory ligaments, will be a predisposing factor to *maternal* visceroptosis. A constantly overlaid stomach, and a weighty, dilated colon are aggravating causes.

There are a number of individuals who have advanced visceroptosis without any symptoms, and it is not the extent of the visceral displacement which influences the severity of the symptoms. Gastropnoia and coloptosis rarely, if ever, exist as clinical entities. They are usually associated with ptosis of other viscera, and this should be remembered when treatment is undertaken for these conditions.

Visceroptosis may give rise to a large number of symptoms which, in certain cases, may suggest a diagnosis of chronic gastric or duodenal ulcer, chronic appendicitis, tubo-ovarian disease, chronic disease of the gall-bladder, or chronic pancreatitis. The symptoms of *visceroptosis*, although in some cases very closely resembling those of chronic peptic ulcer, are less clearly defined. They are variable and multitudinous. In a well-established case the patient will complain of abdominal pain which is made worse by the intake of food. The pain arises during meals and increases as more food is taken. This is accompanied by a sensation of fulness, distension, and a feeling of weight and dragging in the epigastrum.

This epigastric discomfort persists for some hours, and is often accompanied by gastric flatulence, regurgitation, and belching. Vomiting will partially relieve the pain, as will also dieting or resting in the recumbent position. Alkalis will have little or no effect in the amelioration of the symptoms. Constipation may be marked, mucous

colitis may develop, and there may be various concomitant nervous symptoms.

The complexion is usually sallow, the conjunctivæ pale, whilst the skin may be lax, thrown into folds, and inelastic. The epigastrium is flat or sunken, and there may be divarication of the recti. The lower half of the abdomen is usually protuberant or pendulous but may be scaphoid. The abdominal wall is weak and yielding, and splashing, gurgling noises can be heard on palpation, whilst various abdominal viscera can easily be felt to move on manipulation or on change of the patient's position.

As the stomach is long, atonic and ptosed, it drops through the pelvic inlet and drags the duodenum downwards. With the stomach in this position, owing to the absence of the "gastric cushion," the pulsations of the aorta are easily felt and may be clearly visible.

Every case of visceroptosis should be investigated by means of X-rays, in view of the possibility of associated visceral lesions.

(f) *Pancreatic disease.* Diseases of the pancreas are associated with symptoms of dyspepsia, but their resemblance to chronic peptic ulcer is very slight, and the differentiation between them should, with careful inquiry, present no difficulty. Acute hæmorrhagic pancreatitis may be mistaken for a perforated peptic ulcer; sub-acute inflammatory lesions of the pancreas may mimic a large callous penetrating gastric ulcer in which pain and vomiting are outstanding features.

(g) *Ileo-cæcal tuberculosis.* Ileo-cæcal tuberculosis may produce reflex dyspeptic symptoms, such as epigastric pain and vomiting, or may imitate the symptom-complex of a duodenal ulcer.

On physical examination, however, there will be evidence of disease in the region of the right iliac fossa, where a hard palpable tumour or thickening of the gut may be felt, which, combined with other symptoms, calls for radiological inquiry. The clinical distinction from growth is sometimes difficult.

### (3) *Organic Gastric and Duodenal Disorders*

(a) *Gastritis and duodenitis.* Chronic gastritis and chronic duodenitis cannot be diagnosed on signs and symptoms alone, as there are no characteristic features which denote the presence of such lesions. It is only after gastric analysis, occult blood tests, and radiological examinations have been carried out that a diagnosis can be formed and

differentiation made between these conditions and chronic peptic ulcer.

The symptoms of duodenitis may at times be indistinguishable from those of duodenal ulcer. There are intermittent attacks of dyspepsia, hunger pain, and the relief afforded by diet and medical treatment. In some cases the simulation may be so close that even occult blood will be found in the stools, and on radiological examination there will be a spasm or deformity of the duodenal bulb which may lead to precipitate operative measures. Konjetzny, Nagel, and others have described cases of duodenitis submitted to operation where excision of the duodenal bulb showed no evidence of chronic ulceration, but only an inflamed mucous membrane.

Achlorhydria is a common feature, as duodenitis is always associated with some degree of gastritis. In the differential diagnosis of chronic peptic ulceration, therefore, such conditions as chronic gastritis and chronic duodenitis must receive due consideration.

(b) *Syphilis of the stomach.* Syphilis of the stomach is a very rare condition, and probably does not occur in more than three out of every 1000 cases of organic disease of the stomach, although Continental and American writers place the incidence higher than this.

Syphilis of the stomach is usually a late tertiary manifestation of the acquired variety, but may sometimes occur in the congenital disease.

The lesion commences as a gummatus infiltration of the submucosa. Many small gummata form, coalesce, break down, and discharge into the cavity of the stomach, so that eventually a ragged, ulcerated area is produced. Occasionally the infiltration of the submucosa is diffuse, when the condition will resemble leather-bottle stomach. In these cases the stomach is small, shrivelled, hard and leathery, with a thickened peritoneal coat. On section the thickened, fibrotic submucosal layer is strikingly evident, and the mucous membrane, undergoing the changes characteristic of chronic gastritis, becomes fixed to the underlying layers.

On abdominal examination a palpable tumour may be felt as a result of the thickening. In cases of gastric syphilis the ulceration, which may be very extensive, shows a predilection for the lesser curvature and the pyloric portion. There will be occult blood in the stools, in the vomit, and in samples taken for gastric analysis. In consequence of the associated gastritis, achlorhydria is often present.

Gastric syphilis may give rise to all the complications associated

with chronic peptic ulcer. There may be a severe hæmatemesis from erosion of a blood-vessel, perforation may occur with fatal peritonitis, and cicatricial deformities are by no means infrequent. The symptoms in gastric syphilis may superficially resemble those of chronic peptic ulcer, whilst the signs may be those of cancer of the stomach. A number of cases are diagnosed as cancer of the stomach and are accordingly submitted to operation. Although there may be very severe symptoms, extending over long periods and associated with sharp attacks of vomiting, hunger pain, and diffuse epigastric distress, and although physical signs of gross organic disease of the stomach may be prominent, the general condition of such patients is, on the whole, very good. The appetite remains excellent, and there is no anæmia and no appreciable loss of weight. The pain appears, as a rule, to be unrelated to the intake of food. The X-rays may show a localised or a generalised involvement of the stomach. No radiologist, however competent, can be sure of a correct diagnosis of gastric syphilis on X-ray examination alone; in fact, he would still be uncertain of his diagnosis if he had the resected portion of the stomach in his hand (Alvarez). A diagnosis of gastric syphilis can be made with confidence in those cases where a history of syphilis is given, where the Wassermann reaction is positive, where the radiologist demonstrates deformities of the stomach, and where healing of the gastric lesion, accompanied by marked improvement in, or the disappearance of, all symptoms, seems to be directly connected with intensive anti-syphilitic treatment. Eusterman (*Coll. Papers Mayo Clinic*, xiii, 62, 1921) attaches great importance to the *therapeutic test*, and affirms that a rapid cure of the condition under anti-syphilitic treatment is as reliable a test as any on which to base a diagnosis of syphilis of the stomach.

(c) *Cancer of the stomach.* The symptoms of cancer of the stomach may mimic those of chronic gastric or duodenal ulcer. This more particularly applies to those cases in which hunger pain is a prominent feature.

(d) *Tuberculous ulceration of the stomach.* Tuberculous ulceration of the stomach is a rare disease in which a diagnosis by means of clinical and accessory methods of investigation is almost impossible. The disease is rarely recognised during life, except in the occasional case of perforation, or where gastric resection has been undertaken for a suspicious lesion of the stomach which proves on microscopical examination to be tuberculous in origin.





FIG. 68.—PSEUDO-DIVERTICULUM OF THE DUODENUM. ULCER OF THE DUODENUM WITH GROSS DEFORMITY OF THE CAP AND PSEUDO-DIVERTICULUM. SUCH DIVERTICULA CONTAIN MUCOUS TISSUE IN THEIR WALLS AND ARE NOT TRUE DIVERTICULA, BUT THE RESULT OF FIBROUS CONTRACTION. (*H. Cecil Bull.*)

The infection may take place from the interior of the stomach or via the blood stream or lymphatics. The following varieties of tuberculosis of the stomach, usually seen in combination, are recognised :

- (i) Miliary tuberculosis.
- (ii) Tuberculous ulceration.
- (iii) Hyperplastic tuberculosis.
- (iv) Tuberculous pyloric stenosis.

Tuberculous ulceration and hyperplastic tuberculosis of the stomach may simulate cancer rather than chronic peptic ulcer. The differentiation between a tuberculous pyloric stenosis and one due to benign stricture may be impossible, even where the presence of phthisis would suggest the former condition.

(c) *Duodenal diverticula.* Duodenal diverticula consist of small pouchings or blind sacs with narrow apertures opening into the duodenum. They vary in size from that of a pea to that of a pear. Although usually single they may be multiple. They are often seen in association with visceroptosis or with chronic duodenal ileus. There are two types—true and false—and these may be either congenital or acquired. In the *true* variety, which is usually congenital, the sac is generally

composed of all the coats of the gut. Pseudo-diverticula, which occur in connection with cicatrization of the duodenal bulb resulting from an old-standing duodenal ulcer, from duodenal bands and adhesions, or from chronic peri-cholecystitis, must be distinguished from the type under discussion. They are rather traction diverticula or sacculations, dependent upon inflammatory organic changes which have occurred in or outside the duodenal wall in the region of the bulb (see fig. 86).

The true congenital type may, on the other hand, be found connected to the posterior surface or to the greater curvature of the duodenal bulb without any associated organic lesion in this portion of the gut (fig. 69). It has been estimated by Kellogg that about 2 per cent of all diverticula of the gastro-intestinal tract occur in the duodenum, and that autopsy records indicate that the condition occurs more frequently in males than in females (62 per cent in 61 cases), although Case in a total of 6847 barium meal examinations found 85 duodenal diverticula (1.2 per cent), 60 per cent of these cases being females. The commonest site for these diverticula is the second and third part of the duodenum, where they show a preference for the region of the ampulla of Vater (fig. 70).

Those of the *false* variety arise where the blood-vessels enter the bowel, along the inner and posterior aspects of the gut—the weak spot.



Fig. 68.—DIVERTICULUM FROM THE FIRST PART OF THE DUODENUM. THE DIVERTICULUM ARISES FROM THE LOWER AND OUTER ASPECT OF THE DUODENAL CAP, AND LIES OVER THE HEAD OF THE PANCREAS. (H. Cecil Hall)

They are protrusions of the mucosa and muscularis mucosæ through the muscular wall. Here the wall of the sac is composed of these structures in addition to the compressed and chronically inflamed extra-peritoneal tissues in the vicinity. Owing to the position they occupy they may be wholly or partially extra-peritoneal.

It is probable that they are more common than has been assessed by various radiologists. Some estimate their incidence as occurring in about 5 per cent of all barium meal examinations. Spriggs and Marxer in 1000 consecutive barium meal examinations conducted by them found 38 cases—3·8 per cent.

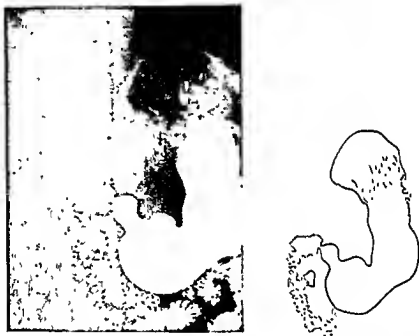


Fig 70.—DIVERTICULUM FROM THE SECOND PART OF THE DUODENUM—THE COMMONEST SITE. (H. Cecil Bull.)

Over 50 per cent of duodenal diverticula give rise to no symptoms at all. Some, on the other hand, will produce dyspeptic symptoms which may even simulate those of chronic gastric or duodenal ulcer. In some cases they will produce pain in the epigastrium which will arise half an hour to two and a half hours after meals; but as a rule such measures as dieting and the administration of alkalis, etc., afford little or no relief. Duodenal diverticula may become inflamed, ulcerate, give rise to hæmorrhage, or even perforate. As a sequel to perforation, a localised subphrenic abscess may develop. The larger varieties may become filled with food and form a tumour, which may press upon the duodenum and produce obstruction, and

also interfere with the flow of pancreatic juice or bile, thus favouring an infection of the pancreatic or biliary ducts.

Diverticula which produce no symptoms are best left alone as the operation for their excision may be difficult and not free from danger. Where operation is indicated complete excision of the sac, followed by invagination of the base of the sac into the lumen of the gut, is the method advocated as being that most likely to produce a satisfactory result. As many of these diverticula are attached to that portion of the duodenum which is not covered by peritoneum, most scrupulous care should be taken in the closure of the wound after excision. In certain cases where the mouth of the diverticulum is wide there may be an extensive gap to be sutured in the duodenal wall after excision of the pouch. In such cases, and particularly where there is any tension on or weakness of the suture line, it is a wise precautionary measure to occlude the pylorus and to perform a posterior gastro-jejunostomy to guard against the immediate danger of leakage and the possibility of subsequent stenosis.

Although excision is the treatment of choice, if this proves to be impossible, invagination of the diverticulum should be attempted. Cases occurring in the region of the duodeno-jejunal flexure and producing duodenal stasis may lend themselves to excision; but when this is impracticable a duodeno-jejunostomy will afford relief of symptoms.

Again, operation will be indicated where there is inflammation or gangrene of the sac, in which case the sac should be excised and the stump invaginated into the gut, as in the operation of appendicectomy. Owing to the sodden nature of the duodenum and the friability of the parts suture may be difficult, and as a precautionary measure both intra- and extra-peritoneal drainage should be provided.

As these diverticula are usually situated behind the duodenum or are imbedded in the pancreas or posterior to it, the duodenum should be mobilised by Kocher's method to render them freely accessible. Pouches beyond the inferior angle of the duodenum may be approached through the posterior peritoneum beneath the transverse mesocolon.

(f) *Chronic Duodenal Ileus.* (See also page 622.) This is a condition in which the duodenum is greatly distended as a result of the compression of its third part by the root of the mesentery, and of drag on the intestines. Other predisposing causes are extension of growth from the stomach or colon, malignant glands, or inflammatory lesions

in the region of the mesenteric root, such as gastro-jejunal ulcer or tuberculous glands (fig. 71).

In chronic duodenal ileus not only is the duodenum distended and its walls thickened, but the pylorus also may be dilated. The dilatation comes to an abrupt end where the superior mesenteric artery crosses the gut. Beyond this point the small intestine is collapsed. The condition predisposes to chronic gastric and duodenal ulcer. A well-defined case may be associated with dyspepsia, bilious vomiting, and symptoms similar in many respects to those of duodenal ulcer. The



Fig. 71 - DUODENAL DELAY, A REFLEX CONDITION. FROM A CASE OF TUBERCULOUS PERITONITIS. (U. Cecil Ball)

patient may suffer from intermittent attacks of epigastric pain, but the pain is not, as a rule, very severe and does not arise at any fixed interval after the intake of food. It is made worse by exercise, walking, and standing for long periods, but is relieved by postural treatment.

Physical examination may be negative, but more often than not signs of visceroptosis are in evidence. Some of the disappointments following operations for gastro-jejunal ulceration are due to the persistence of chronic duodenal ileus. A considerable amount of inflammatory thickening and fibrosis occurs at the root of the mesentery in gastro-jejunal ulceration, which may progress or persist even after



Fig. 71.—AN OMENTAL GRAFT IS SUTURED IN SUCH A MANNER AS TO PROTECT THE VERTICAL SUTURE LINE AND TO PREVENT THE FORMATION OF ADHESIONS.  
(After Killogg)



Fig. 73.—THE TRANSVERSE INCISION THROUGH THE PREHEPATIC FOLD AND ADHESIONS WHICH BIND THE DUODENUM TO THE UNDER SURFACE OF THE LIVER IS MADE VERTICAL BY CLOSING THE GAP BY THE METHOD SHOWN IN THIS ILLUSTRATION.  
(After Killogg.)



Fig. 72.—HEPATICODUODENAL FOLD. THE DUODENUM IS DRAWN UP TO THE UNDER SURFACE OF THE LIVER BY STRONG HANDS AND ADHESIONS, AND BY A WELL-MARKED HEPATICODUODENAL LIGAMENT, CAUSING A SLIGHT DEGREE OF CHRONIC PYLORIC OBSTRUCTION. THE INCISION FOR DIVIDING THIS FOLD IS HERE SHOWN.

(Adapted from "The Duodenum," by Dr. Edward L. Killogg, New York, Paul B. Hoeber Inc., 1933.)

a secondary operation has been performed to deal with the recurrent ulcer. The duodenal ileus remains undetected at operation, and may be an aggravating factor in the reactivation of ulceration in the stomach, in the duodenum, or at the site of the stoma if a new anastomosis has been made. The question will therefore arise of performing a duodeno-jejunostomy at the completion of operations on such cases.

(g) *Duodenal bands.* These may be congenital or acquired. Those of the acquired variety are secondary to inflammation of the gall-bladder, bile-ducts, or duodenal bulb. An illustration of a congenital duodenal band is the so-called hepatico-duodenal fold, which springs from the under-surface of the liver. This fold anchors the first portion of the duodenum, and even the pylorus, to the under-surface of the liver in the region of the portal fissure, and then sweeps across the gut to find an attachment to the colon. This is well depicted in figure 72, whilst figures 73 and 74 illustrate the operative procedure undertaken to rectify this obstruction. This tethering of the duodenum in such an abnormal position by an unusually short hepatico-duodenal fold, or a cystico-duodenal ligament which is a small band uniting the anterior surface of the duodenal bulb to the infundibulum of the gall-bladder, results in a mild degree of chronic obstruction and impairment of the motor functions of the stomach, accounting for such symptoms as epigastric pain, gastric distension, a sense of fulness, and occasional bouts of vomiting. These small bands, congenital or acquired, may produce pylorospasm and its consequent train of symptoms.

As the symptoms in some of these cases closely simulate those of chronic duodenal ulcer, X-ray investigation will often be found of great assistance or even indispensable, although the distortion, angulation, and constriction of the duodenal bulb resulting from these bands calls for great skill in interpretation.

(h) *Innocent new growths of the stomach.* (See page 519.) These are uncommon and give rise to few symptoms. They may cause pyloric obstruction, or, if large, produce a sense of fulness or even pain in the epigastrium. Vomiting is rare except where there is pyloric obstruction. Benign tumours may cause hæmatemesis or melaena. Occult blood tests and a barium meal examination will often indicate the condition present.

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## CHAPTER V

### COMPLICATIONS OF PEPTIC ULCER

THE following are the commonest complications of chronic peptic ulcer :

(1) Acute.

- (a) Hæmorrhage. (See page 578.)
- (b) Perforation.

(2) Chronic.

- (a) Adhesions.
- (b) Fistula.
- (c) Hour-glass stomach.
- (d) Pyloric obstruction.
- (e) Ulcer-Cancer. (See page 561.)

#### PERFORATION OF GASTRIC AND DUODENAL ULCERS

This is the most important complication of chronic peptic ulcer, and has been estimated to occur in 10-15 per cent of all recognised cases of chronic gastric and duodenal ulcer. It is the most common cause of death in ulcer cases. "This is well seen from the statistics relating to 500 consecutive cases of chronic ulcer found post-mortem at the Leeds General Infirmary. Out of 96 cases in which death was attributable to a gastric ulcer, 62 (64.5 per cent) followed acute and 6 sub-acute perforation ; out of 200 cases in which death resulted from duodenal ulcer 170 (85 per cent) followed acute and 5 (2.5 per cent) sub-acute perforation." (Hurst and Stewart, *Gastric and Duodenal Ulcer*. Oxford Med. Publications, 1929.)

The frequency of perforation is obviously increasing yearly, as the records of any large general hospital throughout this country will show.

### *Types of Perforation.*

Three types of perforation are described :

(a) *Acute.* In acute perforation a sudden rupture of the base of the ulcer takes place, with the result that the contents of the stomach or duodenum pour into the peritoneal cavity (fig. 75).

(b) *Sub-acute.* In sub-acute perforation only a circumscribed area of the peritoneal cavity becomes contaminated by the leakage. Such localisation may be dependent upon a variety of factors, such as the smallness of the perforation, the stomach being empty, adhesions being present in the vicinity of the ulcer, or the plugging or sealing off of the rent shortly after perforation either by adjacent omentum, a neighbouring viscus, flakes of lymph, a particle of food, or a protrusion of mucous membrane through the rent.

(c) *Chronic.* Here the ulcer has penetrated a neighbouring viscus, and the base of the crater is no longer formed by the stomach or duodenum, but by the structure which it has eroded. Sometimes as the result of a slow leak a localised abscess may develop in the vicinity of the ulcer, in the lesser sac—*perigastric abscess*, or underneath the diaphragm—*subphrenic abscess*.



Fig 75—ACUTE PERFORATION OF A CHRONIC GASTRIC ULCER.  
(Museum, Royal College of Surgeons.)

### *Acute Perforated Peptic Ulcer.*

(a) *Sex Incidence.* Perforation is more than twenty times commoner in males than it is in females, and this is irrespective of the site of the lesion.

TABLE I. SEX INCIDENCE.

<i>Name of Author.</i>	<i>Males.</i>	<i>Females.</i>	<i>Total.</i>
Scotson . . . . .	170	11	181
Black . . . . .	50	0	50
Hamilton Bailey . . . . .	84	9	93
Gilmour and Saint . . . . .	58	6	64
Dineen . . . . .	138	4	142
Shawan . . . . .	223	4	227
	—	—	—
<i>Totals . . . . .</i>	723	34	757
	—	—	—

(b) *Age Incidence.* Perforation is commoner during the middle years of life—30-45, being rare before 20 and after 50, and probably has its highest incidence around the age of 40. Perforation may, however, occur in an infant of a few days old or in extreme old age. On the whole, gastric ulcers perforate at a later age than duodenal ulcers. The age incidence at the time of perforation of 227 cases is analysed by Shawan (*Ann. Surg.*, p. 210, Aug., 1933) in Table II.

TABLE II. AGE INCIDENCE.

<i>Age.</i>	<i>Number.</i>	<i>Percentage.</i>
Under 20	5	2.20%
20-30	61	26.87%
30-40	82	36.12%
40-50	54	23.78%
50-60	22	9.69%
60-70	3	1.32%
	—	—
	227	—
	—	—

(c) *A Previous History of Gastric Disturbance.* Patients who perforate may give :

- (i) A very long history—years.
- (ii) A very short history—days or weeks.
- (iii) No previous history of gastric disturbance, acute perforation being the first symptom.

In about 80 per cent of cases a very definite history of peptic ulcer will be obtained, leading to the conclusion that the older the ulcer the more likely it is to perforate. Too much reliance cannot be placed upon the accuracy of histories obtained when patients are examined whilst suffering the tortures of perforation. A further inquiry when such patients are convalescing will often yield a clear history of chronic dyspepsia, frequently dating back many years.

"The average length of ulcer history prior to perforation was well over twice as long in the female sex as in the male, and hence it may be assumed that perforation of a peptic ulcer is more likely to occur in a male than in a female subject." (Scotson, *B.M.J.*, p. 680, Oct., 1933.)

In some 10-15 per cent of cases there is a short history of gastric disturbance—days or weeks. In these cases the symptoms are often aggravated just prior to perforation. In some instances the perforation would appear to be brought on by trauma, physical exertion, indiscretions in diet, alcoholism, or even worry. Hurst states that it is most unusual for a perforation to occur during strict medical treatment, or even while the patient is careful as regards his diet and method of living. I have, however, known of two cases where perforation occurred while the patient was undergoing medical treatment as an in-patient, another case which perforated on the morning fixed for operation, and a further case which perforated during a harium meal examination of the stomach. In each instance it was a chronic duodenal ulcer which had perforated.

In a few cases no previous history of indigestion is given, there being no inaugural symptoms prior to the dramatic onset of perforation. While the incidence of perforation in these cases may be fairly assumed to be from 5-10 per cent, some authors place it at a very much higher figure. For instance, in the 50 cases operated on by Black, 13 denied any previous history of digestive upset, and in 227 cases recorded by Shawan 32 cases also denied a previous history of dyspepsia.

It is probably no exaggeration to state that not more than 10 per cent of perforated cases have had systematic courses of medical treatment, that approximately 40 per cent have had only irregular and occasional treatment by dieting and with doses of sodium bicarbonate and various bismuth mixtures, and that 50 per cent have undergone no treatment whatsoever.

In a certain proportion of cases a history of perforation in the past is given, and there are on record a large number of peptic ulcer cases which have perforated after a second, third, fourth, or even a fifth operation for relief of the condition. A patient, therefore, who survives

a second perforation and suture of the ulcer is in urgent need of partial gastrectomy as soon as his condition permits of such a procedure. If gastro-jejunostomy is undertaken for recurrent perforation strict dieting and medicinal treatment will be essential for a number of years to diminish the risk of the formation of an anastomotic ulcer, but after gastrectomy these precautions are necessary for only a short time.

(d) *Site of Ulcer.* Ninety per cent of peptic ulcer perforations are duodenal, and 90 per cent of peptic ulcers that perforate are on the anterior wall of the stomach or duodenum. The site of the ulcer is obvious in the majority of cases; it is either gastric, pyloric, or duodenal. In some instances the anatomy of the duodenal bulb and of the pylorus may be greatly distorted as the result of the ulceration, the veins of Mayo may be obliterated or dragged out of their normal course, the pyloric sphincter may be indurated with scar tissue or swollen with inflammatory products, and the involved portion of gut may be so fixed and encased in tethering adhesions, that it is, in fact, difficult to determine at sight whether the ulcer that has perforated is duodenal or pyloric. The doubtful cases are classified by some authors as duodenal ulcers, and by others as pyloric ulcers, there being no criteria for these cases owing to the anatomical distortion described above. The margin of error in the classification of these cases may be very great as the operation of suture of a perforated peptic ulcer has often to be performed expeditiously, the saving of the patient's life, and not the determination of the exact anatomical position of the ulcer, being the main concern of the moment.

Again, the majority of such operations, or at least the recording of the notes, are undertaken by resident house-officers who have no particular interest in the case from a statistical point of view. It must therefore follow that in the drawing up of statistics dealing with the differentiation between the percentage incidence of perforated gastric and perforated duodenal ulcers there will be wide variations owing in some instances to an insufficient scrutiny of the parts at operation, necessitating the interchangeable use of the terms "pyloric" and "duodenal," or else to the preconceived and dogmatic views held by the surgeon. We would therefore advise that where there is real ground for doubt such perforations should be classified as duodenal rather than pyloric.

Whereas the large indurated ulcer is usually found in association with a long history of dyspepsia, the small soft type of ulcer is more often seen in patients with a short or negative history of

previous gastric trouble. The majority (90 per cent) of chronic duodenal ulcers that perforate are situated in the first part of the duodenum and on the anterior or antero-superior surface. About 10 per cent of duodenal ulcers that perforate are situated on the posterior wall of the duodenum. They do so when the ulcer spreads towards the superior or inferior surface of the gut. The perforation being small and slit-like renders it most difficult to find and to close at operation. Owing to the small size of the perforation and to the fixation of the gut to the pancreas or adjacent viscus, a localised intra-peritoneal abscess, e.g. subphrenic, is more likely to occur than general contamination of the peritoneal cavity.

An extra-peritoneal perforation may occur with these posteriorly-placed ulcers, and the extravasated fluid collect in the region around the right kidney and produce signs of a perinephric abscess. The duodenal contents travel downwards behind the peritoneum towards the right iliac fossa, producing a swelling in this region which may simulate an appendix abscess. When such an abscess is drained an external duodenal fistula is formed.

The majority (90 per cent) of perforated ulcers in the stomach are found on the anterior surface of the lesser curvature. It is rare to find a perforated ulcer near the cardia. When occurring in this position they are difficult to suture and may even be overlooked. Any callous ulcer that is situated in the region of the greater curvature, whether perforated or not, is probably malignant.

Posterior ulcers of the stomach usually perforate superiorly at a point where they are not attached to the pancreas, i.e. in the region of the lesser curvature. Perforation may then occur into the lesser sac or between the layers of the gastro-hepatic omentum. Multiple perforations occurring simultaneously have been described, but are very rare.

Having dealt successfully with one perforation at operation, a further search for any other perforation should invariably be made. Multiple perforations are usually found close together. It is more common to find that the base of one large chronic ulcer has perforated at two separate points than that two separate ulcers have perforated simultaneously. It is most exceptional to find a chronic gastric ulcer in association with a perforated duodenal ulcer, and vice versa. In females a perforated duodenal ulcer is commoner than a perforated gastric ulcer, although the general opinion is that the reverse is the case.

(e) *Size of Perforation.* It may be generally stated that the larger the perforation the higher the mortality. Gastric ulcer

perforations are usually very much larger than duodenal ulcer perforations; consequently the prognosis is always more grave and the mortality higher with these gastric cases. The aperture is round, oval, or slit-like, and varies considerably in size. It may be a minute puncture, such as would be produced by a pin-prick, but it is usually from  $\frac{1}{8}$ – $\frac{1}{4}$  inch in diameter. Perforations with a diameter of  $\frac{1}{2}$  inch or even more have been recorded.

Perforation is a rapid process, even in chronic ulcers, and is due to the sudden sloughing of an unsupported portion of the floor of the ulcer secondary to a slow progressive process of devascularisation (Hurst and Stewart). Immediately after perforation has occurred peritonitis develops, but at first it is non-infective, and is produced by the irritant action of the acid gastric juice.

It is difficult to determine how long it takes for this simple irritative peritonitis to develop into a virulent septic peritonitis, the result of the invasion of the peritoneal cavity by pyogenic organisms. It depends upon many factors, such as the size of the perforation, the reaction and composition of the gastric contents, and the partial or complete flooding of the peritoneal cavity. When foul gastric contents, neutral or alkaline in nature from a secondary gastritis or from the ingestion of alkaline drugs prior to perforation, are free to escape through a large rent in the stomach, the onset of a virulent septic peritonitis can only be delayed for a few hours. When, however, the escaped gastric juice is strongly acid in nature, and therefore actively bactericidal, infection is considerably inhibited. In the average case it may safely be assumed that during the first twelve hours the peritonitis is non-infective; where, however, a perforation has existed for more than twelve hours the peritoneal fluid will be infective in character.

The amount and the nature of the fluid in the peritoneal cavity will have an important bearing on the prognosis, as the more fluid there is (and particularly if this fluid is thick) the worse the outlook and the higher the mortality. When large accumulations of fluid are found, the intestines, through being partly submerged and freely bathed in the turbid irritating fluid, are unable to form adhesions or to limit off the contamination. When the perforation is small and the stomach is empty a localised abscess may form. When the pus tracks upwards towards the diaphragm a subphrenic abscess develops, while when it becomes shut off in the pelvis a pelvic abscess results.

(f) *Is Acute Perforation more common in Acute or Chronic Peptic Ulcers?* Acute perforation may occur in acute or chronic ulcers, and

the general opinion is that in over 90 per cent of cases the ulcer that perforates is a chronic one. Lord Moynihan (*Addresses on Surg. Subjects*, p. 245, Saunders, 1928), drawing from his wide experience, writes as follows :

"The condition is therefore one of acute perforation in a chronic ulcer. It is necessary to be precise upon this point, for the literature of this subject contains many inaccurate assertions that perforation occurs in acute rather than in chronic ulcers. In twelve consecutive years at the Leeds Infirmary (1910-1921) there were 61 deaths from perforation of a gastric ulcer ; in 60 cases the ulcer was of the chronic variety. There were 117 deaths from perforation of a duodenal ulcer. In 12 of these cases there was an acute ulcer ; in 4 of the 12 there was a chronic ulcer also, and it was in every case the chronic ulcer which had perforated. In 8 cases an acute ulcer had perforated."

It is sometimes exceedingly difficult at operation to determine whether the ulcer which has perforated is acute or chronic, as even in the case of acute ulcers there is always a thickened, swollen, and indurated area found around the margin of the ulcer.

If perforation of acute ulcers were a common condition, simple closure of the rent is all that would be necessary to effect a complete and permanent cure in all such cases, and there would be no recurrence of symptoms after the patient had recovered from the effects of his operation. It is well known, however, that a large number of cases treated by simple closure experience further trouble which calls for medical treatment or for further operative interference.

### *Signs and Symptoms*

The signs and symptoms produced by the perforation of a peptic ulcer depend upon a number of factors, the most important being the length of time which has elapsed since the perforation took place. There are three distinct stages in the pathological process and in the corresponding clinical picture :

- (1) The stage of prostration.
- (2) The stage of reaction ; and
- (3) The stage of peritonitis.

An acute exacerbation of symptoms is found in about 25 per cent of cases before perforation occurs. Although the partaking of a large meal, straining, coughing, or exercise may be influencing factors in precipitating perforation, on the other hand the rupture may occur while the patient is resting or even asleep. As stated above, this complication is rare when the patient is undergoing adequate medical treatment.



(1) *Stage 1—the stage of prostration.* This is sometimes called the stage of primary shock.

“But if the word ‘shock’ is strictly interpreted it means a condition in which the blood-pressure is low, the pulse fast, and the total blood volume diminished. This is not the state of patients within an hour or two of the occurrence of perforation. If we use the word ‘shock’ to describe the *appearance*, and not the *state* of a patient, then and then only can we say that the victims of a perforation suffer from ‘shock.’” (Moynihan, *Abdominal Operations*, Vol. i, p. 226, 1926.)

During this stage *prostration* is the outstanding feature. The symptoms which arise with such dramatic suddenness are due to the perforation itself and to the irritation of the peritoneum by the escaped gastric contents. This sudden and violent irritation of the peritoneum produces profound reflex effects on the circulatory and nervous systems. It may be so severe as to cause sudden death. After an interval, which may vary from a few minutes to half an hour or even up to two hours, reaction sets in and is followed by the symptoms due to a spreading peritonitis.

At the moment of perforation the patient is suddenly seized with an acute agonising pain which is felt all over the abdomen, but is more intense in the epigastrium over the site of rupture. This may be more marked on the right side when the ulcer is duodenal, and on the left when gastric.

The pain is usually constant. Immediately perforation occurs the patient is plunged into a state of prostration. He lies almost rigid and is frightened to move as he knows that the slightest movement will aggravate the pain. His face indicates the agonies he is suffering; it is anxious, pale, livid or ashen, and is bathed in cold sweat. His eyes are attentive and expressive of fear. The extremities are cold and drenched in perspiration. Retching or vomiting is present in about 50 per cent of cases, and is commoner with gastric than with duodenal ulcer. Retching may be troublesome, particularly when the stomach is empty. Blood is very rarely vomited after perforation has occurred.

The pain may be referred to the top of one or both shoulders. If present on the right side it would suggest a perforated duodenal ulcer; if bilateral that a gastric ulcer situated on the anterior surface has ruptured. This referred pain is caused by irritation of the diaphragm, and is referred through the phrenic nerve to the cutaneous distribution of the fourth cervical spinal segment. (Zachary Cope, *The Early Diagnosis of the Acute Abdomen*, p. 13, Oxl. Med. Publications, 1932.) Such a referred pain is also present in diaphragmatic pleurisy. It

is well to inquire about this pain when the diagnosis lies between acute perforation and acute appendicitis, as it is absent in the latter condition.

The temperature is nearly always sub-normal ( $95-96^{\circ}$  F.); the pulse may be normal, but is generally small, weak, and rapid (about 100); and the respirations are shallow and thoracic in nature owing to the immobility and fixation of the diaphragm—the largest abdominal muscle.

An examination at this stage will show the abdomen to be retracted, and the rectus muscles may stand out, firmly contracted, with visible muscular intersections. The abdomen is immobile, there being no movement on respiration.

On palpation the muscles are tensely rigid and board-like. This rigidity is universal, and extends into the flanks. On the most prolonged and searching examination relaxation will not occur, even for a moment. When seen in the early stage the muscle overlying the perforation is particularly rigid and metal-like, and the tenderness, although generalised, is more acute in the region of the rupture.

*This rigidity of the abdominal muscles arises immediately after perforation has occurred and persists throughout this stage, the stage of reaction, and into the final stage of peritonitis, when it may lessen to some degree owing to toxic effects on the neuro-muscular system.*

The administration of morphia has the effect of easing the pain, rendering the patient more comfortable and less apprehensive, and of slowing the pulse-rate, but has little or no direct influence upon the stubborn rigidity of the abdominal muscles. Complete relaxation is difficult to obtain, even when a general anæsthetic has been administered.

The fluid escaping from a perforated duodenal ulcer may, by tracking down "Moynihan's gutter," reproduce or mimic the signs of acute appendicitis. The gastro-duodenal contents, trickling out of the aperture in the gut, are thrust against the little "hillock" of the transverse mesocolon and deflected downwards to the outer side of the ascending colon to the right iliac fossa, where pooling of the fluid occurs with the production of signs similar to those found in many cases of acute appendicitis.

In cases such as these the rigidity on the *right* side of the abdomen is more pronounced and more obdurate than on the left side. It is only a question of hours, however, before the pelvis is flooded, and as more fluid accumulates it ascends to the left iliac fossa until eventually the whole peritoneal cavity is contaminated. The rigidity in such cases will then become diffuse.

(2) *Stage 2—the stage of reaction.* This has been called the stage of delusion, as “shock” gradually passes off and there is, to all appearances, a general improvement in the patient's condition. Pain, although still present, is not so acute; its sharp edge is dulled. The patient will state that he feels better and that granted a little rest all will be well. He feels warmer, his colour improves, and he loses that strained, anxious appearance. Sweating may still be profuse, but the extremities are now no longer chilled. Vomiting or retching, which may have been very troublesome, ceases. The temperature is normal, and the pulse is bounding and normal in rate. The patient is thirsty and often asks for a drink. The trained eye, however, will detect that the alae nasi may be working, that the respirations are still shallow, jerky, and costal in type, and that the patient lies immobile with the knees drawn slightly upwards.

An examination of the abdomen will reveal that there is something seriously amiss, and that a condition consistent with some grave abdominal catastrophe is present, calling for immediate surgical measures. The abdominal wall is rigid to a marked degree, and is still board-like, tender, and flat. The pelvic peritoneum will be found to be exquisitely tender on rectal examination. There may be some diminution of liver dullness. If there is no abdominal distension and there is a diminution of liver dullness it may be assumed that there has been perforation of a hollow viscus. It is best to percuss in the mid-axillary line, about two or three inches above the costal border, and if distinct resonance be found here then there can be no doubt that a perforation is present (Zachary Cope). This sign is positive in only a minority of all cases. In the presence of abdominal distension it is, however, of no diagnostic value. Shifting dullness may be detected in the flanks, but should not be sought for as every movement of the body produces agonising pain.

It is in this stage, when apparent recovery is taking place, the patient is feeling better, and the temperature and pulse are normal, that most of the errors in diagnosis occur. *The physical signs, however, leave no room for doubt.*

It must be emphasised here that abdominal rigidity during this stage is as marked, or even more so, than it is during the stage of prostration or the stage of peritonitis.

(3) *Stage 3—the stage of peritonitis.* During this stage of frank peritonitis toxic shock is present. Vomiting returns and may be frequent, whilst hicough may sometimes further distress the patient.

Owing to profuse sweating, vomiting, and the pouring out of fluids into the contaminated peritoneal cavity, thirst becomes insatiable and the tissues become shrunk. The body is cold and clammy, the face is now livid, the lips purple, and sordes collect between the teeth. The tongue is dry, fissured, and brick-red in colour. The breath is offensive; the eyes are glistening and bollow, and are surrounded by dark rings. The pulse becomes rapid and small, until eventually it is thready. The temperature may rise a degree above normal, or may fall ominously to 96 or 95 degrees F. Respirations are laboured and very rapid. The abdomen gradually distends and becomes tense and tender, and the rigidity which before was inflexible is now more yielding. Owing to the advent of ileus and meteorism the movements of the intestinal tract gradually subside and are stilled. Flatus and fæces are therefore not passed. Owing to the absence of peristaltic movements true vomiting also now comes to an end, and is replaced by regurgitation of "coffee grounds" through the mouth. There may be retention of urine, or, if urine is passed, the act of micturition is difficult and painful, and the urine voided is scanty, loaded with albumen, and contains traces of indican.

The patient may either drift into delirium followed by coma, or may remain acutely conscious to the end. In an average case death occurs two to five days after perforation.

### *Diagnosis and Differential Diagnosis*

It is essential that an accurate and correct diagnosis be made in cases of perforation. Where this is impossible it is just as important to recognise that there is an acute abdominal catastrophe requiring immediate surgical interference. The diagnosis is not difficult and is probably correctly made in fully 90 per cent of cases. In possibly 10 per cent the signs and symptoms may be equivocal or atypical, suggesting that some acute medical condition, such as diaphragmatic pleurisy, ptomaine poisoning, etc., is present, resulting in a mistaken diagnosis and a lost opportunity of dealing surgically with the lesion without delay.

If the patient gives a history of previous gastric trouble, and especially if he has been treated medically for peptic ulcer, the advent of a sudden agonising abdominal pain, followed by prostration and abdominal rigidity, should leave no doubt that this is a case of acute perforation.

In cases where there is no previous history of indigestion, or where

the patient, being in the throes of intense suffering, is unable to give a coherent or satisfactory account of his past or present symptoms, the diagnosis may present certain difficult features; but if the physical examination is conducted methodically it will be apparent that an acute surgical condition is present, demanding immediate laparotomy.

*The stage of reaction is still too often the stage of delusion in which a wrong diagnosis is made.* But if abdominal rigidity and tenderness are still present, and all the local signs are even more definite and more positive than in the first stage, the diagnosis should not be influenced by the patient's assertion that the pain is better and that his condition is improving.

In the third stage the diagnosis will never be in doubt, as it is unmistakably one of generalised peritonitis.

### *Differential Diagnosis.*

(1) *Medical conditions* which simulate acute perforation, and in which operative interference is definitely contra-indicated:

(a) Colic.

- (i) Renal.
- (ii) Biliary.
- (iii) Intestinal.
- (iv) Lead colic.

(b) The gastric crises of tabes.

(c) Ptomaine poisoning.

(d) Acute indigestion.

(e) Acute thoracic diseases.

- (i) Acute diaphragmatic pleurisy.

- (ii) Acute lobar pneumonia.

- (iii) Acute pericarditis.

- (iv) Coronary thrombosis.

- (v) Dissecting aneurysm of the thoracic or abdominal aorta.

(f) Acute alcoholism.

(g) Meningitis.

(2) *Surgical conditions* which demand operative treatment, but which are at times difficult or impossible to distinguish from acute perforated peptic ulcer:

(a) Acute appendicitis.

(b) Acute hæmorrhagic pancreatitis.

- (c) Acute intestinal obstruction.
- (d) Acute infection of the gall-bladder.
- (e) Mesenteric embolism or thrombosis.
- (f) Ruptured ectopic gestation.
- (g) Acute diffuse peritonitis from any cause.
- (h) Acute exacerbation of a chronic gastric or duodenal ulcer.

*Medical Conditions.* (a) *Colic.* It is very unlikely that renal, biliary, or intestinal colic will cause difficulty in diagnosis or be mistaken for an acute perforation. This cannot, however, be said of lead colic, which often closely resembles a perforation.

In *renal colic* the pain may be intense and collapse profound, but the abdomen is not universally rigid and tender. There may be some rigidity over the affected kidney, but it is of an intermittent nature. There is considerable restlessness, but the movements of the body do not aggravate the pain; they rather tend to afford relief. The radiation of the pain downwards into the groin may be significant, as may be a previous history of hæmaturia or urinary trouble. There is no diminution of liver dullness, and the pelvic peritoneum is not tender on rectal examination.

In *biliary colic* there will be a past history of gall-bladder disease rather than of peptic ulcer. The pain, which is localised to the gall-bladder, may be acute and prostrating, colicky in nature, but continuous between the sharp bouts. It is mainly localised to the right hypogastrium, but radiates to the back between the shoulder blades and upwards into the right scapular region.

During an attack of biliary colic the patient writhes in agony, restlessness is very marked, and retching, or vomiting of frothy bile, may be incessant.

On examination there may be some tenderness and rigidity which is localised to the upper right quadrant of the abdomen, but there is not that universal and inflexible rigidity of the abdominal muscles nor is any tenderness elicited on rectal examination.

*Lead colic* differs from the above forms of colic in that there is generalised abdominal rigidity and tenderness, but the rigidity appears to be, and often is, definitely less continuous and less absolute than in cases of perforated peptic ulcer.

The abdomen is retracted and hard, but by maintaining a firm and even pressure with the hand there will be some yielding here and there. The pain is colicky in nature—not continuous. There may also be a history of intermittent intestinal colic associated with a severe form of constipation. The patient may admit that his work brings him in contact with lead, and the blue line of lead poisoning may be evident on the gums. Even after the patient has finished working with lead for a very long time this indication will persist.

If when the blood is examined punctate basophilia is found in addition to a secondary anaemia, this will constitute valuable corroborative evidence, as it is present only during the acute stages of lead poisoning.

(b) *The Gastric Crises of Tabes.* In this condition there is little or no abdominal rigidity or tenderness, and there may be a previous history of syphilis. It should, however, be remembered that peptic ulcer and tabes may co-exist.

(c) *Potomane Poisoning.* Here vomiting, abdominal pain, and collapse are present. There is, however, no generalised abdominal rigidity, liver dullness is not impaired, the pelvic peritoneum is not tender, nor is there, as a rule, any previous history of chronic indigestion.

(d) *Acute Indigestion.* The diagnosis of some forms of acute indigestion, attributable to dietetic errors, and particularly where there is severe epigastric pain associated with collapse and extreme pallor, may be confusing, and may even lead to a

tentative diagnosis of perforation. The history in such cases and a careful physical examination will avoid the performance of any unnecessary operation.

During the most acute phases of an exacerbation of a chronic peptic ulcer, the signs and symptoms of perforation may also be mimicked.

(c) *Acute Thoracic Diseases.* Each of the diseases mentioned under this heading may, in its initial phase, simulate perforation. This is especially so in diaphragmatic pleurisy and lobar pneumonia. In these diseases, however, the onset is less violent and sudden, and abdominal rigidity, although present, is not generalised and is less absolute. On steady pressure with the hand, rigidity will give place to relaxation, particularly on expiration. The face is flushed, there is a high colour and a cyanotic tinge in the cheeks, and herpes may appear round the lips. The temperature is high from the start— $103^{\circ}$ – $104^{\circ}$ F.—and respiration is hurried and increased out of proportion to the pulse-rate. This pulse:respiration ratio, normally four to one, becomes three or two and a half to one. Physical signs may be present on examination of the chest, but are, as a rule, difficult to detect at the inception of these diseases.

In cases of doubt it is best to wait a few hours, to call in further medical advice, and to conduct another careful examination. The temperature, pulse, and respirations are recorded every hour on a special chart.

*Surgical Conditions.* (a) *Acute Appendicitis.* A diagnosis of acute appendicitis is sometimes made in cases of perforated duodenal ulcer. This is especially so when an examination is conducted shortly after perforation has occurred, or during the stage of reaction. As previously described, the escaping duodenal contents may be deflected to the right iliac fossa in the first instance, and collecting there may produce signs and symptoms which may be difficult to distinguish from those of acute appendicitis. Although the local signs may simulate appendicitis, a careful consideration of the history and the order of onset of the symptoms will assure a correct diagnosis in the majority of cases.

The onset of appendicitis is usually insidious, and the initial epigastric pain complained of by the patient is often mild in character. This is in contrast with the acute, sudden, agonising epigastric pain, followed by abdominal rigidity, which is seen in perforation. Again, shoulder pain is very rarely felt in cases of appendicitis. Nevertheless, the sequence of epigastric pain, nausea, vomiting, and pain in the right iliac fossa, which is seen shortly after the perforation of a duodenal ulcer, may be sufficient in some instances to confuse the issue and lead to removal of the appendix before the true lesion has been discovered.

accompanied by marked toxæmia. A mild degree of jaundice is present in slightly less than 50 per cent of published cases. Grey Turner has drawn attention to ecchymoses which may occur in one or both loins in late cases. Vomiting is often continuous and of the obstructive type. When the stomach is empty retching may be intractable. The urinary diastase is stated to be increased ten times or more.

A diagnosis of acute perforation is often made in cases of acute hæmorrhagic pancreatitis, as the latter condition, owing to its rarity, is seldom borne in mind. In such a case if the possibility of the condition is considered, the diagnosis at once appears obvious.

(c) *Acute Intestinal Obstruction.* In the early stages of obstruction the abdominal wall is usually flaccid. As the obstruction progresses there may be distension, varying with the site of the obstructing agent. From the very first, vomiting is a most characteristic feature. The alterations in the character of the vomited matter—stomach contents, bile, dark fluid, and fæculent vomit—will at once suggest that an unrelieved obstruction is present. In acute perforation there may be vomiting, but it is never fæculent.

(d) *Acute Inflammatory Conditions of the Gall-bladder.* These may be very difficult to distinguish from perforation, particularly the acute obstructive types of cholecystitis which are associated with severe vomiting and epigastric pain. The signs in acute diseases of the gall-bladder are, however, more localised to the right upper quadrant, and occasionally an enlarged gall-bladder can be palpated. The symptoms, too, are not so severe as those of perforation, although vomiting may be uncontrollable. The past history would suggest gall-bladder trouble rather than peptic ulceration.

(e) *Mesenteric Embolism or Thrombosis.* Both these conditions produce sudden violent abdominal pain which is followed in a short time by prostration. When the bowels are evacuated blood will often be found to be present in the stools, or these may be almost wholly composed of blood. The abdominal pain is constant and fairly diffuse, but at intervals it becomes colicky in nature.

On examination the rigidity and tenderness will be found to be very much less than in cases of acute perforation. A deathly pallor accompanies other signs and symptoms found with a severe internal hæmorrhage. The pulse will be quick—steadily rising—and weak, the temperature sub-normal, and the respirations shallow. The collapse, which is present from the first, shows no improvement but rather increases with the passage of time. Through the tender and slightly rigid abdominal muscles a diffuse, ill-defined tumour—clotted blood filling the gut—may be felt. There may be a history of cardio-vascular disease, or of previous embolism elsewhere.

(f) *Ruptured Ectopic Gestation.* In such cases there may be a history of irregular periods, but this is by no means always the case. Shortly after the rupture there is fainting, collapse, and severe abdominal pain which is chiefly localised to the hypogastrium. On examination there will be signs of internal hæmorrhage, blanching of the lips, tongue, sclerotics and nails is noticeable, and there may be air-hunger. There is, as a rule, absence of abdominal rigidity, but some tenderness will be elicited on palpation. The lower half of the abdomen often feels tumid. The temperature may be normal or sub-normal, and there is usually considerable acceleration of the pulse-rate. Respiration is quick and shallow.

On rectal examination there will be some tenderness in the pouch of Douglas, while on vaginal examination the cervix and uterus may be found to be slightly enlarged and of softer consistency than usual. If the rupture is recent no definite pelvic tumour can be identified in the majority of cases.



*Pre-operative Treatment*

As soon as a diagnosis of acute perforation has been arrived at, the following measures are adopted while arrangements are being made for immediate operation :

(a) Morphia, 1 gr., with atropine,  $\frac{1}{2}$  gr. ; omnopon,  $\frac{1}{2}$  gr., with scopolamine,  $\frac{1}{16}$  gr. ; or any pre-anæsthetic of choice, is first injected.

(b) The patient is placed in Fowler's position and maintained in this position while being transferred to the operating theatre.

(c) No fluids are given by the mouth.

(d) 100 cc. of 6 per cent saline and 100 cc. of 10 per cent glucose are injected intravenously.

(e) A catheter is passed to ensure that the bladder is empty.

(f) The abdomen is prepared. This may be commenced shortly after the injection of the pre-anæsthetic and when it is evident that some relief of pain has been afforded ; but it is best to defer the preparation of the abdomen until the patient is under the anæsthetic, i.e. in the operating theatre.

(g) The blood-pressure, both systolic and diastolic, should be recorded, and this information should be handed to the anæsthetist as, if the blood-pressure is low, it may influence the choice of anæsthetic.

(h) The mouth should be thoroughly washed out with some mild antiseptic lotion before the anæsthetic is administered.

*Operative Treatment*

The following operations are performed for acute perforated peptic ulcer :

- (1) Simple suture.
- (2) Suture followed by gastro-jejunostomy.
- (3) Excision of the ulcer and suture (pyloroplasty).
- (4) Partial gastro-duodenal resection.
- (5) Temporary gastrostomy or duodenostomy.

Certain points in the technique of these various operations employed for perforation, including the advantages, the disadvantages, the immediate results, and the relative merits of each, will now be discussed.

*Simple Suture*

This operation is simple and quick to perform, and does not necessitate any special experience or skill in abdominal surgery; nor does it subject the patient to prolonged anæsthesia. The operation aims at warding off the immediate danger in a patient who is gravely ill, while subjecting him to the minimum amount of operative trauma. This being the case the prognosis should be good—the strongest plea for the performance of this operation. Although no special measures are undertaken to guard against the onset of late complications, such as pyloric stenosis, the end-results are, on the whole, fairly satisfactory. Approximately 40 per cent of such cases are symptomatically cured; 30 per cent will have recurrent symptoms necessitating treatment by dieting and with medicines; whilst a further 30 per cent will develop complications requiring further operation.

Those who discountenance this operation state that in certain instances there will be almost complete occlusion of the gut following suture, necessitating a primary gastro-jejunostomy to overcome the obstruction.

The advocates of simple suture affirm, however, that immediate stenosis following closure of the perforation does not usually occur to a degree which would hinder the emptying of the stomach, and that should pyloric stenosis supervene at a later date, it could be satisfactorily dealt with, the patient being in a fitter condition to withstand the operation. The replies by a large number of American surgeons to Guthrie's questionnaire with regard to the advisability of performing a primary gastro-jejunostomy show that the majority are conservative and favour the operation of simple suture alone. As far as the mortality is concerned, a great deal will depend upon the time which has elapsed since perforation occurred. Therefore a series containing a large number of late cases will, of necessity, have a higher percentage of deaths. It is claimed, however, that the mortality from simple suture is lower than that of any other operation which is performed for perforation.

*The Operative Technique of Simple Suture*

There is a choice of three incisions :

- (1) Mid-line.
- (2) Paramedian.
- (3) Transrectus.

*Pre-operative Treatment*

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*The Operative Technique of Simple Suture*

There is a choice of three incisions:

- (1) Mid-line.
- (2) Paramedian.
- (3) Transrectus.

I would favour the mid-line or right paramedian incision. The mid-line incision gives ample exposure and can be rapidly and securely closed.

*Search for the Perforation.* On opening the peritoneal cavity there may be an escape of gas, or the whole wound may be quickly flooded with the fluid contained in the abdominal cavity.

The edges of the incision should be well retracted by the assistant's hands, whilst the surgeon grasps the stomach in his right hand and draws it over to the left to bring the pylorus and duodenum better into view. In most cases the perforation is easily seen. It may be circular or oval, and may vary considerably in size, although it is usually about a quarter of an inch in diameter.

Through this rent the gastric contents will be seen to pour, intermittently or continuously. In the immediate vicinity of the perforation the gut will appear œdematous and thickened with inflammatory exudates. The ulcer itself will be felt to be hard and leathery, and this induration may involve a considerable portion of the viscus. If the perforation does not at once come into view, a methodical search of the whole stomach and duodenum must be made.

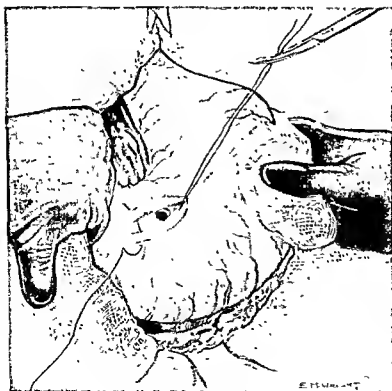
The aperture may be plugged by a portion of omentum, glued to an adjacent viscus, sealed off with flakes of lymph, or be situated in a somewhat inaccessible position.

The continuous welling up or gushing of fluid from one particular spot may lead to its detection. As the ulcer is hard, any localised induration in the stomach or duodenum should be carefully palpated and inspected, in order to discover the site of the ulcer. The possibility of more than one ulcer, or simultaneous multiple perforations, being present should be borne in mind. There may also be perforation of a posteriorly-situated ulcer into the lesser sac. It is possible for an ulcer thus placed to perforate and for the contents of the stomach to flood the lesser sac, and the fluid pour out through the foramen of Winslow and contaminate the general peritoneal cavity. The exact site of perforation is difficult to find in these cases, and even more difficult to close.

A careful mental note must be made of the precise situation of the perforation, the diameter of the aperture, the characteristics of the ulcer and the surrounding gut wall, and the extent of the peritoneal contamination. These should be recorded in the notes of the case as soon as the operation is completed and while the details are still fresh in the mind of the operator. It is well, too, to make a small diagram of the stomach and duodenum, on which to indicate the site of the rupture. This is very useful for future reference and for statistical purposes.



*Fig. 76.*—PERFORATED GASTRIC ULCER. INTRODUCTION OF THE FIRST STITCH.



*Fig. 77.*—PERFORATED GASTRIC ULCER. INTRODUCTION OF THE SECOND STITCH.

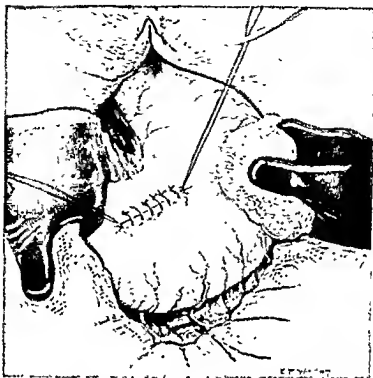


Fig 76.—PERFORATED GASTRIC ULCER. THE PERFORATION HAS BEEN CLOSED BY A SERIES OF CLOSELY-APPLIED INTERRUPTED LEMBERT SUTURES. THIS SUTURE LINE MAY BE FURTHER REINFORCED BY A CONTINUOUS SERO-MUSCULAR SUTURE OR BY AN OMENTAL GRAFT.

*Methods of Closing the Perforation.* The fluid in the immediate vicinity of the stomach is mopped up as far as possible, and the perforated portion of gut is brought well into view, and steadied with the hand ready for suture.

Where possible the perforation should be closed in such a way as to prevent narrowing of the stomach or duodenum, i.e. in the transverse rather than in the longitudinal axis. Where it is found easier and more expeditious to introduce the sutures in the longitudinal axis, however, there should be no hesitation in adopting this method, even though it may produce a certain degree of stenosis.

The suturing is performed with a strong half-curved intestinal needle (atraumatic for preference), carrying a strand of fairly thick 20-day chromic catgut. Wherever possible the sutures are introduced into healthy tissue in the vicinity of the perforation, to prevent cutting out and to ensure firm closure. It is customary to close the rent with a series of closely-placed interrupted Lembert or mattress sutures, as depicted in figures 76, 77, 78, 79, 80, 81, 82, 83 and 84. The first line of sutures may be reinforced by a

series of interrupted Lembert sutures, and further supported by an omental graft or tags of adjacent omentum. A purse-string suture is not recommended for closing the perforation, except perhaps where the perforation is very small and the surrounding cedema and induration are negligible.

It should never be employed for a large perforated callous ulcer, as, on attempting invagination, the tissues will be found to cut out. Again, a purse-string suture will often interfere with the blood supply to the edges of the sutured ulcer.

*Cleansing the Peritoneal Cavity.* Irrigation of the peritoneal cavity has been wisely abandoned, as it prolongs the operation and increases shock. The irrigation may be harmful in disseminating infected fluid and in actually diffusing any localised peritonitis which may be present. I have abandoned systematic mopping out of the abdominal cavity unless food particles are found in the contaminated fluid or have become lodged between the coils of intestine.

During operation an electric suction apparatus may be used by the assistant to remove any large accumulation of fluid, or if this is not

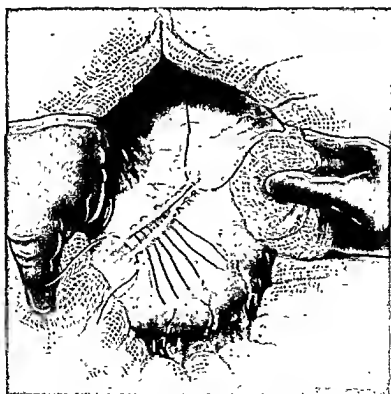


Fig. 70.—PERFORATED GASTRIC ULCER, SHOWING THE METHOD OF CLOSING THE PERFORATION BY A SERIES OF INTERRUPTED MATTRESS SUTURES.



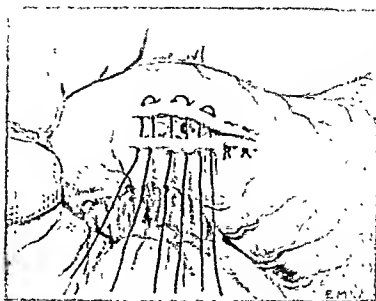


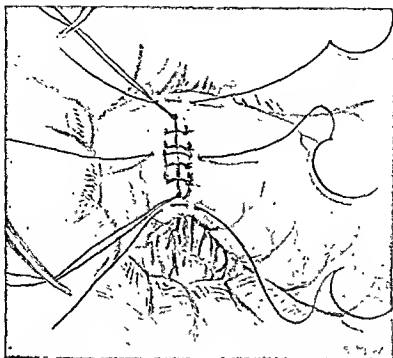
FIG. 80.—PERFORATED DUODENAL ULCER, SHOWING THE METHOD OF CLOSING THE PERFORATION BY A SERIES OF INTERRUPTED MATTRESS SUTURES.

available Cripps pads may be inserted into each flank at the commencement of the operation and removed at intervals as they become saturated.

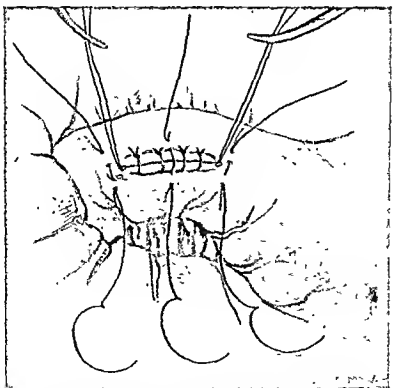
*Drainage.* In cases of doubt it is best to drain the pelvis through a small suprapubic stab wound. When the perforation is



FIG. 81.—PERFORATED DUODENAL ULCER CLOSED BY A SERIES OF INTERRUPTED MATTRESS SUTURES. THE SUTURE LINE IS FURTHER PROTECTED AND INVAGINATED BY DRAWING THE OMENTA TOGETHER IN THE MANNER SHOWN.



*Fig. 82.*—PERFORATED DUODENAL ULCER. THE PERFORATION IS CLOSED BY A SERIES OF INTERRUPTED SUTURES WHICH HAVE BEEN INTRODUCED AT RIGHT ANGLES TO THE LONGITUDINAL AXIS OF THE GUT SO THAT NO NARROWING OF THE LUMEN OF THE GUT RESULTS WHEN THE SUTURES ARE TIED. THIS METHOD IS TO BE PREFERRED WHEREVER CONDITIONS PERMIT OF ITS SAFE PERFORMANCE.



*Fig. 83.*—PERFORATED DUODENAL ULCER CLOSED BY A SERIES OF INTERRUPTED SUTURES.

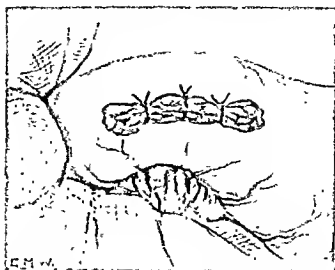


Fig 61—PERFORATED DUODENAL ULCER CLOSED BY INTERRUPTED SUTURES AND THE SUTURE LINE PROTECTED BY AN OMENTAL PAD OR GRAFT WHICH IS STITCHED INTO POSITION.

recent and there is little contamination, drainage will not be necessary. The tube is removed at the end of 24–48 hours. Insertion of a drainage-tube through the lower end of the epigastric incision is not advisable, as it predisposes to infection of the wound and post-operative ventral hernia.

*Closure of the Wound.* The wound is closed in the usual manner with a continuous stitch for the peritoneum, tension sutures, and interrupted sutures for the anterior sheath of the rectus muscle. A few widely-placed interrupted sutures of silkworm-gut will sufficiently approximate the skin edges. A small wisp of glove-rubber or corrugated tubing may be placed at the lower end of the wound to drain the subcutaneous tissues. In desperate cases where the patient appears moribund at the completion of the operation this method of suture is too protracted, and the abdomen should be expeditiously closed with interrupted through-and-through silver-wire tension sutures, which have been found in practice to be very satisfactory in such cases (see page 1232).

### *Suture followed by Primary Gastro-Jejunostomy*

A short-circuit operation may be indicated when, on the completion of the suture of a perforation in the pyloric region or in the duodenum, stenosis appears to be complete.

I have performed suture and primary gastro-jejunostomy on very rare occasions only, when suture of the perforation had produced occlusion of the pylorus or duodenum and when all the conditions present were very favourable. There are, however, a number of surgeons who consider the addition of gastro-jejunostomy to be an essential step which should be omitted only in desperate cases. Their claims for the routine performance of this added procedure are worthy of consideration, not only on the strength of published statistics, but for other reasons which we will now discuss.

The advocates of this method state that gastro-jejunostomy does not increase the mortality in early cases, in spite of the operation being protracted, but may rather reduce it, and that instead of spreading the infection it tends to diminish it.

Gastro-jejunostomy, in addition to overcoming any immediate apparent obstruction produced by the infolding of the ulcer, relieves the tension on the line of suture, and plays an important part in preventing the onset of such complications as recurrence of perforation, pyloric stenosis, and severe hæmorrhage. The short-circuiting, by relieving intra-gastric tension and by increasing the alkaline content of the stomach, assists the process of healing of the ulcer and augments the chances of a permanent cure.

When suture alone is performed they state that the ulcer will fail to heal in a higher percentage of cases, thus prolonging invalidism, demanding protracted courses of medicinal treatment, and frequently secondary operation.

The exponents of suture and primary gastro-jejunostomy assert that post-operative convalescence is generally uninterrupted, smoother and quicker than when simple suture alone is employed, and that serious post-operative complications are less frequent. They consider that the incidence of anastomotic ulcer has been exaggerated and that the late results are much better than those which follow simple closure.

The champions of this operation are numerous and have published statistics of their respective death-rates with the two operations. Here are the figures submitted by four surgeons.

<i>Surgeons.</i>	<i>Death-rate.</i>	
	<i>With suture.</i>	<i>With suture + gastro-jejunostomy.</i>
Deaver and Pfeiffer . . . .	17%	5%
Morrison . . . . .	47%	3%
Platou . . . . .	20%	0%

Not even the most ardent advocates of suture with primary gastro-jejunostomy would, however, advise its performance under the following conditions :

- (a) When there is generalised peritonitis.
- (b) When the patient is taking the anæsthetic badly.
- (c) When the operation presents great technical difficulties, e.g. the patient is fat and there is a perforated gastric ulcer associated with considerable fixation of the stomach posteriorly.
- (d) When the patient is aged or in very poor physical condition.
- (e) When intoxication is advanced.
- (f) When the operator has had but little experience in abdominal surgery.

### *Excision of the Ulcer and Suture (Pyloroplasty)*

In this operation the ulcer is completely excised, the incision being made through adjacent healthy tissue, thus often rendering closure of the wound in the gut comparatively simple. The suture of this healthy tissue is quickly, safely, and easily done, and only takes a few extra minutes. The aperture which remains after excision of the ulcer is closed in such a way that the lumen of the gut is actually increased in size, thus preventing the subsequent formation of stricture at the site of the suture.

The advantages of this operation are therefore apparent for these reasons: the ulcer is excised, rendering hæmorrhage, recurrence of perforation, and the onset of ulcer-cancer most improbable; and by performing a plastic operation subsequent pyloric stenosis cannot occur. As a routine procedure, however, it has certain disadvantages.

- (a) It can only be performed in selected cases.
- (b) Adhesion or fixation of the ulcer to the posterior abdominal wall may make mobilisation difficult, and the operation protracted or even impossible to perform.
- (c) When a large callous ulcer is completely excised with a healthy margin of tissue, the resulting gap may be very great, and approximation of the edges present considerable difficulty. The closure may eventually be possible only by producing a considerable degree of tension on the suture line, which is a subsequent source of anxiety and a possible danger, as a secondary perforation may result from the sutures cutting out, or from a portion of the sutured area becoming gangrenous and sloughing.

(d) A secondary peptic ulcer may develop at the site of the plastic operation and lead to the formation of a stricture, or necessitate further operative interference.

This operation was advocated by Dowden (*Edin. Med. Jl.*, ii, 145, 1909), but has had very few supporters. Its performance may be indicated, however, when the perforation is small and accessible, when the gut wall immediately around is healthy, when plastic closure would seem to present no technical difficulties, and when the condition of the patient is generally satisfactory.

Dowden had no deaths in his series. Dunhar shows that the operation in his hands has given most gratifying immediate and late results. Moynihan records 22 cases with only one death (a mortality of 4.5 per cent). Barger (Stockholm) in a series of 78 cases had a mortality of 11.5 per cent, but lost none of 45 cases which were operated upon within six hours of the occurrence of the perforation. Williams and Walsh (*Lancet*, ccxviii, 9, 1930), however, record the very high operative death-rate of 33 per cent.

### *Partial Gastro-Duodenal Resection*

von Haberer (*Wien. klin. Woch.*, xxxii, 413, 1919) was the first to perform and advocate partial gastro-duodenal resection for perforated peptic ulcer. It appears that this method is gaining in popularity in certain continental clinics, but as far as can be ascertained at the present moment, there are very few English or American surgeons who would perform such an extensive operation for acute perforation when simpler measures of saving the patient's life are available with, moreover, less operative risk.

This operation, as performed by certain experts in gastric surgery in clinics devoted to such work, demands further investigation as it shows a surprisingly low immediate death-rate, and a high percentage of ultimate cures far exceeding the late results obtained by adopting more conservative measures.

The plea made by these continental resectionists for this method is that multiple peptic ulceration is a very much commoner disease on the Continent than it is in England, and that perforated duodenal ulcer is there often associated with chronic gastric ulcer, with multiple erosions of the pyloric segment of the stomach, or with acute ulcerative gastritis and duodenitis. Also that recurrent perforation is frequent after simple closure, and a large majority of cases so treated require subsequent gastrectomy. Again, many cases develop gastro-jejunal

ulceration after short-circuit operations, often necessitating a secondary gastric resection which is exceedingly difficult to perform and which is followed by a high death-rate.

Judine (1933), of Moscow, the most ardent advocate of partial gastrectomy for perforated ulcer, reports that in his last 212 patients 168 were treated by gastrectomy, with only 6 per cent of deaths. Hoche and Marangos (*Arch. f. Klin. Chir. Ap.*, 626, 1932), of Vienna, describe 18 cases treated by partial gastrectomy with a mortality of 22.4 per cent. They collected from published records 405 cases subjected to resection within 12 hours of perforation, and found that only 9 per cent of these patients died. Convincing as these figures are, we feel that most British surgeons are in agreement with Gilmour and Saint (*Br. J. Surg.*, Vol. xx, p. 78, 1932-3) who write on this subject as follows:

"Every surgeon must be impressed by the figures quoted above, since in some series the mortality is less than that resulting from the treatment of the perforated ulcer by the simplest possible method—namely, laparotomy and suture. It is obvious, however, that such results can only be obtained where the operator has more than average ability and experience in gastric surgery, quite apart from the fact that the patient's general condition must be good.

The sphere of partial gastrectomy as the treatment for perforated ulcer must necessarily be always restricted to clinics specializing in gastric surgery. We are quite sure that it would be unwise for the average general surgeon to attempt to establish this large resection operation as his routine treatment for perforated ulcer, and think he would be well advised to content himself with some less formidable procedure. We think our experience of a 0.5 per cent mortality in cases operated upon under twelve hours, with a total mortality of 4.7 per cent, fully justifies the simplest operative measures—that is, suture alone."

### *Temporary Gastrostomy or Duodenostomy*

I have performed temporary gastrostomy in one case of perforated gastric ulcer.

The patient was a man of 40, with a large perforation of the stomach of some 12 hours duration. He was in poor condition and was taking the anaesthetic very badly. Simple suture was attempted, but had to be abandoned as the margins of the ulcer were soft and friable, and the sutures persistently cut out, leading to much trauma and bleeding. An attempt was made to occlude the rupture with an omental graft, but owing to the size of the opening in the stomach and the difficulty experienced in keeping the graft in position, this method proved unsatisfactory and had also to be abandoned.

A rubber tube, large enough to fit snugly into the rent, was introduced through the stomach into the duodenum to serve as a plug and as a temporary gastrostomy. Six weeks later it was possible to re-open the abdomen and perform a partial gastrectomy.

"When perforation of a gastric ulcer has resulted in a hole more than half a centimetre wide, and the surrounding stomach wall is cheesy

with inflammation, it is waste of time to attempt closure by sutures. A rubber catheter passed through the hole and down into the duodenum will serve both as a stopper and as a means of giving unlimited fluids. Lives will be saved in this way which would be lost by conscientious orthodoxy." (Keynes, *St. Bart's. Hosp. Jl.*, Jan. 1934.)

In desperate cases, therefore, when the opening is very large and its margins are œdematous and friable to a degree, such an emergency method can be expeditiously performed with a fair prospect of tiding the patient over the critical stage of his illness, until his condition permits of dealing with the ulcer by a more radical method.

In summing up these considerations we would advocate the operation of simple suture, irrespective of (a) the time factor—the time that has elapsed since perforation occurred; (b) the nature of the rupture—whether large or small, with margins friable or indurated; (c) the site of the perforation—whether gastric, pyloric, or duodenal; and (d) the general condition of the patient—whether good, poor, or bad. Gastro-jejunostomy should be performed only in those cases where closure of the perforation has resulted in complete occlusion of the pylorus or duodenum.

### *Prognosis*

This will depend upon :

(1) *The amount and nature of the fluid in the stomach.* The fuller the stomach is at the moment of perforation, the worse the outlook. The mortality is always higher where large quantities of fluid have escaped from the rent, as this of necessity produces a generalised peritonitis.

(2) *The size of the perforation.* Here again, the larger the perforation the poorer the prognosis. The mortality is 3 to 4 times greater with large perforations.

(3) *The time factor.* The time that has elapsed between the perforation and the operation is the most important single factor in the prognosis, as all published statistics will prove. The following are the figures given by Shawan :



<i>Hours lapsing before operation.</i>	<i>Time Factor</i>			<i>Mortality per cent.</i>
	<i>Recovered.</i>	<i>Died.</i>	<i>Total.</i>	
Under 6 . . . .	92	11	103	10.6
7-12 . . . .	57	11	68	16.1
13-18 . . . .	11	11	22	50.0
19-24 . . . .	7	7	14	50.0
Over 24 . . . .	5	13	18	72.2
Not determined . . . .	0	2	2	100.0

(4) *Position of the ulcer.* The prognosis will depend partly upon the anatomical site of the rupture. It is best with anteriorly-placed duodenal ulcers, and becomes progressively worse with those situated upon the lesser curvature of the stomach, than with those in the pyloric region, whilst with rupture of an ulcer situated on the posterior wall of the stomach or duodenum, and particularly when associated with fixation or penetration of the pancreas or an adjacent viscus, the outlook is very grave.

(5) *The type of operation performed, the skill of the operator, and the choice of anæsthetic.*

### *Post-operative Complications*

In Grey Turner's series of 147 cases, 23 patients developed some serious complication, and this bears out the general statement that following such operations there will, on an average, be complications in one case in every six, those affecting the chest being by far the commonest—pneumonia.

Subphrenic or pelvic abscess may develop in the latter stages of recovery, about the third week. Gastric and duodenal fistula, parotitis, burst abdomen, and phlebitis are rare complications.

Death occurring within the first two days of operation is usually due to post-operative shock; if within seven days, to peritonitis or pneumonia.

### *After-Treatment*

The following is a brief résumé of certain suggestions relating to nursing instructions, diet, and general management, which may be useful in indicating the lines of treatment advised.

During the first 24-48 hours after operation, post-operative shock will have to be combated. Following this, measures will have to be adopted to guard against chest complications, to maintain the heart's action, and to treat any peritonitis which may be present. Dieting and the administration of fluids, both with regard to the quantity

and the quality administered, will receive close attention during the first 4-5 days. A scheme for dieting which assumes that the patient progresses and improves according to a regulated schedule, although useful in supplying the nursing staff with a definite plan of treatment, is unsatisfactory in certain cases where peritonitis and its effects are unduly protracted and severe, or resolve with surprising rapidity.

(a) *Sedatives.* It should be remembered that the patient was probably given some pre-anæsthetic before being taken to the operating theatre. On his return to the ward, therefore, no further injection of morphia or the equivalent will be required until he is fully round from the anæsthetic and complains of pain. It is a good practice, nevertheless, to give aspirin, 30 grs., and potassium bromide, 40 grs., in 6 oz. of saline, per rectum, as soon as the patient has returned to bed. This aspirin-bromide mixture may be repeated in 12-16 hours' time.

During the second 24 hours another injection of morphia may be required, but this drug is not, as a rule, administered again until immediately after the first evacuation of the bowels, which usually takes place on the third or fourth post-operative day. For the first few nights following operation a sleeping draught may be required.

(b) *Toilet of the Mouth.* Patients often complain that their mouths are very dry and sore for the first three or four days following operation, and during this period they are unable to attend to the toilet of the mouth for themselves in a satisfactory manner. The nurse should therefore clean out the mouth as soon as the patient is round from the anæsthetic, and after this six-hourly until light diet is started. The patient should be encouraged to rinse out his mouth frequently with some mild antiseptic lotion. A dry mouth is not only uncomfortable, but is a positive danger as it predisposes to parotitis.

(c) *Fowler's Position.* The value of this position is obvious in cases of perforation. Its assumption should be gradual during the first 12 hours as the patient may be suffering from shock. When fully recovered from the effects of shock he should be placed in the high Fowler position and maintained thus until all evidence of peritonitis is at an end. About the 4th or 5th day, as a rule, he may be changed into the low Fowler position, and he is encouraged to maintain this position until he is fit to get out of bed.

(d) *The Wound.* The dressings are changed after 24 hours, and the wound re-dressed. If a suprapubic drain has been inserted, it is withdrawn in 24-48 hours. It is never allowed to remain *in situ* for more than 48 hours. The skin stitches are removed on the 8th-10th day, and the tension sutures on the 12th-18th day.

If the wound is inflamed or suppurating a stitch may be removed here and there, and hot fomentations be applied three times a day.

If the patient has a severe cough or there is abdominal distension, additional support in the way of strapping should be applied to guard against the possibility of burst abdomen.

(e) *Radiant Heat to the Abdomen.* There is nothing more comforting than the application of radiant heat for short spells, say of 10–15 minutes three times a day. Supervision is essential as the treatment may prove exhausting and even lead to collapse in patients who are very debilitated and toxæmic.

(f) *Charts.* During the first week the temperature, pulse, and respirations are charted four-hourly. Special charts may be required, e.g. a *vomit chart* where there is continued vomiting, the amount and time of each vomit being recorded; a *pulse chart* when the pulse is rapid—over 120 for more than four hours; or a *urine chart*, recording the total amount of urine passed every 24 hours until the patient is out of danger. If the patient has to be catheterised, the amounts withdrawn, the nature of the urine, and the number of occasions on which catheterisation is performed during each 24 hours, are noted.

(g) *Gastric Lavage.* A small Ryle tube should be passed if vomiting is severe or the pulse very rapid. The contents of the stomach are aspirated and irrigation is then performed with normal saline.

The possibility of acute dilatation of the stomach should be borne in mind, and the first indication of this severe complication may be an accelerated pulse-rate. After gastrectomy the pulse-rate is often very rapid for the first 48 hours.

The irrigation of the stomach should be very cautiously performed, successive small amounts being injected and then withdrawn, thereby avoiding any additional tension on the suture line.

(h) *Cardiac Stimulants.* These are often necessary during the first 48 hours following operation.

Coranune, 1 cc. intramuscularly, t.d.s., is given for the first three or four days, while the A.S.A. mixture of Box is useful in reducing the pulse-rate and strengthening the heart's action. This is composed of:

R

Atropine sulph.  $2\frac{1}{10}$  gr.

Strychnine hydrochlor.  $\frac{3}{10}$  gr.

Adrenalin (1 in 1000) 10 minims.

This mixture should be injected six-hourly for the first 48 hours.

Intramuscular injections of ephedrine,  $\frac{1}{4}$  gr., or caffeine sodium benzoate, 2 grs., may be helpful in combating immediate post-operative shock. The intravenous injection of strophantbin,  $\frac{1}{200}$  gr., may be tried when the pulse is thready or imperceptible at the wrist.

(i) *CO<sub>2</sub> Inhalations.* Post-operative CO<sub>2</sub> drills, in which CO<sub>2</sub> (5-10 per cent with oxygen) inhalations are given to the patient for 10 minutes in each hour for several hours during the first or second post-operative day, are useful in guarding against chest complications and in encouraging deep breathing.

(j) *Intravenous Therapy.* One pint of Crooke's gum saline 6 per cent is given intravenously at the completion of the operation as an important routine method in all cases. Continuous intravenous medication with salines and glucose by the slow drip method has proved invaluable, but in the presence of serious pulmonary or cardiac complications the intravenous administration of large quantities of fluids is harmful.

(k) *Rectal Salines.* During the first 24 to 36 hours distilled water, tap water, normal saline, or saline with 5 per cent glucose, is introduced into the rectum by the continuous Murphy drip method. Subcutaneous salines are, as a rule, not given, as they tend to distress the patient.

(l) *Diet. First day:* After 12 hours two drachms of warm water are given on one or two occasions, but nothing else is administered by the mouth. During this time the mouth is frequently washed out, and fluids are supplied by the continuous drip method, by intermittent intravenous injections, or by rectal infusions of saline.

*Second day:* Warm sterile water by the mouth, 1 oz., gradually increasing up to 2 oz., hourly. Rectal saline, 6 oz., 6-hourly.

*Third day:* A glycerine enema is given first thing in the morning, and if there is no vomiting, paraffin,  $\frac{1}{2}$  oz., b.d. Warm sterile water by the mouth, 2 oz., gradually increasing up to 3 oz., hourly, alternating with 2-3 oz. feeds of citrated milk or half-strength Glaxo. Rectal saline, 6 oz. 8-hourly, where possible.

*Fourth day:* A soap or turpentine enema is given in the morning. If the bowels are thoroughly evacuated after the administration of this enema, an injection of morphia,  $\frac{1}{6}$  gr., may be required for the relief of discomfort. Warm sterile water, 3-4 oz. 3-hourly, alternating with citrated milk or diluted Glaxo, Benger's, or Allenbury's, 3-4 oz.

*Fifth day:* At this stage the diet may be considerably increased if the patient is making satisfactory post-operative progress and signs of peritonitis have subsided. If, however, any increase in the feeds is associated with nausea, vomiting, flatulence, distension, or general gastro-intestinal upset, the patient must be given only small frequent feeds of water, albumen water, or citrated milk by the mouth, the amounts depending upon his condition.

If all goes well, however, the patient is at this stage put on Lenhart's diet (see page 388).

After the *sixth* post-operative day it is advisable to consult a physician with regard to further steps in dieting and general medicinal treatment. Before the patient is discharged he is given a copy of Hurst's post-ulcer regime (see page 576) for his future guidance.

(m) *Alkaline and Tonic Treatment.* During convalescence the usual medicines employed in the medical treatment of peptic ulcer are given, in addition to intramuscular injections of iron and arsenic which act as a general tonic and hæmatinic.

#### SUB-ACUTE PERFORATED PEPTIC ULCER

Sub-acute perforation occurs only in connection with chronic peptic ulcers. In such cases a history of chronic gastric or duodenal ulcer is always obtained, and just before perforation occurs there is an acute exacerbation of the symptoms. The onset of perforation itself is sudden, but the peritonitis which results is strictly localised owing to the small quantity of fluid which escapes from the rent in the stomach or duodenum.

The initial signs and symptoms of a sub-acute perforation are identical with those of acute perforation—sudden epigastric pain, abdominal rigidity and tenderness, and prostration—but they are always less intense in nature. The abdominal rigidity is more marked over the region of the extravasation; elsewhere guarding may be absent, or, if present, it yields on pressure. There is no tenderness of the pelvic peritoneum on rectal examination.

On examination it may be impossible to make a differential diagnosis between sub-acute perforation ("leaking peptic ulcer") and acute perforation, nor is this distinction often a matter of great import, as operation must be performed in such cases whenever the diagnosis is in doubt. In certain cases, however, on the strength of the history,

the sequence of the symptoms, and the localisation of the signs, such a differentiation can readily be made.

If operative interference is withheld and medical treatment is undertaken, one of two results may be expected: (a) the abdominal tenderness and rigidity may gradually disappear and the ulcer heal; or (b) a localised abscess—perigastrio or subphrenic—may form, which may rupture and infect the general peritoneal cavity, producing a septic peritonitis.

Expectant treatment may be justifiable under certain conditions. For instance, in the early acute case, where the diagnosis is not in doubt, and where the patient is a poor operative risk, it is wiser to adopt such methods of treatment, provided that all facilities are at hand for immediate operation should this prove to be necessary.

Again, if the patient is seen for the first time many days after perforation has occurred, and there is sufficient evidence that the infective process is definitely limited or that a localised abscess has formed, more is to be gained by waiting than by performing an injudicious exploration for a condition which is likely to resolve. When operation is performed at a later date in such cases, owing to the absorption of inflammatory products and a possible reduction in the size of the ulcer, operative measures for the cure of the condition are rendered easier and more satisfactory. On the other hand, if the perigastric abscess does not resolve, expectant treatment will ensure its being securely shut off when drainage is undertaken.

It should be emphasized that if the diagnosis is uncertain, or if the patient shows no signs of improvement after a few hours of expectant treatment, operation should be undertaken without further delay.

#### ADHESIONS

Perigastric adhesions may be due to intrinsic or extrinsic conditions. The intrinsic conditions are secondary to disease of or injury to the stomach or duodenum. Chronic peptic ulcer is the commonest cause, and particularly where a chronic perforation has occurred or the ulcer has become attached to internal structures, a possible result of localised peritonitis. Following operations for perforated gastric or duodenal ulcer, subphrenic abscess, or injury to the stomach, adhesions often form and may produce deformity of the stomach or interfere with its motor functions. Perigastric adhesions may be crippling after any operation upon the stomach or gall-bladder. For instance, in the

operation of anterior gastro-jejunostomy, owing to the close proximity of the line of anastomosis to the abdominal wall, adhesions may result, particularly where there has been much operative trauma or soiling of the parts, or where the anterior sero-muscular suture has been applied without sufficient exactness. Again, after cholecystectomy or operations upon the common duct, if the raw surfaces which result from these operations are inadequately covered, the pylorus may adhere to them with resultant mechanical defects which, in time, will surely lead to troublesome symptoms. Perigastric adhesions may also result from tuberculous peritonitis, or be a post-operative complication of general septic peritonitis. A number of cases can be directly attributed to carelessness or faults in operative technique which may include drying and chilling of the intestines through long exposure; crude, unsystematic, and rough packing off of the abdominal viscera with dry swabs and packs; prolonged application of clamps; introduction—intentional or unintentional—of chemical fluids into the abdominal cavity; soiling of the intestines with blood from the abdominal wound or from anastomotic openings; and rough handling of the viscera.

The stomach may become adherent to the abdominal wound, and especially where the edges of the peritoneum have been inadequately approximated, leaving raw edges turned inwards, or big unsupported gaps in the suture line. Wound sepsis, or burst abdomen necessitating re-suture, favours the production of intra-abdominal adhesions which may drag upon or compress the stomach. After operation the great omentum may become adherent to any raw surface which may result from the excision of a Fallopian tube, the cauterisation of a fibroid, or from an incompletely peritonised or oozing cervical stump which remains after the performance of a sub-total hysterectomy. Owing to its fixation in the pelvis the great omentum exerts a constant downward pull on the stomach, which interferes with gastric peristalsis and produces dyspeptic symptoms. There may be very marked perigastric adhesions without any symptoms, but when symptoms do occur they are nearly always due to a reflex pylorospasm or to mechanical obstruction. Pain, when present, is due to increased gastric tension, and is worse when the pylorus is partly obstructed. Attacks of pain occurring after meals are brief, sometimes lasting only a few minutes. The pain is increased by exercise or by any undue exertion, such as excessive stretching. There may be some deep tenderness and even muscular rigidity over the region occupied by the adhesions.

A diagnosis is always beset with difficulty, and reliance will have to

be placed chiefly upon a history of a previous abdominal operation or any condition which might have produced localised or generalised peritonitis. An X-ray examination of the gastro-intestinal tract may afford valuable corroborative evidence. Exploratory operation should not be undertaken lightly, and is not to be recommended unless there is very strong evidence of the presence of crippling bands and adhesions, or of partial obstruction.

The operation may be a simple affair, requiring only the severance and removal of a few adhesions. When, however, the stomach is widely attached to the abdominal wall, or the upper abdominal viscera are matted together, it may be an undertaking of great magnitude, entailing not only the freeing and re-position of the viscera, and guarding against subsequent adhesions by careful peritonisation of raw surfaces, but also dealing as may seem best under the circumstances with any associated pathological lesion in the stomach, duodenum, or gall-bladder.

#### FISTULA

There are two varieties of gastric and duodenal fistula—external or cutaneous, and internal.

(i) *Gastro-cutaneous Fistula*. This is a condition in which the interior of the stomach communicates by a fistulous tract with the external surface of the body.

#### *Causes:*

(1) Traumatic.

(a) Direct injury to the stomach, e.g. gunshot wounds, stabs, etc.

(b) A foreign body which has perforated the stomach and produced a localised abscess. When this abscess is opened at operation or bursts spontaneously through the abdominal wall, a fistula results.

(c) Gastric operations, and especially operations for perforated gastric ulcer and perigastric abscess.

A gastro-cutaneous fistula, however, may develop after any operation upon the stomach, such as gastrectomy or gastro-jejunostomy, and is due in these cases to faulty suturing.

(2) Pathological.

(a) *Primary*. The two commonest primary pathological lesions in the stomach which are responsible for the majority of gastro-cutaneous fistulae are chronic gastric ulcer and malignant disease.



(b) *Secondary.* In these cases the fistula results from diseases of neighbouring organs, such as the colon, the gall-bladder, or the liver.

The pathological conditions, whether they be primary or secondary, are more often responsible for the internal variety of fistula than for the external.

Fistulae may be *direct* or *indirect* in formation. They are *direct* when the floor of an ulcer or of a growth becomes adherent to the abdominal wall or to a neighbouring viscus and perforates it. Here the fistulous tract is very short, and the aperture between the two viscera may be, and usually is, large. The *indirect* type originates through the intervention of a localised abscess. The apertures are usually small, and the tract communicating between the two viscera is long, attenuated, vicarious, and tortuous.

The *signs and symptoms* in an external gastric fistula are due to (a) the primary pathological condition in the stomach; (b) the fistula itself; and (c) the loss of gastric juice and nourishment. A carefully-taken history will often yield very valuable information suggesting the underlying cause. For instance, there may be a history of trauma, or of a former operation upon the stomach. Whereas in some cases there may be a complete absence of all symptoms, and the patient may appear to be in sound health without experiencing any inconvenience from the presence of the fistula, on the other hand there may be extreme thirst, a constant sensation of hunger, and a tightness or burning pain in the epigastrium which is aggravated by stretching movements. Wasting may be progressive in spite of an eager appetite, the urine scanty, and the bowels very constipated. The fistulous opening will be apparent, and its position should be noted as this will suggest the portion of the stomach involved. It is frequently situated in the left side of the epigastrium, just above the umbilicus, or if there has been a recent abdominal wound the discharging sinus will be found somewhere in the line of incision, usually at the lower end. The skin in the immediate vicinity of the fistulous opening will be red, excoriated, and inflamed for a variable area round it, and where the discharge has not been efficiently controlled by frequent dressings and constant attention to the wound it will sear red lines where it has trickled away from the saturated dressings and coursed over the abdominal wall. The discharge is usually highly acid in reaction, odourless, and opalescent, and may be mixed with digested or undigested food, particles of which may be recognised as having been but recently ingested. If a coloured draught is administered, the length of time which elapses before this is discharged through the opening may afford

some indication as to whether the fistula is connected with the stomach, duodenum, or with some other portion of the intestinal tract. In gastro-cutaneous fistula the discharge follows almost instantaneously. The presence or absence of bile and the nature of the digestive ferments present in the fluid collected from the fistula will help to determine its source, as will also a barium meal examination to a much more accurate degree.

*Treatment.* This may be :

(1) *Expectant.* The history of the case, the length of time that the fistula has existed, and the general condition of the patient will determine whether it is better to adopt expectant methods of treatment or to resort to operation. Where the fistula follows a primary operation upon the stomach, has been present for a short time only and shows signs of spontaneous healing, where there is no pyloric obstruction, and the loss of gastric juice does not in any way interfere with the patient's general health, digestion, or nutrition, operation may be deferred.

(2) A preliminary jejunostomy may be called for in some cases of fistula due to cancer of the stomach for the purpose of supplying the patient with nourishment, and also prior to excision of the fistula in non-malignant cases associated with marked emaciation.

(3) *Excision of the fistula.*

*Operation.* The operation for excision of a gastric fistula may be difficult on account of the fixation of the stomach to the abdominal wall in the immediate vicinity of the external opening, and also on account of extensive perigastric adhesions. The abdominal incision should be ample and planned with considerable care (fig. 85). A superficial oval-shaped incision is first traced out on the stomach wall, widely encircling the affected segment. Then, starting at the upper point of this oval, the incision is carried down on either side,

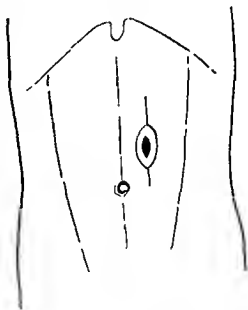


Fig 85.—OPERATION FOR GASTRO-CUTANEOUS FISTULA—THE INCISION.

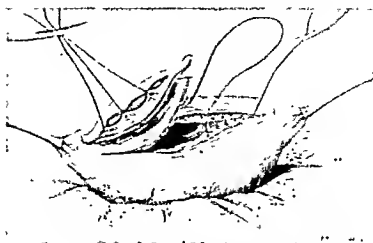


Fig. 86.—EXCISION OF A GASTRO-CUTANEOUS FISTULA. THE MARGINS OF THE MUCOUS MEMBRANE OF THE STOMACH ARE BEING APPROXIMATED WITH A CONTINUOUS SUTURE.

little by little, through all the layers of the stomach. Meanwhile, in order to prevent leakage and contamination with gastric contents, the edges of the mucosa are picked up and approximated as they are cut, so that when the lower end of the oval is reached only a minute gap in the mucosa remains to be closed (fig. 86). The edges of the sero-muscular layers of the wound are united by a continuous stitch, and the suture line is buried by Cushing's method, as shown in figure 87. The abdominal wound is then closed.

(a) *Internal Gastric Fistula.* There are many varieties of internal gastric fistula. As a result of injury—usually operative—or disease

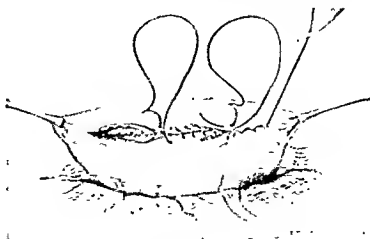


Fig. 87.—FISTULA OF A GASTRO-CUTANEOUS FISTULA SHOWING THE THREE-TYE SUTURE METHOD OF SECURELY CLOSING THE OPENING IN THE STOMACH.

which may be primary in the stomach or secondary in a neighbouring viscus, the stomach may communicate with the gall-bladder, the duodenum, the jejunum, the transverse colon, the pleural cavity, the pericardium, the mediastinum, or even the heart. As in the external variety, these fistulæ are either *direct* or *indirect*.

*Gastro-colic fistula* is by far the commonest internal variety.

#### *Causes :*

- (1) Cancer of the stomach.
- (2) Cancer of the transverse colon.
- (3) Anastomotic ulcer.
- (4) Tuberculous disease of the colon.
- (5) Perigastric or subphrenic abscess, due either to disease of the stomach or of a neighbouring viscus.

The most frequent cause of gastro-colic fistula is cancer of the stomach, and the commonest site of the opening in the stomach is the region of the greater curvature, opposite the incisura.

*Symptoms.* In the *indirect* variety of gastro-colic fistula, owing to the long narrow tortuous tract which exists between the two viscera, there may be very few symptoms. In the *direct* variety, however, true fecal vomiting occurs. The vomited material often resembles the watery stools that are so frequently passed. Diarrhœa is severe, and particles of recently-ingested food can often be identified in the stools. The diarrhœa may be so acute and distressing that it may be the only symptom complained of by the patient. There may be epigastric pain, gastric flatulence, and evil-smelling breath, loss of weight being progressive owing to the short-circuiting of food into the colon.

*Diagnosis.* The history and clinical findings will suggest the diagnosis, confirmation of which will be obtained by means of skiagrams.

*Treatment.* The treatment of peptic gastro-colic fistula is discussed on page 412. When the fistula is due to a primary cancer of the stomach or colon, exploratory laparotomy will decide whether a combined resection of the stomach and colon is feasible. If radical measures are impossible it is very doubtful if any palliative procedure will afford even temporary relief.

(iii) *External Duodenal Fistula.* This may follow operations upon :

*The gall-bladder and bile-ducts.* In cases of old-standing cholecystitis where there are extensive adhesions, and especially where the duodenum is firmly adherent to the gall-bladder, the process of separating these two organs prior to exposure and isolation of the biliary ducts may result in a minute puncture or laceration of the duodenal wall which may remain undetected at the time. Such a mishap is particularly prone to occur where a small traction diverticulum of the duodenum is hidden within a sheaf of dense adhesions. Again, where cholecystectomy and supra-duodenal choledochotomy have been performed, requiring the freeing of the duodenum from a mesh of adhesions and necessitating much handling and possibly bruising of the gut, the added pressure of a drainage-tube upon the traumatised area may produce pressure necrosis and sloughing, with the consequent formation of an external duodenal fistula. After operations upon the gall-bladder and common bile-duct it is most important, therefore, to protect the duodenum with omentum or adjacent peritoneum.

*The duodenum.* An external duodenal fistula is most frequently seen after operations for closure of a perforated duodenal ulcer, especially of a retroperitoneal perforation, and after incision for drainage of a subphrenic abscess consequent upon a leaking duodenal ulcer. It has been known to occur after the operations of trans-duodenal choledochotomy, and pyloroplasty for duodenal ulcer, particularly where there has been considerable tension on the suture line or where the gut wall is friable from inflammation. I have seen this complication also after partial gastrectomy, leakage occurring from the duodenal stump which had been closed with fine catgut. The perforation of a duodenal diverticulum and traumatic rupture of the duodenum account for a few cases, as does also the operation of excision of diverticula situated in the second and third parts of the duodenum.

*The kidney.* A portion of the duodenum lies over the upper part of the hilum of the right kidney, and is liable to injury during the performance of a difficult nephrectomy. It is conceivable that if the kidney pedicle is not carefully isolated a small portion of the duodenal wall may be picked up in the clamp and become crushed or else be ligatured to the vascular pedicle. The first intimation of this grave error may be the sudden flow through the incision of bile-stained,

foamy duodenal contents. This ominous sign may appear suddenly even after the lapse of several days and at a time when the patient seems to be making a satisfactory recovery from his operation. The wound will gradually become red and tender, gape, and eventually break down completely through the action of the digestive ferments and irritating chyme. Duodenal juice will flood the right kidney fossa and pour over the wound, excoriating the skin around. The patient's sufferings will be intense, and rapid and progressive emaciation follow.

An external duodenal fistula is always a very serious complication. The emaciation which ensues is due mainly to the loss of fluids, salts, nourishment and of the alkaline values in the pancreatic juice, in addition to the absorption of toxic digestive fluids.

*Diagnosis.* This is rarely, if ever, in doubt. The history of the case, the position of the external fistulous opening, and the character of the discharge, are often sufficient of themselves to indicate the condition present. In difficult and doubtful cases an X-ray examination will often supply a solution.

*Expectant Treatment.* This will include :

(a) Protection of the skin. The most frequently prescribed salve for protection of the wound in the region of the fistulous opening is a mixture of equal parts of zinc oxide and castor oil. An ointment containing one of the heavy metals is also very useful. I have found 30 per cent calomel in lanoline an effective application. Powdered animal charcoal or beef peptone have also been recommended.

(b) Plugging the external orifice with vaseline gauze, strapping the wound, and other methods which aim at obstruction of the flow of the irritating chyme, are occasionally employed with most unsatisfactory results.

(c) Restriction of food by the mouth. The patient should avoid taking liquid foods and drinks, and all oral feeding should be restricted to a minimum. Only dry foods are permitted by mouth. Fluid and nourishment will have to be administered rectally by the continuous Murphy drip method.

(d) The introduction into the fistula of a small rubber tube which is attached to a suction apparatus will keep the area dry and aid healing. If a jejunostomy has been performed the fluid collected by the suction apparatus should be introduced at intervals through the jejunostomy tube.

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(d) The introduction into the fistula of a small rubber tube which is attached to a *suction apparatus* will keep the area dry and aid healing. If a jejunostomy has been performed the fluid collected by the suction apparatus should be introduced at intervals through the jejunostomy tube.



(e) Feeding through an indwelling Einhorn tube. This tube may be introduced through the nose or mouth, and allowed to pass through the stomach and duodenum into the gut beyond the site of the fistula. If doubt exists as to the tube being in a satisfactory position beyond the opening into the duodenum, X-ray screening will supply the required information. When the tube is judged to be in the lower reaches of the duodenum or upper coils of the jejunum, food can be introduced into a highly absorptive portion of the intestinal tract, well below the site of the duodenal fistula.

*Operative Treatment.* This will consist of either :

- (a) Closure of the fistula by suture.
- (b) A short-circuit operation, whereby the gastric contents are diverted from the fistulous opening.
- (c) A combination of (a) and (b) ; or
- (d) Jejunostomy.

*Simple closure.* This operation is doomed to failure in the majority of cases as adhesions from local peritonitis are extensive, and the involved segment of gut is soft and friable to a marked degree. In some instances, however, it may be possible to expose the duodenum and close the opening with a three-tier suture. This operation is undertaken in certain cases where duodenal fistula has developed after gastrectomy. The stump of the duodenum is here exposed, mobilised as far as safety will permit, and the opening closed in the manner described, and reinforced with an omental graft or a portion of the ligamentum teres.

*Posterior gastro-jejunostomy with pyloric occlusion.* After this operation the suction apparatus is employed to keep the fistulous tract as dry as possible.

*The operation of choice*—and this should be done where possible—entails closure of the fistula, protection of the suture line with an omental pad, pyloric occlusion, and gastro-jejunostomy ; but this is an operation of considerable magnitude in a patient who is emaciated, toxic, and feeble to an extreme degree, and it can only be recommended when the patient's general condition safely permits of its performance.

*Jejunostomy.* This should be done after Witzel's method under local anaesthesia. It should be advised when conservative measures

fail, and when simple closure or a short-circuit operation is deemed too hazardous.

Conservative and operative measures are successful in a number of cases, the former having a lower death-rate than the latter, as shown by Kittelson; but the mortality is very high where the fistulous opening is large, and where there is a rapid loss of weight.

KITTELSON (*Surg., Gynec., and Obstetrics*, Vol. 56, p 1056, 1933), in an analysis of 96 cases of external duodenal fistula, showed that in 30 cases the fistulae followed operations upon the gall-bladder and bile-ducts, in 22 cases operations for perforated duodenal ulcer, in 8 cases after nephrectomy, in 8 cases after resections of the stomach for carcinoma, in 7 cases after operation for acute appendicitis with obstruction, in 6 cases after rupture of the duodenum, and in 7 cases from other causes. Sixty-five patients were treated conservatively with a mortality of 27.7 per cent, and 30 patients were treated surgically with a mortality of 50 per cent. The mortality for the whole group, including all types of treatment, was 35.8 per cent.

(iv) *Internal Duodenal Fistula.* This condition is comparatively rare. The fistulous communication may occur as the result of an operation, trauma, or disease between the duodenum on the one hand, and the stomach, colon, small intestine, common bile-duct, or gall-bladder on the other. The communicating channel may be direct when the opening is large, or indirect when the communication between the two is narrow. Probably the commonest type of internal duodenal



Fig. 68.—CHOLECYSTO DUODENAL FISTULA FOLLOWING THE ULCERATION OF A GALL-STONE INTO THE DUODENUM. BARIUM IS PASSING FROM THE SUPERIOR ASPECT OF THE DUODENAL CAP INTO THE COMMON BILE AND HEPATIC DUCTS. (*H. Cecil Bull.*)

fistula seen is that which occurs between the duodenum and the gall-bladder or the duodenum and the biliary ducts, as a result of gall-stones (fig. 88).

### HOURL-GLASS STOMACH

In hour-glass contraction the stomach is divided off into two—and, very rarely three, or more—compartments. The stricture or strictures which are responsible for this sub-division of the stomach may occur at any point between the cardiac and pyloric orifices.

#### *Ætiology and Pathology*

##### *Causes.*

(1) Congenital.

(2) Acquired.

(a) *Intrinsic causes.* These are due to organic disease of the stomach.

(i) Chronic gastric ulcer—especially the saddle-shaped ulcer.  
This accounts for fully 90 per cent of cases.

(ii) Cancer of the stomach—1 per cent of cases.

(iii) Syphilis of the stomach.

(iv) Large gastro-jejunal ulcer.

(v) Stenosis resulting from corrosive poisoning.

(b) *Extrinsic causes.*

(i) Perigastric adhesions.

(ii) Hour-glass stomach due to pressure of the colon when distended with gas.

(iii) Spasmodic hour-glass stomach due to reflex causes, e.g. chronic appendicitis.

The following details only concern hour-glass stomach when produced by chronic gastric ulcer.

*Sex.* About 90 per cent of cases occur in females. Sherren found that of 96 cases of hour-glass stomach operated on by him, 88 were women.

*Age.* In most cases there is a long history of dyspepsia, often dating back many years. The condition is most commonly seen

between the ages of 50 and 70, showing a maximum incidence at about 60. Although occasionally cases have been reported under the age of 20, the disease rarely occurs in patients under 30.

*Incidence.* This complication occurs in 6–10 per cent of all chronic gastric ulcer cases. Walton, Hurst, and Stewart place the incidence at 9·8 per cent. In 173 cases of chronic gastric ulcer investigated by Stewart, there were 17 cases of hour-glass stomach. Moynihan's figures are very similar to these, being 7·8 per cent of the gastric ulcers operated upon by him, whilst the Mayo Clinic estimate of the incidence is 6 per cent.

*Pathology.* The development of hour-glass stomach depends upon the *size*, the *form*, the *position*, and the *chronicity* of the ulcer. The

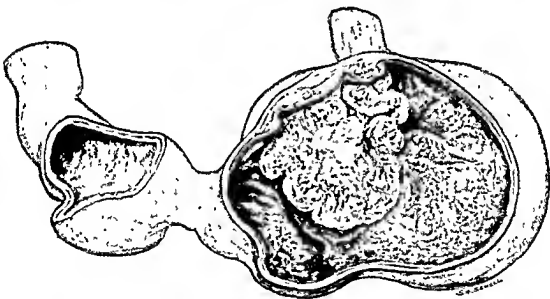


Fig. 89.—SIMPLE HOUR GLASS STOMACH WITH A CARCINOMATOUS GROWTH IN THE PROXIMAL POUCH.  
(Museum, Royal College of Surgeons.)

so-called saddle-shaped ulcer (see fig. 62) is often a precursor. The constriction is nearly always single, and is situated nearer to the pyloric than the cardiac end of the stomach (fig. 89). The cardiac pouch, therefore, will often be found to be larger than the pyloric pouch. In cases of pyloric obstruction, which is stated to co-exist in 25 per cent of cases of hour-glass stomach, the distal pouch, i.e. pyloric, may be enormously distended, and the proximal pouch be small and insignificant. It should therefore be emphasised that in every operation upon the stomach the whole organ, from the œsophageal opening to the

duodenum, should be very carefully palpated and examined, so that the possibility of the presence of a small proximal pouch may not be overlooked and a purposeless gastro-jejunostomy, or other drainage operation, be performed upon the distal pouch.

The constriction may take years to develop when situated in the body of the stomach, the greater curvature being gradually drawn towards the lesser as the ulcer heals and the fibrous scar contracts.

In a long-standing case, and particularly where the ulcer is active, it will be found that the stomach undergoes considerable rotation on its transverse axis. The lesser curvature becomes shortened and is dragged into a posterior position, while the greater curvature is displaced upwards and lies in a more anterior position. It has been computed that in approximately 40 per cent of cases there is no ulcer crater; in other words, the chronic ulcerative process is spent, and healing has occurred with the production of much fibrous tissue and the formation of an hour-glass constriction. The channel connecting the two loculi may be very minute, admitting only the smallest probe, or large enough to admit one or two fingers.

The position of the stricture and the diameter of the isthmus will determine the severity of the symptoms in the majority of cases.

Hypertrophy of the proximal pouch will occur as a result of the stenosis, but it is usually not marked except in the more acute types of obstruction. In due course this pouch will dilate, increase considerably in size, and may sag over the isthmus and lower segment (figs. 90 and 91). Perforation, hæmorrhage, and ulcer-cancer are very rare complications of a chronic gastric ulcer which has produced an hour-glass constriction.

*Types of Hour-glass Stomach.* The various types of hour-glass stomach are depicted in figure 92.

### *Signs and Symptoms*

*History.* The past history of a patient with hour-glass stomach will at once suggest that there has been a chronic gastric ulcer present for many years. At a certain period in this history the symptoms become changed as the result of the obstruction which supervenes. The early symptoms are marked by periods of indigestion alternating with periods of complete freedom. At a later date the periods of indigestion are more prolonged and the periods of freedom are shorter, until eventually the symptoms recur every day and after every meal without



*Fig. 90.—GASTRIC ULCER ON THE LESSER CURVATURE OF THE STOMACH SHOWING SPASTIC INCISURA WHICH POINTS TO THE ULCER CRATER (H. Cecil Bull.)*



*Fig. 91.—Hour glass stomach. AT OPERATION A LARGE SADDLE SHAPED ULCER WAS FOUND. (C. W. McKenneg.)*

any intermissions. If, therefore, a patient gives a past history of gastric ulcer in which the typical periodicity is lost and the attacks become increasingly longer until they are continuous, it is probable that an hour-glass contraction or cancer of the stomach is present.

*Pain.* At the onset the pain arises at 1-2 hours after meals, but as the condition progresses and obstruction supervenes it will be felt immediately after the ingestion of food. In an early case the taking of food may afford some relief, but later the pain becomes more or less constant and is aggravated by food. It is relieved, however, by vomiting, and to a less extent by the administration of alkalis. There is radiation of pain over the left side of the chest to the spine, or to the left shoulder if the ulcer is perforating the pancreas. In a patient with a history of gastric ulcer this radiation of pain to the left shoulder is typical of fixation of the ulcer to and penetration of the pancreas, and is a symptom of the very greatest significance.

On abdominal examination there may be some tenderness in the middle of the epigastrium or to the left of this over the left rectus muscle. The upper part of this muscle may be on guard, and on deep palpation the visceral tenderness may be acute, and a tumour may even be felt.

*Vomiting.* This depends upon the position of the stricture and the size of the isthmus. The nearer the stricture is to the cardiac orifice the worse the vomiting will be; in fact, in some of these cases a diagnosis of cancer of the œsophagus is made as there is also pronounced emaciation, dysphagia, and even regurgitation of food.

In a *typical* case, however, vomiting is absent in the early stage of the disease, but becomes increasingly frequent as the condition advances and the stricture progresses. Here large quantities of gastric contents are usually voided at each vomit, and undigested food or the remnants of meals taken 12-48 hours previously may be recognised.

*Loss of weight.* Owing to vomiting, loss of appetite, and the inevitable pain that follows the ingestion of food, loss of weight is often marked. These patients are thin, emaciated, feeble subjects, and it is not surprising that a diagnosis of cancer of the stomach is often provisionally made before they are submitted to X-ray examination. In very thin patients visible peristalsis may occasionally be seen in the cardiac pouch.

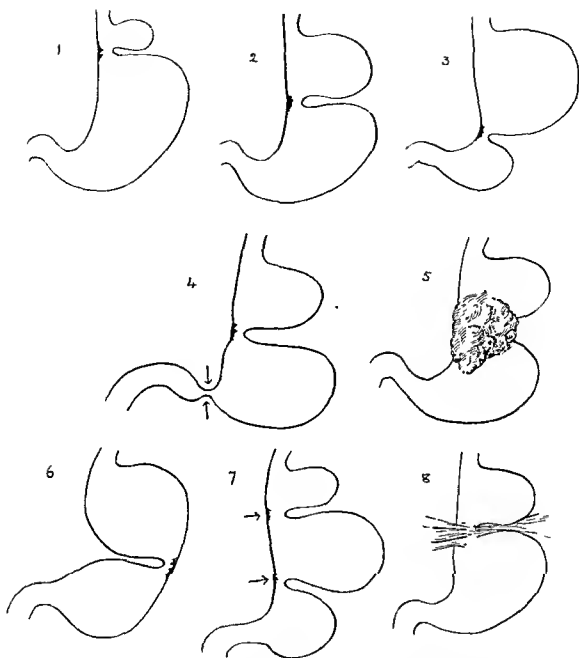


Fig. 92.—TYPES OF HOUR-GLASS STOMACH. NOS. 1, 2, 3, 4 AND 7 SHOW THE TYPES OF HOUR GLASS STOMACH WHICH ARE PRODUCED BY ULCERS SITUATED UPON THE LESSER CURVATURE OF THE STOMACH. THE SIZES OF THE PROXIMAL AND DISTAL POUCHES ARE DEPENDENT UPON THE SITUATION OF THE ULCER.

NO. 4 SHOWS AN HOUR GLASS STOMACH COMPLICATED BY PYLORIC STENOSIS.

NO. 5 SHOWS AN HOUR GLASS CONSTRICTION DUE TO A CANCER OF THE BODY OF THE STOMACH.

NO. 6 DEPICTS A RARE TYPE OF HOUR-GLASS STOMACH IN WHICH AN ULCER SITUATED ON THE GREATER CURVATURE HAS LED TO A GREAT DEAL OF FIBROSIS AND THE DRAWING OF THE LESSER CURVATURE TOWARDS THE ULCER. THE MAJORITY OF ULCERS IN THIS POSITION ARE MALIGNANT.

NO. 7 IS A TRILOCULAR STOMACH DUE TO THE OCCURRENCE OF TWO CHRONIC ULCERS ON THE LESSER CURVATURE.

NO. 8 SHOWS AN HOUR-GLASS STRICTURE OF THE STOMACH PRODUCED BY ADHESIONS OR PERITONEAL BANDS.



*Appetite.* The appetite at first may be good, but owing to the fear that pain will follow the ingestion of food the patient will refrain from eating. At a later stage there is true anorexia, due to a secondary gastritis which develops in the distended proximal pouch.

### *Treatment*

*Medical.* In certain *rare* cases where the ulcer has healed, where the patient is not losing weight, and particularly where vomiting is an infrequent symptom and can be controlled by gastric lavage, dieting and medicine, and where the isthmus is large enough to permit the passage of food, medical treatment may be persevered with for an indefinite period. In the *majority* of cases, however, surgical treatment is clearly indicated after a preliminary course of medical treatment.

*Surgical.* As most of these cases show considerable emaciation and as they are feeble from constant pain and incessant vomiting, a short course of pre-operative treatment is advantageous in order to render them as fit as possible for surgical measures.

The *pre-operative regime* will include :

(1) *The administration of fluids and assimilable foods :*

(a) *By the mouth.* Small frequent sips of water, glucose, whey, albumen water, etc.

(b) *Intravenously* by the continuous drip method, by which glucose and salts are introduced into the circulation.

(c) *Per rectum.* The Murphy drip method is employed, and tap water, saline, or saline with glucose are introduced.

(2) *Gastric lavage.* The stomach should be washed out at least twice a day with a Ryle or other small stomach tube. After aspirating all the contents of the stomach the cavity of the viscus is irrigated with a few oz. of a solution of one drachm of hyd. perox. to one pint of water. This will aid in the removal of mucus and diminish any gastritis that may be present. A few days before the operation normal saline is substituted for the hyd. perox. solution. The contents of the stomach should in all cases be thoroughly aspirated 1-2 hours before the patient is removed to the operating theatre.

(3) *The bowels* should be evacuated by means of enemata, and paraffin,  $\frac{1}{2}$  oz. should be given twice a day by mouth.

(4) *Blood-transfusion* may be beneficial.

(5) An acid tonic given three times a day by mouth may be helpful.

### *Operations for Hour-glass Stomach*

A large number of operations have been undertaken for this condition; many are ingenious, but the majority, although often

affording temporary relief, are very unsatisfactory so far as final results are concerned.

*Operations.*

(1) Gastro-jejunostomy.

(a) Single } anterior or posterior.  
(b) Double }

(2) Gastro-gastrostomy (gastro-anastomosis).

(3) Gastroplasty.

(a) The Heineke-Mikulicz method.

(b) Kammcrer's operation.

(4) Sleeve resection.

(5) Walton's operation (V-excision of ulcer, posterior transverse gastro-jejunostomy, and pyloric occlusion).

(6) Partial gastrectomy.

*Gastro-jejunostomy*

(a) *Single gastro-jejunostomy* (fig. 93). This operation is now very rarely performed for hour-glass stomach. It was recommended in those cases where the stricture was situated in the pyloric portion of the stomach and not more than 2-3 inches distant from the pyloric outlet. As the distal pouch was small and insignificant, drainage of the proximal pouch was effected either by the anterior or posterior method of anastomosis. If the distal pouch was of fair capacity, the operation was doomed to failure, particularly in those cases where there was any

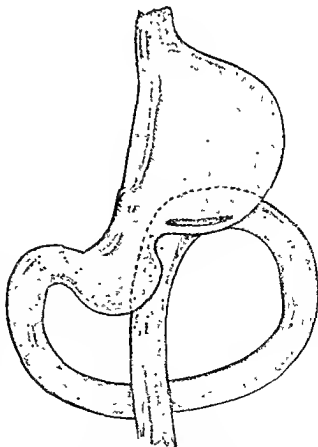


Fig 93—Hour glass stomach. SINGLE GASTRO-JEJUNOSTOMY. NOTE THAT THE PROXIMAL POUCH IN THIS CASE IS VERY SMALL.

concomitant pyloric obstruction. The distal pouch emptied its stagnant contents into the duodenum with the greatest difficulty. The addition of pyloroplasty in these cases was of very doubtful benefit. The ulcer on the lesser curvature was not excised as a routine measure. The short-circuiting operation, too, may be one of great difficulty, as the causative ulcer may be fixed to the pancreas, adhesions may be

extensive, and the cardiac pouch difficult to approximate to the jejunal loop without tension.

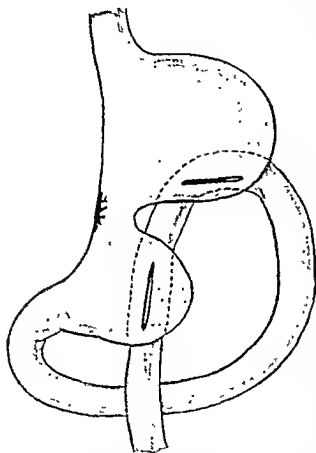


Fig. 94.—HORN CLASH STOMACH. DOUBLE GASTRO-JEJUNOSTOMY.

very large opening or openings will have to be made in the mesocolon, which at a later date may contract and produce mechanical interference with the jejunal loops.

*Gastro-gastrostomy (gastro-anastomosis)* (figs. 95, 96 and 97). This is a simple operation to perform and has a low mortality. A sufficient portion of the pouches on either side of and below the isthmus is clamped, and an anastomosis is made between these two parts. It is advisable that this operation should be combined with some form of pyloroplasty. (Parnett.) The portions of the stomach used in the

(b) *Double gastro-jejuno-stomy* (fig. 94). In this operation a fairly long loop of jejunum is taken close to the flexure, and two separate anastomoses are made, the first to the cardiac pouch and the second to the pyloric pouch. This operation, whether by the anterior or posterior method, is most unsatisfactory and cannot be recommended as it is difficult to perform and there is a high percentage of failures.

When the posterior operation is performed, a

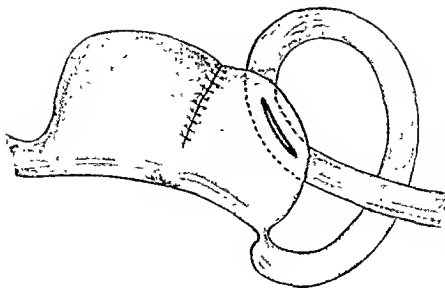


Fig. 97.—Hour glass stomach. Gastro-gastrostomy with gastrojejunostomy to the distal pouch in a case of hour-glass stomach with pyloric stenosis.

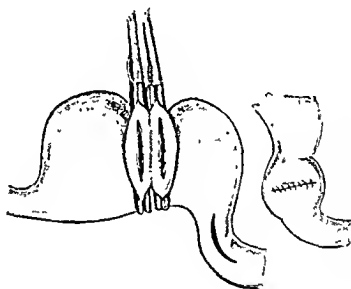


Fig. 98.—Hour glass stomach. Gastro-gastrostomy with pyloroplasty.

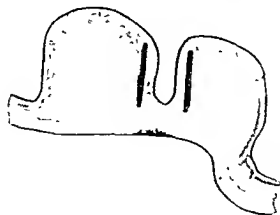


Fig. 95.—Hour glass stomach. Gastro-gastrostomy. The lines of incision in the two gastric pouches are shown.

anastomosis are healthy, and the anastomosis works well without any subsequent stenosis. The ulcer is not, as a rule, excised.

Hurst, Rowlands, Gordon-Taylor, and others describe the results of this operation as being entirely satisfactory. Hurst, for instance, records 25 cases without a death, and excellent late results. Paterson, however, has found that in about 30 per cent of cases the late results are disappointing.

In the rare event of perforation of a chronic gastric ulcer which has produced an hour-glass stomach, the perforation should be closed with interrupted sutures of stout catgut, the suture line protected with an ample omental pad, and a gastro-gastrostomy performed. If the patient is in good condition at the completion of this operation, it is wise to relieve tension in the stomach and aid rapid evacuation of its contents by undertaking a pyloroplasty.

*Gastropasty.* (a) *After the Heineke-Mikulicz method* (figs. 98 and 99). This is the operation of pyloroplasty applied to the stomach. A longitudinal incision, 3-4 inches long, is made through the anterior wall and in the long axis of the stomach, as shown in fig. 98. The incision is carried through all the coats of the stomach, from the middle of the cardiac pouch, across the isthmus, and into the pyloric segment. The wound thus produced is sewn up transversely by a two- or three-tier suture. This operation, however well performed, and even in selected cases, is followed by a high percentage of recurrence, as the incision is made through scar tissue and some contraction will therefore almost inevitably follow.

(b) *Kammerer's operation* (fig. 100). This is an application of Finney's operation of pyloroplasty. Here again the incision as it approaches the lesser curvature is made in indurated and inflammatory tissue. Contraction of the stoma will almost inevitably take place, giving rise to recurrence of symptoms. It is, however, a simple procedure and the temporary results appear to be satisfactory.

#### *Sleeve Resection* (figs. 101, 102, 103 and 104)

This operation is sometimes indicated, the best results being obtained when it is combined with some form of pyloroplasty. Sleeve resection may be followed by a recurrence of hour-glass constriction, or there may be considerable interference with the muscular function

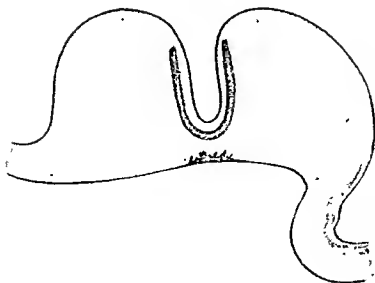


Fig. 100.—Hour glass stomach. Karyer's operation.

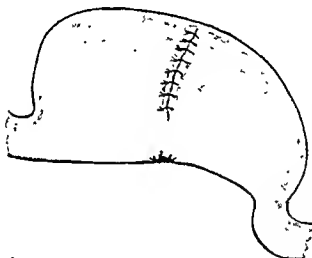


Fig. 99.—The Heister-Mikulicz operation completed.

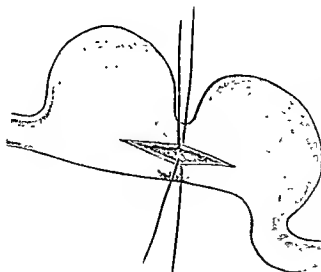


Fig. 98.—The Heister-Mikulicz operation for hour glass stomach.

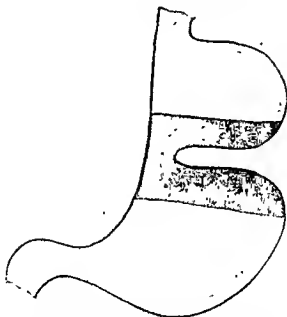


Fig. 101.—HOTR-GLASS STOMACH, SLEEVE RESECTION.  
THE DAPELA SHADED AREA REPRESENTS THE SEGMENT  
OF STOMACH REMOVED BY THIS OPERATION.

of the stomach, giving rise to some troublesome and persistent post-operative symptoms. In the operation of sleeve resection the lesser sac is exposed through an opening made in the gastro-colic omentum, and the posterior wall of the stomach is inspected and any adhesions here are carefully separated. An area of stomach is isolated after

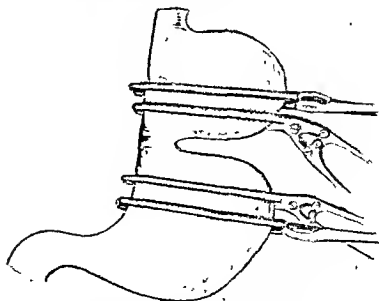
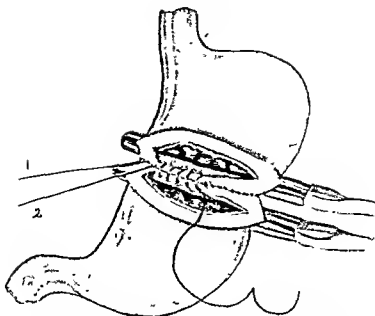
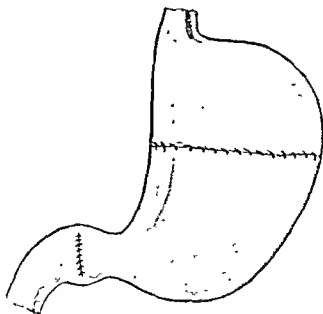


Fig. 102.—SLEEVE RESECTION OF THE STOMACH, SHOWING  
CLAMPS IN POSITION PRIOR TO RESECTION.



*Fig. 103.*—SLEEVE RESECTION OF THE STOMACH. THE ANASTOMOSIS OF THE PROXIMAL AND DISTAL PORTIONS OF THE STOMACH IS BEING PERFORMED AS IN THE OPERATION OF GASTRO-JEJUNOSTOMY.

ligaturing the blood-vessels at the outer limits of the area mapped out for excision. Payr clamps and Sherren clamps are applied in the manner indicated in figure 102. The portion of stomach embraced by the Payr clamps and including the isthmus and the ulcer is excised. The ends of the stomach are then brought together and anastomosed by the end-to-end method.



*Fig. 104.*—SLEEVE RESECTION OF THE STOMACH. OPERATION COMPLETED AND PALATOPLASTY PERFORMED.



*Walton's Operation* (V-excision of ulcer, pyloric occlusion, and posterior transverse gastro-jejunostomy) (figs. 105, 106 and 107).

Walton (*Surgical Dyspepsias*, p. 405, Arnold, 1930), describes the essential steps of this operation as follows :

" This operation brings into line the treatment of hour-glass stomach with that of any form of ulcer on the lesser curve. The ulcer itself is removed and with it the danger of perforation, hemorrhage, or the late onset of carcinoma ; while the performance of a gastro-enterostomy will overcome the tendency to ulcer formation and thus prevent a fresh ulcer arising at the site of excision. If there be an ulcer with stenosis at the pylorus it will be embedded in the usual manner, but even if the pylorus be free it should be occluded so that every opportunity be given to the gastro-enterostomy to act in a satisfactory manner.

" By making the V-excision with a widely open angle the two limbs can be made of practically any length, the one being continued up to the oesophageal opening and the other towards the pylorus. When these two limbs are approximated and sutured the hour-glass constriction will be entirely overcome and the stomach restored to its normal shape. The width of the base of the V-shaped portion removed will vary with the amount of stenosis. . . .

" In the majority of cases the technique will present but little difficulty, clamps being applied proximally and distally to the ulcer and the incision being made between them in the usual way. The gastro-enterostomy will be made on the posterior surface and will be transverse, and will be so placed that the proximal half drains what was the cardiac pouch and the distal half the pyloric pouch. It will therefore have to pass along the isthmus of normal mucosa, otherwise the sutures of the line of excision will be cent. For this reason every care must be taken to preserve the normal band of mucosa

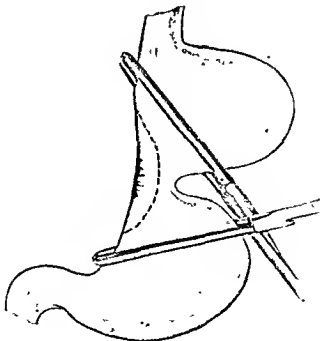
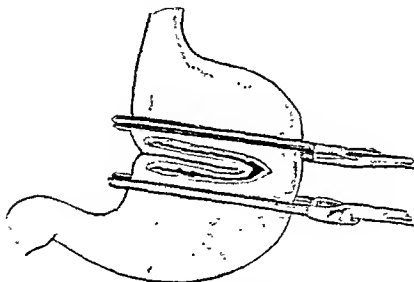


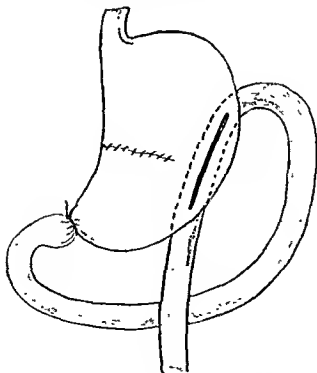
Fig 105.—Hour-glass Stomach. WALTON'S OPERATION  
THE APPLICATION OF THE CLAMPS.

at the base. In certain cases the isthmus is so narrow that a band of stomach wall sufficient to permit of the gastro-enterostomy cannot be left and under these circumstances the operation is not feasible.

"In my own series of twenty-three cases there were two deaths and twenty complete cures."



*Fig. 106.*—WALTON'S OPERATION. A WIDE V EXCISION OF THE ULCER HAS BEEN PERFORMED, AND THE CLAMPS ARE APPROXIMATED, DRAWING THE TWO POUCHES OF THE STOMACH TOGETHER BEFORE COMMENCING THE ANASTOMOSIS



*Fig. 107.*—WALTON'S OPERATION COMPLETED BY OCCLUSION OF THE PYLORUS.

*Partial Gastrectomy (figs. 108 and 109)*

This operation should be advised in every case except where general and local conditions are unfavourable to its performance. It will therefore be contra-indicated in patients who are gravely ill as the result of prolonged vomiting and starvation, and in whom an intensive pre-operative regime of treatment has led to little or no amelioration of the general condition—a rare experience.

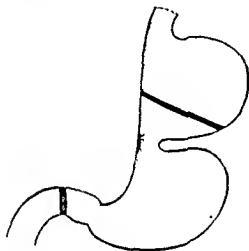


Fig 108—HOLE-GLASS STOMACH. PARTIAL GASTRECTOMY. THE DARK LINES INDICATE THE AREA TO BE EXCISED.

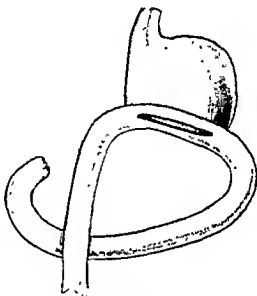


Fig 109—PARTIAL GASTRECTOMY FOR HOLE-GLASS STOMACH.

Gastrectomy is urgently indicated when multiple chronic peptic ulcers are present, where doubts exist as to the benignity of a large callous ulcer, and where hæmorrhage from the ulcer bed is an added complication.

### PYLORIC OBSTRUCTION

#### *Causes*

- (1) *Intrinsic.*
  - (a) Chronic peptic ulcer.
    - (i) Pyloric.
    - (ii) Duodenal.

(b) New growths of the stomach.

(i) Carcinoma.

(ii) Polypus.

(c) Simple stenosis of the pyloric canal.

(2) *Extrinsic*.

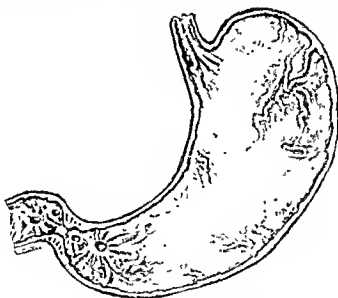
(a) Adhesions due to perigastric inflammation.

(b) Pressure on the pylorus or on the duodenum by a diseased neighbouring viscus, e.g., cancer of the head of the pancreas.

(c) Traction upon or kinking of the duodenum, e.g., by mobile kidney; gastropptosis.

### *Ætiology and Pathology*

(1) *Peptic Ulceration*. The commonest cause of pyloric obstruction is peptic ulceration, and the most frequent single cause is duodenal



*Fig. 110.—PYLORIC OBSTRUCTION DUE TO CHRONIC PEPTIC ULCERATION. THIS FIGURE ILLUSTRATES TWO GASTRIC ULCERS, ONE SITUATED IN THE VESTIBULE AND THE OTHER IN THE PYLORIC CANAL, BOTH BEING ON THE POSTERIOR WALL OF THE STOMACH. TWO CHRONIC, POSTERIORLY-PLACED DUODENAL ULCERS ARE ALSO DEPICTED IN THE COMMON SITES WHERE THEY GIVE RISE TO PYLORIC OBSTRUCTION.*

ulcer, which has been estimated to be seven times commoner than gastric ulcer (fig. 110). It is said that 25 per cent of cases of duodenal ulcer produce pyloric stenosis. The condition is more frequently seen in males than in females, as duodenal ulcer occurs more often in the former sex.

Anteriorly-placed duodenal ulcers tend, as a rule, to be smaller

than those posteriorly situated. It is these latter ulcers which are the chief cause of stricture. They become fixed to the pancreas, and at the same time lead to a great deal of fibrosis. The position of such an ulcer—close to the pylorus, large, excavating, and swollen with cedema—is, of itself, sufficient to produce obstruction. In about 50 per cent of cases of pyloric obstruction due to ulcer, the ulcer will be found to be completely healed at operation or at post-mortem examination.

In old-standing cases the hard, scarred, puckered, and distorted pylorus may be dragged and fixed into an abnormal position, but it is more often loosely anchored to the posterior abdominal wall by long, stretched, avascular adhesions. The hard mass in the pyloric region, white and scarred, may resemble a new growth, and it may occasionally be exceedingly difficult to distinguish it from a cancerous lesion.

In cases of some duration no stippling of vessels can be produced by friction. On invaginating the gut some clue as to the nature of the lesion may be gained. For instance, in carcinoma the "knobbly" edge or crater of the growth may be felt, and seedlings be visible on the peritoneal coat of the gut in the immediate vicinity of the stricture.

In most cases of pyloric obstruction the emissive ulcer has been present for some considerable time—a period of many years; but the duration of the symptoms is almost wholly dependent upon the exact anatomical site of the ulcer. For instance, when situated in the pyloric canal, at the pyloric outlet, or in the duodenum just distal to the pylorus, the advent of obstructive symptoms is not long delayed. As soon as obstruction supervenes, the muscle of the stomach hypertrophies owing to the additional work imposed upon it. This hypertrophy is at first confined to the pyloric region of the stomach, but slowly extends into the body, then into the fundus, and may even involve the lower portion of the œsophagus. Unless the obstruction is relieved, dilatation of the whole stomach will follow, until eventually it becomes saccular and loses its anatomical divisions.

In an advanced case the dilatation may be enormous and the viscus may occupy the major portion of the abdominal cavity and even sink down into the pelvis.

(2) *New Growths of the Stomach.* There are two common varieties of cancer of the pyloric region of the stomach which produce obstruction—the cauliflower mass and the scirrhous cancer of the pyloric canal.

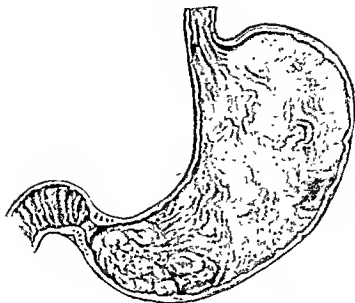


Fig. 111.—PYLORIC OBSTRUCTION DUE TO CAULIFLOWER GROWTH.

The *cauliflower growth* if situated in the pyloric segment may, by its massive size, cause a mechanical block of the lumen of the pyloric canal (fig. 111). It is, however, the less usual cause of obstruction.

*Scirrhus cancer* of the pyloric canal is a comparatively common cause of stenosis, as this slow-growing form of growth is associated with a great deal of fibrosis and gradual contraction of the pylorus (fig. 112). It converts the pyloric canal into a dense, rigid, narrow, thick-walled

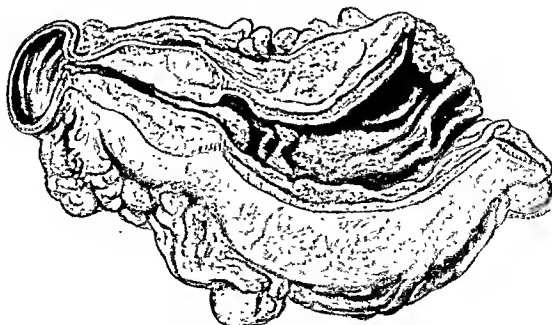


Fig. 112.—SCIRRHUS CANCER OF THE PYLORUS, PRODUCING OBSTRUCTION. DRAWN FROM A SPECIMEN REMOVED AT OPERATION. (Author's case.)

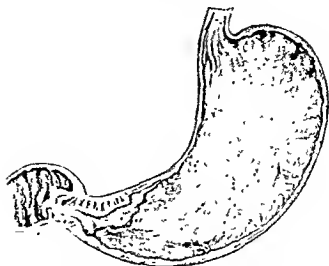


Fig. 113.—SCIRRHUS CANCER OF THE PYLORUS, PRODUCING OBSTRUCTION.

tube, and produces a very gradual dilatation of the stomach (fig. 113). As the disease progresses, however, the dilatation increases to such an extent that the stomach is capable of holding several pints of fluid.

A *polypus* may act as a ball-valve and so produce obstruction (fig. 114), or it may pass through the duodenum, dragging the stomach wall with it and causing an intussusception of the stomach.

(3) *Simple Stenosis of the Pyloric Canal.* A gastric ulcer on the lesser curvature, chronic appendicitis, chronic diseases of the gall-

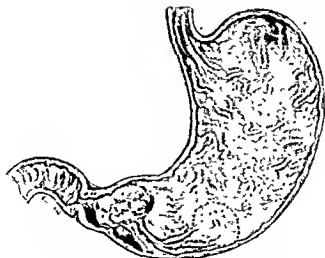


Fig. 114.—PYLORIC OBSTRUCTION DUE TO A PEDUNCULATED POLYPUS. THE POLYPUS MAY ACT AS A BALL-VALVE, CAUSING INTERMITTENT OBSTRUCTION.

bladder, visceroptosis, and other abdominal conditions may produce a reflex pyloric spasm, but there is no evidence that such spasm can cause hypertrophy of the muscle of the pyloric canal or fibrous stenosis. Occasionally simple stricture or fibrosis is found in the pyloric canal without any evidence of previous ulceration. Walton, however, considers that it is possible that a certain proportion of these are cases of congenital hypertrophy of the pylorus which have survived to adult life. A number of such cases have been described in which the pathological characteristics of the pyloric tumour are identical with those of infantile stenosis.

(4) *Perigastric Inflammation as a Cause of Pyloric Stenosis.* The commonest causes of perigastric inflammation and those which are most likely to give rise to pyloric stenosis are :

- (a) Acute cholecystitis with gall-stones.
- (b) Pancreatitis.
- (c) Sub-acute perforation of a peptic ulcer.

Pyloric obstruction produced by perigastric adhesions is usually not of a severe nature, and rarely becomes complete. In some cases of acute obstructive cholecystitis the duodenum, the omentum, and even the hepatic flexure of the colon may become welded to the gall-bladder in a hard, fixed lump which may, on examination, simulate a malignant growth or a perigastric abscess.

Compression of the duodenum may occur during this acute phase, but this is more likely at a later date owing to the slow contraction of the massive surrounding adhesions.

Gall-stones may be a cause of pyloric stenosis. In cases of cholecystitis associated with cholelithiasis the duodenum may become fixed to the gall-bladder, and a gall-stone—usually a large one—ulcerate through the contiguous walls and pass into the duodenum. If large, it may remain lodged in the duodenum itself. If it successfully navigates this portion of the gut, its journey onwards through the small intestine is usually unimpeded until it encounters the lower reaches of the ileum, where the gut narrows, and where impaction frequently occurs, giving rise to acute intestinal obstruction. The site of perforation eventually becomes the site of obstruction.

(5) *Pressure on the Duodenum or Pylorus by a Diseased Neighbouring Viscus* (figs. 115 and 116). The following diseases in neighbouring



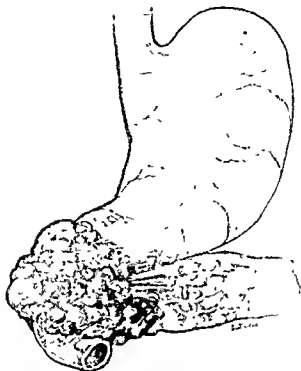


Fig 115.—PYLORIC OBSTRUCTION DUE TO CANCER OF THE HEAD OF THE PANCREAS.

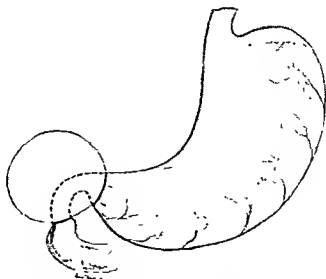


Fig 116.—PYLORIC OBSTRUCTION DUE TO COMPRESSION OF THE DUODENUM OR PYLORUS BY A TUMOR.

viscera may compress the pylorus or duodenum, and give rise to pyloric obstruction :

- (a) Cancer of the head of the pancreas.
- (b) Chronic pancreatitis.
- (c) Diseases of the gall-bladder and bile-ducts.
- (d) Duodenal diverticula.
- (e) Cancer of the hepatic flexure of the colon.
- (f) New growths of the liver.
- (g) Hydatid cysts of the liver.

(6) *Traction upon or Kinking of the Duodenum.* A mobile kidney may pull the attached duodenum downwards and produce temporary

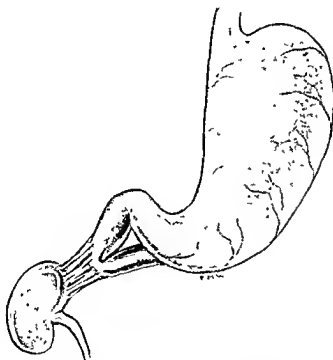


Fig 117.—PYLORIC OBSTRUCTION DUE TO MOBILE KIDNEY.

kinking of the gut (fig. 117), giving rise to obstruction and to one of the varieties of Dietl's crises. The obstruction takes the form of short, sharp, intermittent attacks, with long intervals of freedom. This form of obstruction is sometimes seen in visceroptotic females and associated with marked mobility of the kidneys.

*Gastropptosis* may be a further cause of pyloric obstruction (fig. 118). If the pylorus becomes fixed as the result of perigastric adhesions, it can readily be appreciated that the large, dilated, hypotonic stomach,

having become still further elongated and weighed down by its contents, may cause further drag and kinking at this point of attachment.

These cases of gastropptosis very closely resemble those of stenosis due to ulcer, but a barium meal examination will settle the diagnosis. In gastropptosis, owing to atonic dilatation, peristaltic waves are usually absent, the duodenal cap is normal, and there is no evidence of ulceration or other organic lesion.

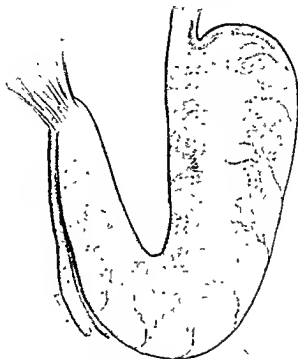


Fig. 118.—GASTROPTOSIS MAY BE A CAUSE OF PYLORIC OBSTRUCTION.

### *Signs and Symptoms*

Although there are many causes of pyloric obstruction, once the condition is well established there is but little variation in the symptoms produced. The symptoms are due to two main factors: (a) the mechanical stoppage of the outlet of the stomach; and (b) secondary gastritis.

Where peptic ulcer is the causative factor the history alone will supply an important clue to the diagnosis, as symptoms typical of peptic ulcer will, in most cases, be found to have been present for many years.

On careful inquiry, therefore, the majority will give a history of

duodenal ulcer, but with the onset of pyloric obstruction the characteristic symptoms of peptic ulcer will disappear, there will be no intermissions, nor will there be any period of freedom from symptoms.

*Pain.* As soon as pyloric stenosis supervenes, true hunger pain ceases and is replaced by pain which is more or less continuous, but of a milder nature. Food may aggravate the pain, as will also the first meal taken after a bout of vomiting. Although the pain is mostly continuous during the day, occasional irregular and severe bouts, colicky in nature, may be experienced. These colicky pains may develop into sudden severe attacks of violent cramp in the epigastrium, and are said to be due to tetanic contraction of the whole stomach. When present they may be regarded as being pathognomonic of organic pyloric stenosis.

It should be noted that the intake of food aggravates the pain but never relieves it; in fact, it is only after vomiting or after the stomach has been emptied by a stomach tube that relief is afforded. Such patients will therefore often resort to self-induced vomiting or become practised in the use of a stomach tube. More pain is experienced in the evening when the stomach has become distended with the accumulation of food taken through the day.

*Vomiting.* Vomiting is one of the most outstanding characteristic symptoms of pyloric stenosis. At first it is fairly frequent, once or twice a day, and this continues as long as there is hypertrophy of the stomach. As dilatation supervenes the intervals between the bouts of vomiting become longer, but the amounts voided are more copious. In a severe case, if the stomach is completely emptied through vomiting or by means of a stomach tube, it may take one or two days for it to refill. As the dilatation becomes worse the stomach, as the result of atony, is capable of retaining very large quantities of decomposing material, even amounting to pints or quarts. When voided, this foul, frothy, fermenting fluid may contain particles of food ingested several days previously.

*Appetite.* When obstruction is due to peptic ulcer, the appetite in the early stages may be good; but as the obstruction increases it becomes less, until eventually there is anorexia which is due, in part, to a secondary gastritis and to the patient's fear that the partaking of food will cause vomiting. There may be such complete anorexia

in late cases that this symptom, combined with the physical signs, will suggest that the patient is suffering from a cancer of the stomach.

*Thirst.* Thirst is a very common symptom and is increased by vomiting. Here again the patient is afraid to drink as he attributes his vomiting to the intake of water or liquid nourishment.

*Weight.* There is a progressive loss of weight, which is due to vomiting, lack of appetite, self-induced starvation, or a combination of the three. This loss of weight is accompanied by marked dehydration of the tissues, which is evidenced by the inelastic and parched condition of the skin. As is usual with such loss of weight, amounting generally to several stooes, there is weakness, giddiness, lassitude, drowsiness, and headaches.

*Bowels.* Constipation is usually very marked. This is due to the small quantities of food and fluid which can pass the obstruction. When the obstruction is incomplete, the foul contents of the stomach may irritate the intestines and give rise to diarrhoea.

*Urine.* The urine in such cases is always scanty, and frequently contains acetone bodies.

### *Examination*

On examination wasting is evident, the extent depending upon the degree of obstruction and the length of time it has existed. Wasting in benign obstruction is often more marked than in malignant, as the former condition is of longer duration and the stenosis is more absolute. The patient will be found to be thin, pale, and anæmic; the tongue will be coated, and the breath foul.

On examining the abdomen the upper half may be found to be dilated and the lower half retracted; but where dilatation is advanced the whole abdomen is distended. Visible peristalsis is occasionally seen in the region of the umbilicus, the waves passing from left to right. Gurgling noises due to peristalsis or to movement of the body may also be audible. Palpation may cause splashing sounds and elicit some tenderness over the area occupied by the dilated stomach. The area of stomach resonance is diminished, and its extent will be found to vary according to whether the patient is lying down or standing up.

There should be a systematic search for an abdominal tumour, especially in the region of the pylorus. If on examination a mass is found in the pyloric region, its physical characteristics may proclaim its nature. A small, tender, somewhat ill-defined tumour is suggestive of a benign obstructive lesion, whilst a hard, craggy mass is typical of cancer.

Tetany may be present in severe cases of pyloric obstruction, and when present there will be evidence of alkalosis. An X-ray examination after the administration of a barium meal is essential in clinching the diagnosis. This will not only demonstrate with the greatest accuracy the degree of obstruction, but also the causative lesion, its nature and its position. Examination of the stools may also be of value. For instance, in a case of duodenal ulcer causing pyloric stenosis and confirmed by X-rays, a repeatedly positive occult blood test will prove that the ulcer is still active. An examination, too, of the blood serum will help to indicate to what degree alkalosis is present. A test meal may yield very useful information, as will also the passage of a stomach tube and aspiration of the gastric contents. The very large amounts withdrawn daily, combined with the chemical findings, will suggest the presence of organic obstruction to the outlet of the stomach, and may even provide a differential diagnosis between benign and malignant stenosis.

### *Differential Diagnosis between Benign and other Types of Pyloric Obstruction*

(1) *Carcinoma of the Pyloric Portion of the Stomach.* In this condition there is usually a short history, less than a year, obstructive symptoms being abruptly ushered in comparatively early in the course of the disease. Wasting, although present, is not so marked as in benign obstruction, and the loss of appetite almost coincides with the onset of the disease.

The history of gastric discomfort and pain after food is short, unlike cases of simple stenosis in which there may often be a previous history of typical hunger pain, dating back many years.

A hard tumour may be felt in the pylorus, the nature of which will be revealed by X-ray examination.

(2) *Carcinoma of the Head of the Pancreas.* It is only in the very late stage of this disease that obstruction of the gut occurs. By this

time the patient will be jaundiced on account of the blockage of the common duct or the ampulla of Vater.

The hard growth in the pancreatic head may be palpable, as may also be an enlarged gall-bladder. The liver will usually be found to be enlarged below the costal margin to the extent of two or three finger-breadths. There may also be ascites.

(3) *Perigastric Inflammation.* Perigastric inflammation may be due to acute cholecystitis. A large mass will be found to the right of the epigastrium in the region of the gall-bladder. This mass will be continuous with the liver above, and will move on respiration. There will be muscular guarding and tenderness over this area, in addition to the usual constitutional signs and symptoms. When the omentum or hepatic flexure becomes welded to the inflamed structures in this region, a large tumour may be formed which may be indistinguishable from that of carcinoma.

A carcinoma, however, which is large enough to invade the liver by direct spread must be very advanced, and of such long standing as to produce secondaries in the liver and metastases elsewhere.

The history will be helpful, as in perigastric inflammation there will be a story of gall-bladder trouble or possibly of intermittent attacks of colic followed by jaundice.

(4) *Obstruction due to Kinking of the Pylorus by a Mobile Kidney or Large Atonic Stomach.* In such cases visceroptosis is evident and on palpation a mobile right kidney can easily be identified. The stomach will be found to be large and sinking down into the pelvis. Obstructive symptoms are incomplete and irregular, and are characterised by short, sharp, acute attacks which last for very brief periods. Although during an attack vomiting may be very severe, it only lasts for a few hours, or at the most for a day or two, followed by periods of freedom. During a bout of vomiting the nature of the vomited material and the total amount voided will differ from that seen in cases of organic obstruction. With kinking the amounts are less and the vomit does not show the characteristic features of decomposition and fermentation which are seen where there is a mechanical organic obstruction. The effect, moreover, of posture in producing relief of symptoms is more marked.

(5) *Chronic Duodenal Ileus.* In some severe cases of duodenal ileus the vomiting may be so severe as to simulate pyloric stenosis. A barium meal examination will, however, determine the site of the constricting agent.

### *Treatment*

The presence of organic obstruction of the pylorus, whatever its nature, is an indisputable indication for operation.

#### (1) *Pre-operative.*

Cases of pyloric obstruction should be carefully prepared for operation on the following lines. If such pre-operative treatment is carried out for a period of days, great improvement will result and operation will be attended with little risk.

(a) The stomach should be emptied twice a day, morning and night, with a Senoran evacuator, or if the obstruction is very great a Ryle tube may be passed through the nostril and be left *in situ* to provide continuous drainage.

(b) Any alkaline medicines which have been prescribed should at once be discontinued, and a mixture containing small doses of hydrochloric acid should be given, t.d.s.

(c) Sips of sterile water should be given frequently by the mouth.

(d) 6 oz. of 5 per cent glucose in saline should be given per rectum six-hourly.

(e) 100 cc. of 6 per cent saline should be injected intravenously twice a day, alternately with 100 cc. of 10 per cent glucose solution. If alkalosis is marked, four 10-oz. doses of 2 per cent ammonium chloride solution should be given per rectum.

(f) A blood-transfusion may be helpful.

(2) *Operative Treatment.* (a) *For Pyloric Obstruction due to Peptic Ulcer.* The operation of choice is a posterior gastro-jejunostomy. On completion of the short-circuiting operation the scarred pyloric region should be enfolded with a few interrupted Lembert sutures. If conditions permit, the abdomen should then be explored for any concomitant lesion. The appendix should be removed if possible, but this step should be omitted if the patient's condition is feeble or if it is deemed inadvisable to prolong the operation. If a diseased gall-bladder is found it is often wiser to defer its removal until a later date when recovery from the first operation is complete.

The gastro-jejunostomy is best performed without clamps, and the jejunal loop may be placed transversely or vertically. If clamps are used they will have to be applied very gently, as the stomach is often greatly dilated and its walls are thinned, stretched, and friable to a degree.

The results of gastro-jejunostomy for pyloric obstruction due to peptic ulcer are exceedingly good. The mortality is only 1-3 per cent, and 95 per cent of cures are recorded. The post-operative complication of gastro-jejunal ulceration is very rarely seen—less than 1 per cent.

Gastro-duodenostomy has been performed as an alternative method



to gastro-jejunostomy, but the final results do not appear to be so uniformly satisfactory.

When a gastro-jejunostomy is performed the stoma should be at least three inches in length, to allow for the narrowing which will take place subsequent upon the contraction of the stomach.

(b) *For Pyloric Obstruction due to New Growths of the Pylorus.* Partial gastrectomy should be performed, and this operation should be undertaken wherever the growth can be resected with safety, even in the presence of secondary deposits in the liver.

If the growth is irremovable a gastro-jejunostomy will be necessary in order to relieve the obstruction, although Devine's method of exclusion of the growth is a better procedure.

If the obstructing agent is a solitary, pedunculated polypus, its base should be freely excised through an opening made in the anterior wall of the stomach—gastrotomy. If, however, the base of the polypus is indurated or multiple polypi are confined to the pyloric segment, partial gastrectomy is the most satisfactory undertaking.

(c) *For Obstruction due to Adhesions produced by Perigastric Inflammation.* If it is impossible to deal with the primary cause owing to an insuperable barrier of matted adhesions, gastro-jejunostomy should be performed.

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## CHAPTER VI

### TREATMENT OF CHRONIC PEPTIC ULCER

- (A) MEDICAL TREATMENT (see page 573).
- (B) SOME FACTORS WHICH INFLUENCE THE CHOICE OF TREATMENT
- (C) INDICATIONS FOR OPERATION
- (D) CHOICE OF OPERATION AND RESULTS
- (E) PRE-OPERATIVE TREATMENT
- (F) TECHNIQUE OF THE OPERATIONS
- (G) POST-OPERATIVE TREATMENT
- (H) POST-OPERATIVE COMPLICATIONS

#### INTRODUCTION

*The treatment of chronic peptic ulcer is primarily and essentially medical.*

*Surgery is indicated where complications have occurred, where the menace of malignancy cannot be excluded, or where the chronicity of the disease is established by the recurrence and persistence of symptoms in spite of methodical, efficient and adequate medical treatment.*

Comparison between the relative merits of medical and surgical treatment is nowadays impossible, as, except in the presence of complications which demand surgical interference, operation is reserved for those cases which medicine has failed to cure after a reasonable trial. But medical treatment is often doomed to failure because it is too cursory, too casual, or not sufficiently prolonged for lasting benefit to be derived. On the other hand, there is sometimes a tendency to persist with medical measures indefinitely after it should be obvious that cure by these means is very improbable.

Where medical treatment is undertaken it must be adequate, i.e. strict, systematic, carefully planned, and prolonged, following a scheme as laid down on page 575. If carried out on such lines, there is

every reason to believe that such treatment would in many cases be followed by a permanent cure.

While the patient is undergoing strict medical treatment in hospital and is under supervision, there will often be immediate and marked relief from symptoms and improvement in the general and local condition; but it is during the later ambulatory stage of treatment, when so much depends upon the patient's loyal co-operation, that there is often a relapse. This is not altogether to be wondered at, as this stage of the treatment makes certain demands upon the patient, calling for strength of character, perseverance, close attention to diet, the regular taking of medicines, and a faithful adherence to all the details of the regime over a period of many months or even years. Any departure from the strict routine, any slackness in the observance of the medical ritual, or any self-indulgence will contravene and destroy the efficacy of such treatment, favouring a return of the symptoms or even leading to serious complications.

Thus many patients, rather than be subjected to the tedium of a long and, as considered by some, rigorous line of treatment, demand immediate surgical measures in the hope of obtaining more speedy relief from their symptoms.

It should be remembered that quite a number of patients who are thus unwilling in the first instance to undergo medical treatment also fail to follow the necessary regime after operation has been undertaken, and many of the poor results of surgery for peptic ulcer are due to the operation being regarded as a cure in itself rather than as an incident in the course of medical treatment.

*Surgery, as a quick method of cure, should not be lightly advised nor precipitately undertaken.*

During the first few years following the War, many cases of chronic peptic ulcer were operated upon indiscriminately and incompetently, with so large a proportion of resulting failures that a strong reaction in favour of prolonged medical treatment followed. So far did the pendulum swing in this direction, intensive alkaline treatment being extravagantly boomed, both by medical papers and by the Press, as an unfailing means of cure, that it was not long before complications such as acute perforation and hæmorrhage showed a surprising increase throughout the country, as the figures of any large general hospital will show. This has also been recently emphasised by Pyrah who writes as follows:

"There has been an increase in the medical treatment of ulcer and a decrease in the number of cases of chronic ulcer operated upon. The average number of opera-

tions per annum before 1926 (in Leeds) was over 200 ; the average number for the last four years was 140. There has been an increase since 1926 in the number of perforated ulcers of the stomach and duodenum. Between 1919 and 1925 the number of perforations averaged 50 per year ; for the last five years the number has been 105 per year, that is, they have more than doubled.

In assessing the results of medical treatment we must take into account the mortality of complications, such as perforated ulcers, which may arise in patients thought to have been cured medically but who subsequently perforated. I cannot give the figures concerning that, but the deduction from the parallelism of the facts I have quoted, the increase in the number treated medically and the decrease in the number operated upon, shows, I think, that the increase in the perforations must be laid at the door of medical treatment.

The mortality from perforated ulcer from 1926-31 in Leeds in 699 cases was 18.7 per cent ; the operation mortality for chronic ulcers was 5 per cent for duodenal, and somewhat higher for partial gastrectomy, i.e., for gastric ulcers. So the increased number of perforation deaths is over the total number of operation deaths for chronic ulcer cases treated surgically. And many of the cases which had perforated returned later with symptoms, as, frequently, it is impossible to carry out the ideal surgical treatment at the time of the perforation." (Pyrah, *Post-Grad. Med. Jour.*, No. 104, p. 217, June, 1934.)

Both medical and surgical treatment may now be said to have passed through the stage of trial by error.

Surgery to-day is employed only, as Wilkie puts it, with deliberation and discrimination.

In many cases operation opportunely performed may renew the patient's lease of life ; on the other hand, a badly designed operation or operation in an unsuitable case may produce an increased and lasting state of ill-health and invalidism.

The main principles in the treatment of chronic peptic ulcer, whether by medical or surgical measures, will include :

- (1) Elimination of sepsis.
- (2) Avoidance of trauma.
- (3) Correction of stasis.
- (4) Combating acidity.
- (5) Removal of the ulcer.

*Where surgery is undertaken, medical measures form an essential part of the treatment, both before and after operation.*

In the surgical treatment of chronic peptic ulcer a high percentage of cures will result if the following points are observed :

- (1) Wise discrimination in the selection of cases for operation.
- (2) Care in pre-operative treatment to render the patient as fit as possible for operation.

- (3) The selection of the correct type of operation required in the individual case.
- (4) Perfect mechanics and faultless technique in the conduct of the operation.
- (5) Prolonged post-operative treatment.

The final decision as to whether medical treatment or operation should be advised should never be based on any rule-of-thumb method. A careful review of all the available evidence, both local and general, must be made in each case. So difficult may the making of this decision sometimes prove, that the closest co-operation between physician, surgeon, radiologist and pathologist may be required, and in no instance is there greater need for the advantages afforded by team work.

#### (A) MEDICAL TREATMENT (see page 573)

#### (B) SOME FACTORS WHICH INFLUENCE THE CHOICE OF TREATMENT

(1) *Age.* In the absence of complications operation should not be advised in young patients, i.e. under 30 years of age, as in most instances the ulcer is of recent date, obstruction and anchorage are rare, and co-operation in the matter of post-operative caution is less easily ensured. A high incidence of jejunal ulcer and of other unsatisfactory results following the operation of gastro-jejunostomy is recorded in patients under 30.

For instance, Pyrah showed that of 389 cases "61 of the patients who submitted to gastro-enterostomy were under 30 years of age, and 38 per cent of them showed a bad result. Therefore I think that, except for mechanical reasons, gastro-enterostomy should not be performed in patients under 30 years of age with duodenal ulcer."

In young patients suffering from chronic peptic ulcer there are, as a rule, only two complications which call for surgical interference: perforation and recurrent hæmorrhage. Perforation should be treated by simple suture, gastro-jejunostomy being performed only where it becomes obligatory owing to suturing of the perforation having produced pyloric or duodenal occlusion.

The treatment of recurrent hæmorrhage is discussed on page 583.

In young patients especially, partial gastrectomy should be performed wherever possible, for, as has already been emphasised, recurrence of the hæmorrhage or the development of stomal ulceration is very liable to occur in such subjects after gastro-jejunostomy.

The best results of gastric surgery are obtained in those in whom the symptoms have been present for not more than 5 years, and in middle-aged patients, i.e. 40-55 years, and such operations are most frequently performed during this 15-year period. Elderly persons with gastric or duodenal ulcer respond much less readily to medical treatment than those who are still in early middle life, this being due probably to impaired circulation and diminished recuperative powers in the former (Conybeare). As might be expected, the risks of surgical interference are greater in old age, but, where clearly indicated, operation should nevertheless be undertaken, being made as conservative as possible.

(2) *Sex.* Sex does not, in itself, necessarily influence the choice of treatment, although it should be noted that the death-rate following gastric operations is lower in females than it is in males, and on the whole the late results of gastro-jejunostomy for chronic duodenal ulcer are more uniformly satisfactory in the former sex. This at least has been my experience. Again, stomal ulceration is comparatively rare in females after the operation of gastro-jejunostomy (see page 405). Females show a greater tendency to develop anæmia following wide gastric resection, a point which received emphasis from Gordon-Taylor, Morley, and others.

(3) *Physical and Mental Types.* The lean, nervous, anxious-minded young person; the neurotic, the introspective, the visceroprotic, and the obese are all poor subjects for gastric surgery. The result of any type of gastric operation is likely to prove disappointing in patients who have a chronic duodenal ulcer combined with a marked degree of visceroptosis. Wilkie considers that where operation is advisable in such cases, gastro-duodenostomy will usually afford better results than gastro-jejunostomy.

(4) *Occupation, Economic Status, and Habits.* There is a high incidence of "irritable" duodenal ulcer with hyperchlorhydria in the rushed, restless, anxious business man, the doctor, the harrister, and those whose mental activities are always at key pitch. They are, on the whole, unsatisfactory cases for either medical or surgical treatment, as their habits and mode of life hinder their paying the necessary attention to the details of medical treatment and, in addition, most of them belong to a group which is characterised by hypermotility and rapid emptying of the stomach.

It is often found that a holiday with sunshine, relaxation, freedom from mental strain, and a change of occupation will bring about considerable improvement or even a cure much more successfully than any other form of treatment.

Farmers, the phlegmatic type of patient, clerks and others in sedentary occupations usually show good results from treatment, whether medical or surgical.

As has been stressed by Lord Moynihan, Tyrrell-Gray, Hurst, Ryle and others, tobacco is the worst enemy of patients suffering from duodenal ulcer. Moynihan carried out many investigations on medical students and others at a time when they were smoking and at a time when they were not, and found definite evidence of an increased acidity of the gastric juice as a result of smoking. Therefore, to operate upon a patient with a chronic duodenal ulcer and to allow him to smoke again without restraint would strongly predispose him to a reactivity of ulceration or even to the development of a secondary ulcer. Continued indulgence in alcohol and overeating are also regarded as deterrents to the success of treatment.

(5) *Family History.* We have shown elsewhere that with certain patients there is a family history of duodenal ulcer. There are also certain persons who show a congenital and familial predisposition to hæmorrhagic duodenal ulcer, or even to jejunal ulcer following gastro-jejunostomy. Unfortunately such cases do not respond readily to medical measures, and their predisposition to a recurrence of hæmorrhage and to the formation of stomal ulcer following gastro-jejunostomy make it imperative that, when operation is performed, nothing short of a sub-total gastrectomy should be undertaken, as this alone ensures absence or such a low concentration of acid that ulceration does not recur.

(6) *General Condition.* Patients who suffer from chronic ill-health, chronic pulmonary conditions, or other complicating diseases should be treated medically, and operation be undertaken only in the presence of urgent complications.

(7) *Length of Ulcer History.* Patients giving a short history of duodenal ulcer should be treated medically. The same cannot, however, be said of the patient who gives a short history of digestive upset and on X-ray examination is found to have a large chronic "gastric" ulcer, as in such a case it is exceedingly difficult to exclude the possibility of malignancy.

But in a general way it may be said that patients with chronic ulcer giving a *short history*, i.e. less than 3 years, will usually respond to dietetic and medicinal treatment, whereas those giving a *long history*, i.e. more than 6 years, will usually sooner or later require surgical measures.

The importance of chronicity as regards the prognosis of *medical* treatment has been stressed by Nielsen (*Acta. Med. Scand.*, lviü, I, 1923), who states that if symptoms have been present for not more than 1 year, freedom from relapse can be guaranteed in at least 54-60 per cent of cases, or, including improved patients, 60-70 per cent. If, however, the symptoms have been present for more than 5 years, the probability of producing a cure is about 10 per cent only.

Farquharson (*B.M.J.*, p. 144, Jan. 26, 1935), writing on the effect of chronicity on results is of the opinion that, judging by his investigation of a series of cases treated both medically and surgically, the average duration of symptoms prior to treatment bore striking relation to the results obtained. He found, for instance, that *the best results from both forms of treatment were seen in those cases who had a relatively short history of ulcer symptoms*. In the cases completely cured by medical methods the average duration of symptoms prior to treatment was  $2\frac{3}{4}$  years, whilst the corresponding figure for the surgical cases was  $4\frac{1}{2}$  years. In the group of "fair" results the average duration of symptoms was  $6\frac{1}{2}$  years in those treated medically and  $8\frac{1}{2}$  years in those submitted to operation. He gives the following table:

Results.	Average duration of symptoms prior to treatment.	
	Medical.	Surgical.
Very good . . . .	$2\frac{3}{4}$ years.	$4\frac{1}{2}$ years.
Satisfactory . . . .	$5\frac{1}{2}$ " .	6 " .
Fair . . . . .	$6\frac{1}{2}$ " .	$8\frac{1}{2}$ " .
Poor . . . . .	11 " .	$9\frac{1}{2}$ " .

(8) *Radiological Findings.* Both as regards the diagnosis and the choice of treatment in a case of chronic peptic ulceration, the clinician is largely dependent upon efficient and correctly interpreted X-ray investigations of the stomach and duodenum, undertaken by a skilled radiologist. The radiologist can often accurately determine the size of an ulcer when present, i.e. whether it is large—over 1 inch in diameter, of the "letter-box" type, or small—the "threepenny



piece" ulcer. The larger ulcers, those over 1 inch in diameter, are generally accepted as being malignant.

A chronic ulcer which has an *irregular outline*, and particularly if the mucous membrane in the vicinity appears "granular," strongly suggests the possibility of carcinoma.

The *position* of the ulcer may influence the choice of treatment. The very rare ulcer situated on or near the greater curvature calls for immediate resection as in almost every instance such a lesion will prove to be carcinomatous; an ulcer situated in the pyloric vestibule or canal should always be regarded with the gravest suspicion in view of the possibility of malignancy, and in the majority of these cases also radical surgical measures are required.

*Anchorage denotes chronicity.* It is very unlikely that medical treatment will be successful in the case of a large chronic ulcer which has perforated all the coats of the stomach and deeply eroded the pancreas or liver, and here partial gastrectomy will often prove to be the only means of cure.

The demonstration by skiagrams of gross *anatomical deformities of the stomach*, such as occur in pyloric obstruction and hour-glass stomach, calls for no special comment here; the choice of operative procedures in such cases has already been discussed.

The *motility* of the stomach and a rapid emptying rate or delay in emptying, may furnish important evidence when taken in conjunction with other factors such as excess, diminution, or absence of hydrochloric acid in the gastric juice. It is better, for instance, to persevere with medical treatment for the non-obstructive types of chronic duodenal ulcer associated with hyperacidity and a rapidly emptying stomach, for, as previously pointed out, this type of case is very prone to develop jejunal ulceration following a short-circuit operation. On the other hand, in cases of duodenal ulcer associated with stenosis and marked delay in emptying and in which there is a relatively low acidity, stomal ulceration is very unlikely to occur, and here gastro-jejunostomy yields a striking percentage of highly satisfactory results.

In the obstructive type of case medical treatment, although producing some temporary amelioration of the symptoms, very rarely effects a cure.

(9) *Results of Previous Medical Treatment.* Enquiry will have to be made as to previous medical treatment, especially whether this has been carried out with due care and given a fair trial. Repeated

failures with such treatment will suggest that the ulcer is callous, and in such cases surgical interference is indicated before complications arise.

The persistent recurrence of symptoms following medical treatment—i.e. failure of medical treatment—indicates the advisability of operation.

(10) *Complications.* Such complications as pyloric stenosis, hour-glass stomach, repeated hæmorrhages, perforation, etc., call for surgical measures.

(11) *Gastric Acidity.* There seems to be little doubt that operations performed for chronic peptic ulcer show a far higher percentage of good results in patients with low acidity than in those in whom the acidity is high. Failures in operative treatment are due partly to faulty operative technique, but perhaps most of all to the development of fresh ulceration in the region of the new anastomosis, which is so prone to occur in patients with the hypersthenic gastric diathesis where hyperchlorhydria is a constant feature.

Hence it is most important to consider the test meal findings, paying particular attention to the amount of hydrochloric acid in the gastric juice, when making a decision as to treatment. Repeated gastric analysis now forms such an essential part in the investigation of patients suffering from chronic peptic ulceration that this should never be omitted before treatment, either medical or surgical, is instituted.

### (C) INDICATIONS FOR OPERATION

#### (1) *Chronic Gastric Ulcer.*

- (a) Perforation.
- (b) Pyloric stenosis.
- (c) Hour-glass stomach.
- (d) Repeated hæmorrhage in spite of medical treatment.
- (e) Persistent recurrence of symptoms after adequate medical treatment—i.e. failure of medical treatment.
- (f) Inability of the patient to follow the suggested lines of treatment owing to the nature of his work.
- (g) Anchorage of a large callous penetrating ulcer to some neighbouring organ such as the pancreas or liver.
- (h) Multiple chronic ulcers.

- (j) Recurrent symptoms following the operation of closure of a perforated gastric ulcer.
- (k) Chronic ulcer associated with chronic duodenal ileus, duodenal bands or adhesions.
- (l) Chronic ulcer where carcinoma is suspected.
- (m) Expedient circumstances or economic reasons in very rare cases.
- (n) A gastric ulcer which has failed to heal after gastro-jejunostomy and subsequent courses of strict medical treatment.

(2) *Chronic Duodenal Ulcer.*

- (a) Perforation.
- (b) "Pyloric stenosis"—duodenal stenosis.
- (c) Recurrent hæmorrhages.
- (d) Failure of adequate medical treatment.
- (e) Recurrent symptoms after the operation of closure of a perforated duodenal ulcer.
- (f) Chronic penetrating duodenal ulcer associated with chronic gastric ulcer.
- (g) Chronic duodenal ulcer associated with chronic duodenal obstruction.
- (h) Expedient circumstances and economic reasons in very rare cases.
- (j) Recurrence of ulceration following operation.

(D) CHOICE OF OPERATION AND RESULTS

*There is no one universally accepted and practised operation for the cure of chronic gastric or duodenal ulcer.*

The success of any particular operation depends largely upon the skill and experience of the surgeon by whom it is performed. By dint of practice one surgeon may excel in one type of operation, whilst another may perfect his technique in some other which, in his hands, proves equally successful. Thus, as emphasised by Walton, the percentage of successes and failures in gastric surgery cannot be justly assessed on the results of one single type of operation, as so many different factors are concerned in the final issue.

From the results he has obtained from the various operations he has performed, the surgeon is in a position to give his patient a fair idea

of the degree of success to be expected from one or other of the methods he may advocate.

Operations upon the stomach and duodenum for chronic peptic ulcer may be grouped as follows :

- (1) Simple excision of the ulcer.
- (2) Short-circuit operations with or without excision of the ulcer.
- (3) Partial gastrectomy.

*Simple excision of the ulcer* as the treatment of chronic peptic ulcer has been abandoned, as recurrence of ulceration so frequently follows this procedure.

*Gastro-jejunostomy* and, to a lesser degree, other short-circuit operations such as gastro-duodenostomy, hold a prominent place in the treatment of certain cases of simple ulcer, but the results of all such operations are enormously enhanced when removal of the ulcer itself is also undertaken.

It is almost universally agreed that gastro-jejunostomy, combined with infolding of the scarred region or excision of the ulcer, is the operation of choice for simple pyloric stenosis. Gastro-jejunostomy would also appear to be indicated in certain instances where the ulcer is situated high up on the lesser curvature near the cardiac orifice (in which position it may be fixed to the pancreas or liver), when excision of the ulcer or sub-total gastrectomy would be fraught with too great danger (fig. 119).

*Partial gastrectomy* is advocated for the majority of chronic gastric ulcers, wedge excision plus gastro-jejunostomy being reserved for those cases where the ulcer is small, where anatomical distortion of the stomach is minimal, and where the parts concerned readily admit of such a procedure. But even in such cases, where malignancy is suspected or the acid curve is abnormally high, partial gastrectomy is to be preferred.

For the rare ulcer situated in the pyloric segment partial gastrectomy is the operation advised, and we would agree with Joll that *ulcers which begin at or within one inch of the gastric aspect of the pylorus are very rare* and that nearly all cases of cicatricial stenosis of the pylorus are due to duodenal ulcers, the scar tissue resulting from which has spread to the left so that the exact site of *origin* of the ulcer is obscured.

Joll writes :

“I have my contention on an examination of specimens removed by duodeno-pylorotomy for pyloric stenosis associated with a high

hydrochloric acid curve. Among 916 operations for gastric and duodenal ulcer I have found only four chronic gastric ulcers which could definitely be classed as pyloric, but I have in addition performed duodeno-pylorotomy in 43 cases, diagnosed radiologically as due to pyloric ulceration, in which the original site of the ulcer proved to be the posterior surface of the duodenum and within a quarter of an inch or so of the pylorus.

"If this view of mine is correct, statistics relating to pyloric gastric ulcer should properly be relegated to the category of duodenal ulcers, as the actual percentage of pyloric ulcers is so small." (*Proc. Roy. Soc. Med.*, No. 6, p. 1286, July, 1931.)

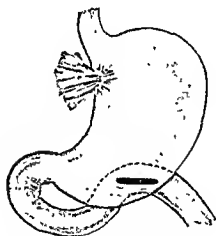


Fig. 119.—GASTRO-JEJUNOSTOMY FOR LARGE ULCER SITUATED NEAR THE CARDIA AND PENETRATING THE LIVER.

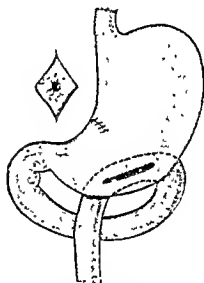


Fig. 120.—SMALL GASTRIC ULCER SITUATED ABOUT THE MIDDLE THIRD OF THE LESSER CURVATURE. WEDGE EXCISION OF ULCER COMBINED WITH GASTRO-JEJUNOSTOMY HAS BEEN PERFORMED.

At the present time there is much controversy with regard to the choice of operation in cases of chronic duodenal ulcer without stenosis where surgery is indicated on account of recurrent hæmorrhage or severe symptoms which cannot be controlled by medical measures. There is no doubt that while a short-circuit operation, by diverting acid from the ulcer site, will bring about a cure of the ulcer itself in the majority of cases, it will do nothing whatsoever to counteract the factors which influenced the production of the ulcer. In fact, a short-circuit operation increases the rapidity with which the stomach empties, thus diminishing the opportunity of neutralisation of the acid gastric con-

tents, and thereby predisposing to the formation of fresh ulcers in the region of the stoma.

There is only one sure method by which the secretion of acid gastric juice can be permanently reduced to a lower level than that at which peptic ulceration can occur, and that is by partial gastrectomy, entailing the removal of at least three-quarters of the stomach.

The encouraging results so far obtained in my own series of cases seem to justify this procedure, mutilating though it may have appeared to be for such a small lesion.

But the main objection to the routine performance of partial gastrectomy in preference to gastro-jejunostomy for chronic duodenal ulcer without stenosis is the mortality of the former operation which is said to be about 5-6 per cent, whereas that of the short-circuit operation is only 1-2 per cent.

"But if the operation is necessary it must be made safe, and I am perfectly certain that the surgeon who can produce a mortality of 1 per cent in gastro-jejunostomy can show as low a figure for physiological gastrectomy." (Ogilvie, *Lancet*, p. 419, Feb. 23, 1935.)

A comparison between the end-results of gastro-jejunostomy and partial gastrectomy will show that although the former operation is capable of producing a fair proportion of brilliant immediate results, the latter will yield a higher percentage of permanent cures and a degree of general fitness and good health far surpassing that afforded by any short-circuit operation.

#### CHOICE OF OPERATION FOR CHRONIC GASTRIC ULCER

- (1) Operations for Chronic Ulcer situated in the Body of the Stomach.
- (2) Operations for Chronic Ulcer situated in the Pyloric Segment of the Stomach.
- (3) Operations for the Complications of Chronic Gastric Ulcer.

#### *Operations for Chronic Ulcer situated in the Body of the Stomach.*

(a) Small ulcer on or near the lesser curvature—acid curve within normal limits. The treatment here recommended is excision of the ulcer or its destruction by the cautery combined with gastro-jejunostomy (fig. 120). Where, however, malignancy is suspected, partial gastrectomy should be undertaken.

It would be a very easy solution to the problem if *simple excision*

of the ulcer could guarantee a cure in every case; but unfortunately this operation is followed by recurrence of ulceration in a very high percentage of cases, as is shown in the B.M.A. Report (1930), where it is stated that local excision of the ulcer by itself gives very poor results, i.e. approximately 50 per cent of failures. Hurst states that recurrence occurred in 4 out of 10 of Dobson's cases within a year; Sherren had 6 failures out of 9 cases; and Collinson had 15 failures out of 39 cases.

It is an unsatisfactory operation, as the motor functions of the stomach are often impaired thereby and, apart from removing a possible source of malignancy, conditions in the stomach are not materially improved, the acidity is not diminished, and the mucous membrane surrounding the sutured area is more liable to ulcerate than any other part of the stomach.

*Gastro-jejunostomy without excision of the ulcer* is seldom advisable, the failures by this method being estimated at over 35 per cent. When, however, excision of the ulcer is followed by a short-circuit operation the results are definitely better. Walton, for instance, in a series of 310 patients treated by excision of the ulcer, pyloric occlusion, and posterior transverse gastro-jejunostomy, had 13 deaths—a mortality of 4.2 per cent, and in a group of these cases which were watched for a minimum of 5 years there were 88 per cent of complete cures. Of those cases which survived 6 developed recurrent ulcers, 5 showed the later onset of carcinoma, and 1 developed a severe form of anæmia.

(b) Small ulcer on or near the lesser curvature—acid curve abnormally high. In such cases partial gastrectomy is preferable to wedge excision plus gastro-jejunostomy.

(c) Large or medium-sized ulcers of the body of the stomach situated on or near the lesser curvature (fig. 121). Some of these may be adherent to the pancreas or liver. For several reasons partial gastrectomy is the method of choice in these cases:

(i) Complications such as the onset of malignant degeneration of the ulcer, perforation, hour-glass stomach, hæmorrhage, etc., are prevented. If, on the other hand, as proved by subsequent microscopical examination, the ulcer is malignant, this operation has already afforded the most radical and effective type of treatment. Where operation is undertaken for chronic gastric ulcer the development of gastro-jejunal ulcer is rarer after partial gastrectomy than it is after gastro-jejunostomy.

(ii) The technique of this operation greatly simplifies the removal of these large ulcers. On the other hand, wedge excision of the ulcer

presents many technical difficulties: the resulting aperture is not easy to close, and the suturing produces considerable distortion of the stomach and interference with its functional capacity.

(iii) The results of this operation are eminently satisfactory. About 90 per cent of patients show unimpaired efficiency for work and have nothing of which to complain except that they must eat moderately for the first few months after the operation. The unsatisfactory results comprise less than 10 per cent, and include a few cases which develop anæmia of the chronic microcytic or, very rarely, of the macrocytic type.

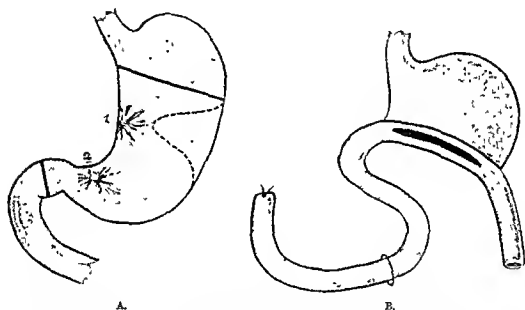


Fig. 121.—A 1. LARGE ULCER OF THE LESSER CURVATURE (WITH OR WITHOUT HOUR-GLASS CONSTRICTION). A 2. CHRONIC ULCER SITUATED IN THE PYLORIC SEGMENT. FOR BOTH THESE CONDITIONS PARTIAL GASTRECTOMY B IS RECOMMENDED

Some leading surgeons give a very low death-rate from this operation, e.g., Lord Moynihan 1.6 per cent. Finsterer in a series of 407 cases had a mortality of 4.4 per cent, but during the last few years this, in his hands, has fallen to 1.8 per cent; Gordon-Taylor 4.2 per cent; whilst von Hahner has been able to reduce his mortality from 8.4 to 6 per cent. But the average operative death-rate from partial gastrectomy for chronic gastric ulcer is found to vary from 6–8 per cent.

In my own cases the mortality of partial gastrectomy in the class of ulcer suitable for wedge resection is under 4 per cent.

(iv) It is accepted by the majority of abdominal surgeons as being the most satisfactory and successful operation for such cases.



(d) Inaccessible or "letter-box" ulcers occurring near the cardia. Here we would recommend gastro-jejunostomy in preference to jejunostomy, cholecysto-gastrostomy, or sub-total gastrectomy.

Ulcers situated high up on the lesser curvature near the cardiac orifice, sometimes penetrating the pancreas or liver to a great depth, are often associated with much pain, recurrent hæmorrhage, vomiting, loss of appetite, and consequent debility, and are most unsuitable for operation.

It is in just these cases that medical treatment often proves unavailing. Partial gastrectomy cannot be recommended as it is a most hazardous procedure under the circumstances, often entails a sub-total resection of the stomach, and is frequently followed by a high percentage of deaths.

*Cholecysto-gastrostomy*, which has at times been advised and practised for this type of case, often has little or no effect in the healing of the ulcer. It has been advocated on the assumption that the bile entering the stomach will have a neutralising effect upon the gastric juice; but bile is a neutral or only faintly alkaline fluid and it is very questionable whether the amount which intermittently enters the stomach is sufficient to cause neutralisation. This is confirmed by Nasarov who investigated a number of cases upon which this operation had been performed. He showed that the reduction of the gastric acidity was very much less than might have been expected, and that hyperchlorhydria often persisted.

In the treatment of this type of case *jejunostomy* has its supporters, but is generally considered to be very disappointing. It is claimed that by the institution of jejunal feeding the stomach is put at rest and the ulcer is given a chance to heal. Unfortunately the stomach cannot be put at rest. As shown by Ivy, the mere thought or sight of food stimulates a copious secretion of gastric juice, and the introduction of food into the jejunum produces the same effect. If no food passes into the stomach whereby the gastric juice may become neutralised or diluted, this juice in its full concentration has direct access to the ulcer and conditions are thus favourable for its continuance and extension.

If, therefore, this operation is performed for the type of ulcer under discussion, the jejunal feeding should be accompanied by the oral administration of alkalis, olive oil, and large doses of atropine, as recommended by Hurst, in order to neutralise and inhibit the secretion of gastric juice.

A jejunostomy very often interferes with and greatly complicates any future operation upon the stomach, such as partial gastrectomy.

I content myself, therefore, in such cases with the performance of a gastro-jejunostomy, fully realising that it is whimsical in its effects and that a further operation for excision of the ulcer will be found to be necessary in many instances.

*Operation for Chronic Ulcers situated in the Pyloric Segment of the Stomach.* Ulcers situated in the pyloric segment of the stomach, i.e. between the pyloric outlet and the incisura, in view of their so often proving malignant, should be treated by gastro-duodenal resection.

*Operations for the Complications of Chronic Gastric Ulcer.* (a) Hour-glass stomach (see page 266). Partial gastrectomy is the treatment recommended for this condition. As has been shown, the results are very good, and in actual practice the operation is feasible in the majority of cases, being attended by a low mortality.

In certain instances where the patient is in poor condition and is unable to stand a severe operation, gastro-gastrostomy combined with some form of pyloroplasty constitutes a satisfactory alternative form of treatment, and particularly in long-standing cases where there is evidence that the ulcer has healed and that the stricture is situated not too high up in the body of the stomach.

(b) Pyloric stenosis (see page 282). Pyloric stenosis due to simple ulcer should be treated by gastro-jejunostomy.

(c) Perforation (see page 220). Simple closure of the perforation, with or without drainage of the pelvis, is advocated in the majority of cases. Gastro-jejunostomy is advised only where the patient's condition is good, where the perforation is recent, and where the closure of the perforation has produced pyloric obstruction.

(d) Hæmorrhage (see page 578). Where possible, operative treatment is best postponed until bleeding has ceased. Where operation has been undertaken and the hæmorrhage is found to be due to the erosion of an artery in the base of a gastric ulcer, the most appropriate treatment in the circumstances is partial gastrectomy. Such procedures as underrunning and tying the blood-vessels in the vicinity of the ulcer, or trans-gastric exposure of the crater and its attempted obliteration with through-and-through sutures are unsatisfactory as they are very liable to be followed by a recurrence of the hæmorrhage, even should they succeed in temporarily controlling it.

(e) Fistula (see page 257). In cases of *gastro-colic fistula* due to a chronic gastric ulcer (a rare condition), Wilkie's two-stage operation

should be practised. This consists of excluding the portion of colon involved in the fistula, re-establishing the continuity of the colon by end-to-end anastomosis, and allowing a period of some weeks or even months to elapse before undertaking the second and major stage of the operation. In many cases this will amount to partial gastrectomy, a segment of stomach and the ulcer, together with the attached portion of the excluded loop of colon, being removed in one piece.

#### CHOICE OF OPERATIONS FOR CHRONIC DUODENAL ULCER

- (1) Operation for Chronic Duodenal Ulcer WITH Stenosis.
- (2) Operations for Chronic Duodenal Ulcer WITHOUT Stenosis.
- (3) Operations for the Complications of Chronic Duodenal Ulcer.

*Operation for Chronic Duodenal Ulcer WITH Stenosis.* The operation universally advised in such cases is infolding or excision of the scarred portion of the duodenum, followed by gastro-jejunostomy.

The results of this operation are excellent, from 90-95 per cent of the patients being completely cured, the complication of stomal ulceration being rare (under 1 per cent), and the death-rate being only 1-3 per cent; in fact, this constitutes one of the most successful of abdominal operations, and for this type of case it is unsurpassed in its splendid results.

*Operations for Chronic Duodenal Ulcer WITHOUT Stenosis.* A variety of operations are practised for this condition :

- (a) *Pyloroplasty.*
  - (i) Horsley's operation.
  - (ii) Judd's operation.
- (b) *Gastro-duodenostomy.*
  - (i) Finney's operation.
  - (ii) Jaboulay's operation.
- (c) *Gastro-jejunostomy with excision, cautery destruction, or infolding of the ulcer.*
- (d) *Partial gastrectomy.*
  - (i) The Péan-Billroth I operation.
  - (ii) The Polya types of partial gastrectomy—anterior and posterior.
  - (iii) Finsterer's operation of partial gastrectomy with pyloric exclusion—"physiological gastrectomy."

There can be no doubt that in Great Britain excision or infolding of the ulcer, followed by gastro-jejunostomy, is still the method preferred by a large number of surgeons for cases of chronic duodenal ulcer in which there is no stenosis. But the need for much careful consideration in the choice of operation for such cases must again be stressed, as the results of the routine performance of gastro-jejunostomy, whether combined with excision or with infolding of the ulcer, although carrying the low mortality of only 1-2 per cent, show a comparatively high incidence of secondary peptic ulceration, i.e. 6-8 per cent, while published statistics would indicate that the late results are unsatisfactory in over 25 per cent of cases.

These results have led to a marked increase in the practice of "physiological gastrectomy" for this type of case.

Where the patient gives a long history of chronic duodenal ulcer (often associated with intermittent hæmorrhages); where there is marked hyperacidity, hypermotility, and rapid emptying of the stomach; where courses of adequate and efficient medical treatment have *repeatedly* failed; where at operation the ulcer is found to be large and deeply penetrating, the duodenum being entangled in a mesh of oedematous and inflammatory adhesions—by no means an uncommon combination—Finsterer's operation of partial gastrectomy with pyloric exclusion would appear to be the method of choice to ensure healing of the ulcer and such a diminished secretion of acid gastric juice that the subsequent onset of marginal ulceration is prevented.

In the type of case under discussion, *gastro-duodenostomy* has a distinguished if limited following. It is claimed that this operation short-circuits the ulcer-bearing area in the duodenal bulb and deflects the acid gastric contents into the second portion of the duodenum—a region more alkaline in content and more tolerant of acid than the proximal jejunum. It is stated that a certain amount of alkaline duodenal fluid is regurgitated into the stomach, which lowers the gastric acidity and at the same time relieves pyloric spasm.

But, in my opinion, the same adverse criticisms which have been levelled at gastro-jejunostomy can be aimed with equal or even greater force at gastro-duodenostomy also.

In a recent review of Wilkie's gastro-duodenostomy cases at intervals varying from 1-12 years after operation, the results were excellent in 64 per cent; 25 per cent of the patients were relieved but not cured, and in 11 per cent symptoms of dyspepsia persisted. Two cases of stomal ulcer occurred and both received immediate and lasting relief from secondary gastro-jejunostomy. In two cases bilious

vomiting persisted after gastro-duodenostomy, owing to axial rotation of the duodenum due to inadequate mobilisation; here secondary gastro-jejunostomy was followed by a good result.

Wilkie states that the ultimate results have been so gratifying after the twofold anastomosis that he has seriously considered performing the double operation in cases with high acidity and a somewhat fixed duodenum. He draws attention, however, to the importance of ascertaining whether or not there is any duodenal ileus in cases of gastric and duodenal ulcer which have been operated upon. If, he says, at the completion of any form of gastric anastomosis, there is evidence of duodenal stasis, a duodeno-jejunostomy should be performed. This added procedure prolongs the operation by only a few minutes and prevents any unpleasant symptoms in the immediate post-operative stage and later.

The *Heineke-Mikulicz* operation, which consists of dividing the strictured portion of the gut in an axis parallel to the lumen and closing the opening in a transverse direction, is now never performed, as the remaining scar tissue continues to contract, causing recurrence of the symptoms.

But there are two types of pyloroplasty which are sometimes practised for small anteriorly-situated chronic duodenal ulcers without stenosis.

The first—*Horsley's* "physiologic" pyloroplasty—is a modification of the *Heineke-Mikulicz* method. *Horsley* (*Surgery of the Stomach and Duodenum*, p. 152, Kimpton) writes :

"In 1919 I described a pyloroplasty which was termed 'physiologic' because it seems to have the advantage of removing an ulcer in the first part of the duodenum and restoring the physiologic function of the stomach to normal, without any undue mutilation. The incision is a straight incision, two-thirds of which is in the stomach and one-third in the duodenum. It should never be carried further than an inch into the duodenum, and the proportion of having the portion of the incision in the stomach at least twice as long as the portion in the duodenum should always be preserved (fig. 122). The pyloric canal is about  $1\frac{1}{4}$  inches (3 cm.) long, and this at least should be divided. To be sure, it has in common with the *Heineke-Mikulicz* operation a straight incision which is sutured transversely, but otherwise there is no resemblance (fig. 123). The *Heineke-Mikulicz* pyloroplasty was conceived and performed to overcome a stenosis and it was intended merely to correct the contraction. The 'physiologic' pyloroplasty is not indicated in a marked stenosis. Though I have used it in a narrow stenosis, I am inclined to believe that even this is stretching its indications a little too much into a field where posterior gastro-enterostomy is extremely satisfactory.

The object of this pyloroplasty is to remove an ulcer and to give physiologic rest to the stomach by dividing the pyloric sphincter and the adjacent pyloric canal. It embodies the same principle that is adopted when the sphincter ani is divided or divided for the cure of an ulcer within its grasp. While the operation has a limited

field, and while in my early enthusiasm I applied it more widely than it should have been applied, it still has definite indications, chiefly in the small group of cases in which there is a duodenal ulcer in the first part of the duodenum, without adhesions and without surrounding infiltration, that has resisted the therapeutic efforts of carefully applied medical treatment. Most ulcers of this kind can be cured by medical treatment consisting largely of the proper regulation of diet with the occasional administration of belladonna or its products and, more rarely, of some alkaline powders. In spite of the best medical treatment, however, occasional patients are seen with small limited duodenal ulcers without adhesions, who still have annoying symptoms from the ulcer.

If the adhesions are very limited and only to the gall-bladder, the gall-bladder may be removed at the same time the pyloroplasty is done and this pyloroplasty will usually give good results. If, however, the adhesions are extensive and if there is

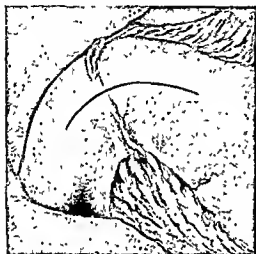


Fig. 122.—HORSLEY'S "PHYSIOLOGIC" PYLOROPLASTY. THE INCISION.  
(After Kellogg)

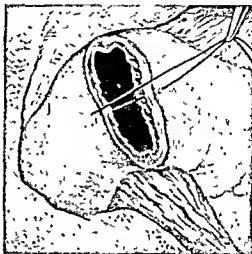


Fig. 123.—HORSLEY'S "PHYSIOLOGIC" PYLOROPLASTY. A TRACTOR SUTURE HELD BY FORCEPS TO FACILITATE THE SUTURING OF THE OPENING IN THE TRANSVERSE AXIS. (After Kellogg)

sub-acute inflammation around the ulcer, as a duodenitis, this pyloroplasty is not so satisfactory and frequently is followed by a recurrence of symptoms. When the adhesions are fairly extensive and a pyloroplasty seems indicated, the Finney pyloroplasty would be better suited to the conditions. If there is a slight stenosis the Finney operation would be better than this physiologic pyloroplasty, but marked stenosis is best treated by a posterior gastro-enterostomy."

The second pyloroplasty which is performed in some 50 per cent of cases of chronic duodenal ulcer at the Mayo Clinic is known as *Judd's operation*.

Judd considers that certain of the symptoms attending duodenal ulcer are due to tension and spasm resulting from the action of the pyloric sphincter muscle, and that the physiological reactions of the stomach and duodenum are changed and the concentration of acids lowered after the performance of his operation.

This operation consists of the mobilisation of the duodenum and the excision of the anterior two-thirds of the pyloric sphincter muscle, the ulcer, and the cap of the duodenum. If an ulcer is found on the posterior wall this may be cauterised or the excision may be extended to include this lesion, leaving only a strip of the posterior wall with its mucous membrane. If there is an area of duodenitis or a small ulcer on the posterior wall, this may be left undisturbed. The wound in the gut is then closed in a transverse direction, constituting a gastro-duodenostomy.

The local operation is really a comparatively simple procedure in cases in which it is indicated. Furthermore, it is not followed by the complications which are too frequently associated with gastro-jejuno-stomy, particularly those of pulmonary origin; indeed, it is unusual for a patient to develop pulmonary complications in association with this plastic procedure.

According to Judd, this operation is particularly indicated if the patient is young and/or of nervous temperament, the ulcer small, scarring minimal, and the pyloric region free from adhesions and easily accessible, as he has not found gastro-jejuno-stomy to give satisfactory results in such cases.

The chief contra-indications to the local operation are anchorage of the duodenum to such an extent that it cannot be mobilised without undue risk in obtaining an approach to the ulcer; considerable narrowing of the duodenum; pouching; and much firm fixation by adhesions.

The main danger—admittedly very rare—following this operation is leakage from the suture line. In the immediate post-operative period retention of gastric contents frequently occurs, necessitating aspiration to prevent distension and vomiting. This delay in emptying of the stomach may persist for a few weeks or even months, but the late results are stated to be very good. Judd had over 90 per cent of complete cures in a series of 1363 cases, with a death-rate of 0.44 per cent. Recurrence of ulceration at or about the suture line occurred in only about 2 per cent of his cases.

Judd therefore considers that when the lesion is limited to a *small* ulcer in the anterior wall of the duodenum, such a pyloroplasty may be recommended with every confidence.

Finney's operation does not appear to possess any distinct advantages over Judd's or Jaboulay's, and, although an excellent procedure, it has not been extensively adopted in this country.

In those cases in which an incision has been made through the

duodenum with the object of performing a pyloroplasty after the method of either Horsley or Judd, and where, owing to the extent of the scarring or to a deeply-eroding posteriorly-situated ulcer, it has been deemed advisable to abandon the operation, it is good practice to excise as much of the scarred portion of the gut as possible and then to close the aperture in the longitudinal axis in such a way that permanent narrowing, almost amounting to complete oecclusion, will necessarily result when the tissues have healed.

Kellogg states that such a method of permanently narrowing the pylorus and duodenum, when followed by gastro-jejunostomy, usually yields satisfactory results.

*Operations for the Complications of Chronic Duodenal Ulcer.*

(a) Perforation (see page 220). In cases of perforation, simple closure, with or without drainage of the pelvis, is indicated. Where suture of the perforation has produced duodenal occlusion, gastro-jejunostomy will be obligatory; but this operation is followed by over 30 per cent of poor late results.

(b) Severe Hæmorrhage (see page 578). If at the time of the operation the patient's condition is poor, the duodenum should be oversewn, the pylorus occluded, and a posterior gastro-jejunostomy performed. If, on the other hand, the patient's condition is satisfactory, partial gastrectomy should be undertaken, as, where this is possible, the results, both immediate and late, will be far superior.

(E) PRE-OPERATIVE TREATMENT

"No patient, however debilitated by emaciation, chronic sepsis, hæmorrhage, or persistent pain, is refused the benefits of operation because he is thought to be a bad surgical risk. Every patient is given a preparatory treatment which lasts for thirty-six hours, but may extend over a week, or two weeks or more, until he has so far responded as to enable him to undergo even the most severe operation with the best chance of success." (Moynihan.)

(1) *The Best Time to Operate.* Before any operation upon the stomach or duodenum is undertaken, a course of preparatory treatment is of the greatest advantage.

In *uncomplicated* cases where the patient is in fair condition, he should be admitted to hospital a few days before operation to accustom him to his surroundings and so that the usual pre-operative medication may be undertaken to render him as fit as possible.



Except where there is gastric retention or an appreciable degree of gastritis, it is unnecessary and meddling to aspirate and irrigate the stomach.

Large quantities of fluids and glucose are given as a pre-operative routine to guard against dehydration and to ensure an adequate supply of glycogen in the liver and muscles.

During this preparatory period there is no need for the patient to be kept constantly in bed; in fact, it is better for him to be up and about for part of the day in order to maintain muscular and circulatory tone and pulmonary ventilation, and to prevent his mind from dwelling unduly upon the thought of his pending operation.

A nutritious semi-solid or liquid diet is prescribed, together with the usual alkaline powders used in the treatment of chronic peptic ulceration.

Where, however, the patient has a *large penetrating lesion*, is exhausted from pain and loss of sleep, and shows evidence of dehydration and emaciation from his abstention from fluids and food, a much longer course of treatment is required, extending over weeks or possibly months. In such cases he must be kept in bed altogether with complete rest and quiet, sleep being ensured by the administration of sedative drugs if necessary. Pain must be relieved by giving olive oil, belladonna, and alkalis, and Hurst's ulcer diet is ordered. The stomach should be washed out once or twice a day with warm normal saline to remove mucus or any decomposing gastric contents, and the reservoirs of the tissues should be flushed with fluids introduced orally, rectally, intravenously, or subcutaneously.

After a few days or weeks of such treatment there will usually be a marked general and local improvement. Considerable absorption of the inflammatory products in the vicinity of the ulcer will take place, gastritis will be diminished, and even a large previously fixed and irremovable ulcer may be rendered resectable by these measures.

If there is marked *anæmia* through continued loss of blood, blood-transfusions will be required, and in such cases operation should be deferred, for several weeks if necessary, until the patient is in a fit condition to withstand it.

Patients suffering from *pyloric obstruction* are never operated upon as emergencies, but a definite plan of treatment is first instituted on the following lines:

(a) Gastric lavage. The gastric contents are aspirated and the stomach is washed out through a Ryle tube 1-3 times a day. Weak

hydrogen peroxide, normal saline, or a solution of hydrochloric acid (0.25 per cent) is usually employed for this purpose.

In cases where there is marked retention and vomiting, the exhaustion of chlorides and the increase in the non-protein nitrogen in the blood and urine, indicate the presence of a severe intoxication (alkalosis). In the early stages of this toxæmia the patient complains of nausea, drowsiness, and headache, and becomes flushed and asthenic, whilst at a later stage there is increased retention of gastric contents, lowered blood-pressure, muscular irritability, prostration, frequent vomiting, oliguria or anuria, gastric tetany, eventually leading to the "typhoid" state and death.

For diagnostic purposes and also to serve as an index of the results of the treatment which has been instituted, it is very important to examine the blood in *all* cases in which there is the least degree of retention in the stomach, to determine the content of chlorides, the urea, and the carbon dioxide combining power of the plasma. In this way comparatively mild cases of toxæmia are frequently discovered before the symptoms have become definitely established.

Tetany is always associated with severe alkalosis and is an indication of marked disturbance of the acid-base mechanism. The painful muscular spasms which occur can to some extent be relieved by the administration of 5 cc. of a 10 per cent solution of calcium chloride or intravenous injections of 10 cc. calcium gluconate (Sandoz) combined with occasional intramuscular injections of morphia and atropine.

(b) The administration of glucose and fluids. 200 cc. of 10 per cent glucose with 1 per cent sodium chloride are given orally from 3-6 times a day in addition to the fluids which are introduced rectally.

I have found the continuous intravenous drip method most satisfactory for this type of case. By controlling the flow to thirty drops per minute, roughly 5000 cc. of fluid rich in salts and carbohydrates can be introduced into the circulation in each 24 hours without discomfort to the patient.

No solid food is permitted during the pre-operative period.

(c) Medicines. As such patients are usually suffering from some degree of alkalosis, the administration of alkalis is forbidden and an acid mixture is substituted.

(2) *Eradication of All Accessible Foci of Infection.* The nose, accessory sinuses, tonsils, teeth, and gums should always be carefully examined, and if any septic focus is found it should be dealt with at

least one month before operation is undertaken. No gastric operation should be performed shortly after the enucleation of tonsils or the extraction of teeth, but ample time, often amounting to a period of 4-6 weeks, should be allowed for healing to take place. If, however, through lack of response to medical treatment the patient's condition is desperate and obstruction must be relieved without undue delay, the removal of such foci must be deferred until he is convalescing from his operation.

It should be remembered that there are at least two other likely sources of infection—the appendix and the gall-bladder. During the performance of a gastric operation the appendix should, if possible, be removed in most cases; the gall-bladder also should be carefully examined, and if found to be diseased cholecystectomy should be performed wherever circumstances permit.

(4) *Sunlight Treatment.* A course of sunlight treatment is often given with beneficial results.

#### (F) TECHNIQUE OF THE OPERATIONS FOR CHRONIC PEPTIC ULCER

##### (1) *General Considerations.*

(a) *Anæsthesia* (see pages 55 and 58).

(b) *Skin disinfection.* The skin may be prepared in various ways, but my own preference is for lavish paintings with *tannin-alcohol* (acid tannic 7.5 per cent in industrial spirit, coloured with fuchsin), tinct. metaphen (Abbott), or industrial spirit followed by dettol.

The prepared area should extend from the nipples to the thighs.

(c) *Abdominal incisions* (fig. 124). The following are the four most usual incisions:

(i) *Mid-line incision.* This is made in the mid-line and extends from the left of the xiphisternum to the umbilicus. When the extra-peritoneal tissues are reached it is important to free these structures from the recti muscles in order to carry the incision through the peritoneum, well away to the right or to the left so as to avoid the ligamentum teres. This incision is closed by Wilkie's method (fig. 125).

This mid-line incision is frequently employed in emergency work, and for most gastric operations.

(ii) *Right paramedian incision.* This is commonly used in gastric surgery, and affords an excellent approach to the pylorus, duodenum, gall-bladder and bile-ducts. It may be advisable to divide the ligamentum teres in order to obtain a better view of the cardiac end of the stomach. Through it the whole abdomen can be readily explored, and where it is deemed necessary to remove the appendix the cæcum can often be drawn through the lower portion of the wound.

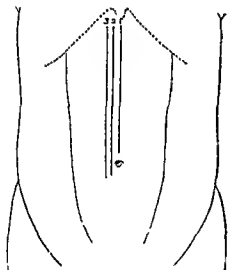


Fig. 124.—INCISIONS FOR OPERATIONS UPON THE STOMACH AND DUODENUM.

- 1 = MID-LINE.
- 2 = PARAMEDIAN.
- 3 = TRANSRECTUS.

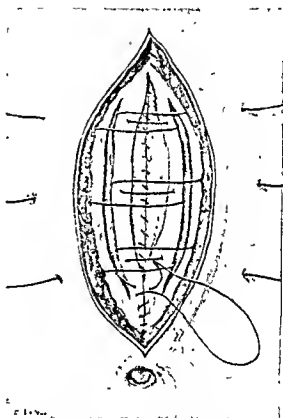


Fig. 125.—A METHOD OF CLOSING A MID-LINE EPIGASTRIC INCISION. (After Wilkie.)

This incision is a vertical one situated 1 inch from the mid-line and extends from the costal margin to a point 1 inch or so below the umbilicus (fig. 126). The subcutaneous tissues and the anterior sheath of the rectus are divided in the same line, and after the belly of the muscle has been dissected free from its inner attachments, the posterior sheath and peritoneum are also divided in the same line as the anterior sheath. The tendinous intersections will have to be very carefully separated, and pains must be taken not to fray or otherwise damage the inner border of the rectus muscle during this dissection.

(iii) *Right transrectus or muscle-split incision.* This incision, like the paramedian, extends from the costal margin to 1 inch below the umbilicus at about 1 inch from the mid-line, all the structures of the anterior abdominal wall being divided in this plane. The muscle-split incision is easier to make and to close than the paramedian, but it is invariably followed by some atrophy and weakness of the inner, and sometimes of the outer, fibres of the right rectus muscle.

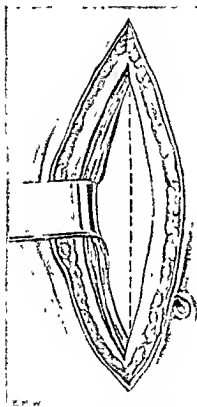


Fig 126.—PARAMEDIAN INCISION. THE RECTUS MUSCLE IS RETRACTED OUTWARDS. THE DOTTED LINE INDICATES THE SITE OF THE INCISION THROUGH THE PERITONEUM

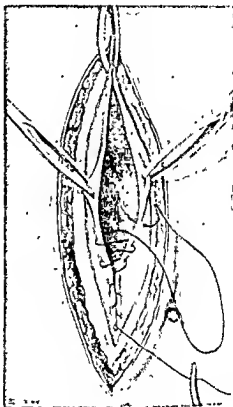


Fig 127.—SUTURE OF THE MARGINS OF THE PERITONEUM BY A "BASEBALL" OR "OCT IN OUT IN" STITCH.

(iv) *A left paramedian or muscle-split incision* is sometimes necessary where a sub-total or total gastrectomy is contemplated. If wider access proves necessary the lateral muscles of the abdominal wall may be divided across transversely and the lower costal cartilages mobilised, as advocated by Marwedel (see fig. 276).

In closing these incisions the peritoneum must be very securely approximated, no gap being left between the individual stitches (fig. 127).

The abdominal wall is usually closed in layers with continuous

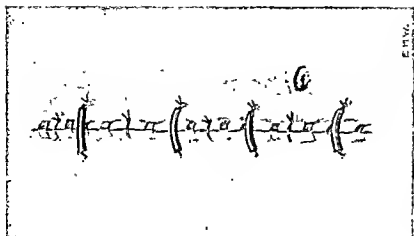


Fig. 128.—A Method of Closing the Incision. The Wound is Closed by Deep Tension Sutures which are Threaded through Fine Rubber Tubes which Protect the Skin when the Sutures are Tied. Michel Clips and a Few Interrupted Sutures of Fine Silk are Put are Introduced with Mathematical Precision. The Clips are Removed on the Fifth Post-operative Day. The Skin Sutures on the Nerve, and the Deep Sutures on the Tenth.

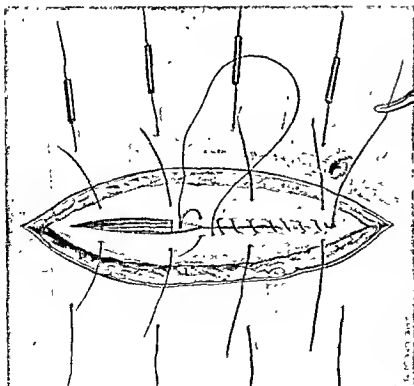
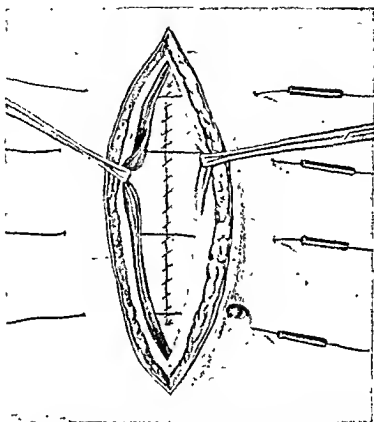


Fig. 128.—SUTURE OF THE ABDOMINAL WOUND. THE INCISION IN THE ANTERIOR SURFACE OF THE RECTUS IS BEING CLOSED BY A CONTINUOUS SUTURE OF NO. 1 TWENTY DAY CHROMIC CATGUT. A FEW INTERRUPTED SUTURES ARE ALSO SHOWN.

sutures of No. 1 or No. 2 20-day chromic catgut, and the skin is united with interrupted sutures of fine silkworm-gut and Kifa or Michel clips (figs. 128 and 129).

Tension or supporting sutures are occasionally inserted as a precautionary measure (fig. 130).



*Fig. 130*—CLOSURE OF THE WOUND. THE PERITONEUM HAS BEEN CLOSED BY A CONTINUOUS SUTURE OF NO. 1 TWENTY-DAY CHROMIC CATGUT, AND FOUR DEEP OR TENSION SUTURES, THREADED THROUGH SMALL RUBBER TUBES, HAVE BEEN INTRODUCED. THESE ARE TIED WHEN THE SKIN INCISION HAS BEEN SUTURED.

The larger gastric vessels and portions of omenta are tied off with No. 2 chromic catgut and whilst, as a rule, No. 0 or No. 00 20-day chromic catgut is used for intestinal suturing (always for the inner hæmostatic suture), fine silk or thread is sometimes used for the outer sero-muscular sutures and for reinforcing the suture line. I never use silk or thread for the mucosal suture, except perhaps in certain operations for cancer of the stomach.

(d) *Armamentarium.* The various forceps, clamps, etc., commonly employed by the author for gastric operations are illustrated in figures 131, 132 and 133.

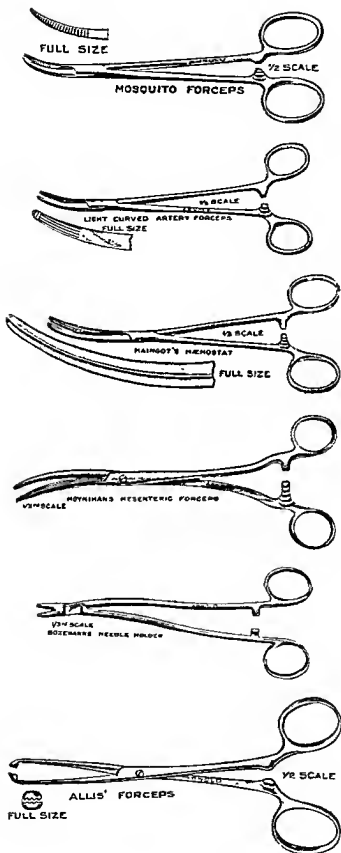


Fig. 131A.—FORCEPS USED IN GASTRIC SURGERY.



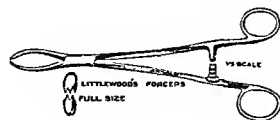


Fig. 131R.—FORCEPS USED IN GASTRIC SURGERY.

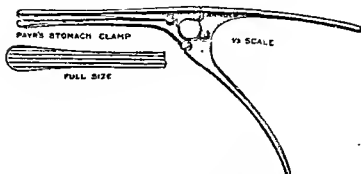


Fig. 132.—THE PAYR CLAMP.

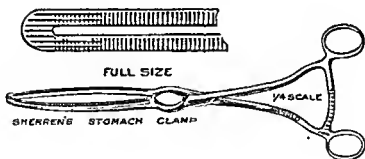


Fig. 133.—THE SHERREN CLAMP.

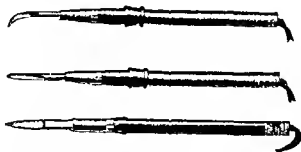


Fig. 134.—POST ELECTRIC CAUTERY, THREE TYPES OF BLADES COMMONLY EMPLOYED IN GASTRO-INTESTINAL SURGERY.

It is of advantage to have a *Post electric cautery* (fig. 134) and a *suction apparatus* at hand during the performance of most operations upon the stomach and duodenum.

The *de Petz clamp* is a very useful instrument in cases of gastric resection (fig. 135).

It somewhat resembles a Payr enterotribe, except that the upper blade is much larger, being adapted to hold a double row of fine staples, and there is a ratchet by means of which the staples are forced through the tissues and turned over to seal off the crushed portion of the gut with a double row of clips before it is divided. The staples replace the first row of intestinal sutures and are inverted into the gut with a series of interrupted Lemmert sutures. These tiny clips eventually

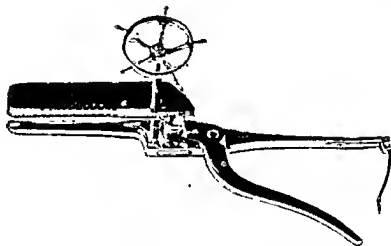


Fig. 135.—THE DE PETZ CLAMP.

slough out, and as their ends are turned under they pass into the intestines, causing no damage, and are discharged per rectum. Sometimes, however, they may remain imprisoned in the tissues for a considerable period without giving rise to any untoward symptoms.

The de Petz clamp is helpful in that it saves time in suturing, prevents soiling of the peritoneum, and ensures complete hæmostasis.

The *Friedrich-Petz clamp*, which is more serviceable than the original Petz instrument, carries clips in separate magazines so that it can be used for several suture lines in succession (fig. 136).

(e) *Hæmostasis*. In all gastric operations hæmostasis must be complete, virtually no blood being lost from the commencement to the completion of the operation, even the minutest bleeding points in the subcutaneous tissues being picked up and tied off with the finest silk or plain catgut ligatures.

In gastro-intestinal anastomoses, by carefully introducing the

through-and-through all-coats suture, by placing the individual stitches exactly vertical to the suture line so that they lie almost side by side and not more than  $\frac{1}{8}$  inch apart, and by drawing each stitch sufficiently tight, the possibility of hæmorrhage is practically eliminated.

I consider that here only two layers of sutures are necessary, although many surgeons still show a preference for three layers, claiming that hæmorrhage is thereby avoided and that the risk of stomal ulceration is diminished. But although the risk of bleeding into the lumen of the gut may be thus reduced, hæmorrhage may nevertheless

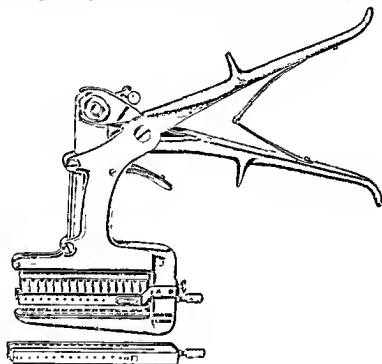


Fig. 136.—THE FRIEDRICH PETZ CLAMP.

occur in the space between the submucosa and the muscular coat, giving rise to a hæmatoma which, should infection supervene, may possibly result in an abscess which may rupture into the gut and thus predispose to the formation of an anastomotic ulcer.

If a loop-on-the-mucosa suture (first described by C. H. Mayo and Connell) is used, *the individual loops should always be placed very close together* and the suture be reinforced with a continuous Lembert suture (fig. 137). Great pains must be taken in turning the corner when this stitch is employed, as it is at this point that hæmorrhage is most likely to occur.

For gastro-intestinal anastomoses the Connell stitch is not so frequently used nowadays as formerly, but it is invaluable and safe

when employed with due care in the operation of duodeno-jejunostomy or when making an entero-anastomosis.

The occurrence of hæmorrhage after gastro-jejunostomy is often unjustly attributed to the use of clamps, with the result that many have now entirely discarded their use. If clamps are used gently and cautiously and are applied by the surgeon himself in such a manner as to avoid any damage to the delicate and friable mucosa and only partially to control the bleeding from the cut surfaces of the parts to be anastomosed ; if the stitching is carried out with meticulous care ; and if the grip of the clamps is released, first after the insertion of the posterior row of sutures and again just before the anterior through-and-through suture is completed, to ensure that the suture does not miss

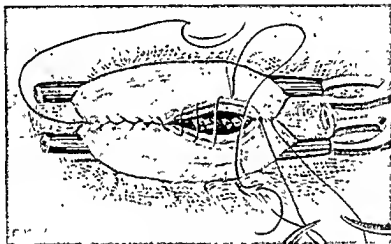


Fig. 137.—THE CONSELL OR LOOP OF THE MUCOSA STITCH.

any bleeding spot, there will be no untoward result from the use of clamps ; in fact, by preventing soiling of the abdominal wound and abdominal cavity through leakage of gastric and intestinal contents, the likelihood of wound sepsis or mild localised peritonitis is prevented, and by bringing the portions of the stomach and jejunum into easy apposition the suturing is rendered quick and simple.

There are, however, certain instances where the use of clamps is not advisable, e.g. where the walls of the stomach have become thinned, friable, and sodden as a result of long-standing dilatation (as occurs in some cases of pyloric stenosis), and usually where gastro-duodenostomy or œsophago-jejunostomy is being undertaken.

(f) Exploratory laparotomy. After tetra-cloths have been affixed to the skin and the peritoneum has been opened, a general exploration of the abdominal cavity is systematically performed (fig. 138).

The viscera are examined in the following order: (i) stomach and duodenum; (ii) liver, gall-bladder, and bile-duets; (iii) pancreas and spleen; (iv) duodeno-jejunal flexure and small intestine; (v) appendix and cæcum; (vi) colon; and (vii) pelvic organs.

The whole stomach and the first and second parts of the duodenum must be methodically examined. It is most important to palpate the whole of the lesser curvature very carefully, and if any lesion is detected the posterior surface of the stomach must be further explored, either through an incision made in the gastro-hepatic omentum or preferably through a rent in the gastro-colic omentum.

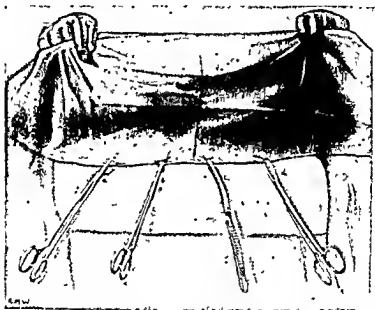


Fig 13N—METHOD OF AFFIXING TETRA-CLOTH TO THE EDGE OF THE WOUND. FOUR DIFFERENT TYPES OF FORCEPS ARE SHOWN IN THE FIGURE: a—MOYNIHAN FORCEPS, b—ALLIS FORCEPS, c—SCOTT RIDOUT FORCEPS, d—LITTLEWOOD FORCEPS.

*If a chronic peptic ulcer is present, its size, shape, position, and other characteristics must be noted and the lesion demonstrated to the assistant.*

In cases where there is a chronic peptic ulcer in the presence of other lesions, e.g., chronic cholecystitis or chronic appendicitis, it is often wise to deal with these latter lesions before proceeding with the gastric operation, and this is possible in most cases. If, however, the patient is in a serious condition as the result of his chronic peptic ulcer, the primary concern should be the performance of the gastric operation, other surgical measures being deferred until some future date.

(2) *Operative Technique.*

The following operations will be described :

(a) *Pyloric Occlusion or Exclusion.*

- (i) The Kelling-Mayo method.
- (ii) The Wilms method.
- (iii) The Bier method.
- (iv) The Devine method.
- (v) Finsterer's operation of pyloric exclusion with partial gastrectomy.

(b) *Excision of Gastric Ulcer.*

- (i) Canterbury excision.
- (ii) Wedge excision.
- (iii) Sleeve resection.

(c) *Pyloroplasty—Gastro-Duodenostomy.*

- (i) Horsley's operation.
- (ii) Judd's operation.
- (iii) Finney's operation.
- (iv) Jaboulay's operation.

(d) *Gastro-Jejunostomy.*

- (i) Posterior gastro-jejunostomy.
- (ii) Anterior gastro-jejunostomy.
- (iii) Retro-colic anterior or posterior gastro-jejunostomy.

(e) *Partial Gastrectomy.*

- (i) The Billroth I types.
  - (a) Finochietto's technique.
  - (b) The Haberer-Finney operation.
  - (c) Schoemaker's operation.
- (ii) The Polya types.
  - (a) Anterior, e.g. Moynihan ; Balfour.
  - (b) Posterior, e.g. Finsterer ; Lahey.

*Pyloric Occlusion or Exclusion*

It is well known that gastro-jejunostomy yields a high percentage of cures when the duodenum is stenosed as the result of a cicatrising ulcer, and that in cases of duodenal ulcer where there is no obstruction the results of this operation are definitely less satisfactory.

The poor results in the non-obstructive ulcer cases are partly due to the fact that some of the gastric contents continue to

pass by way of the pylorus in spite of the newly-made stoma. This naturally prevents the ulcer from being at rest and interferes with the process of healing. It was consequently assumed that if the stenosis could be in some way artificially reproduced, relatively better results would follow. It was not long, therefore, before numerous simple, ingenious, or even un-



Fig. 139.—PYLORIC OCCLUSION. THE KELLING-MAYO METHOD.

necessarily complicated methods were devised for occluding or excluding the pylorus.

While this method is practised by a number of surgeons in non-obstructive cases, provided that gastro-jejunostomy is indicated, others consider that the results are not materially improved by the addition of pyloric occlusion and that certain complications which follow are the direct outcome of this procedure.

The following are some of the methods practised :

*The Kelling-Mayo Method.* In this operation the pylorus is occluded by a silk mattress suture in the manner depicted in figure 139.

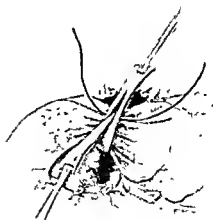


Fig. 140.—PYLORIC OCCLUSION. WILMS METHOD. OCCLUSION OF PYLORUS WITH THE AID OF A FASCIAL GRAFT. THE STRIP OF FASCIA IS TIED IN POSITION WITH A CATGUT LIGATURE AND THEN KNOTTED. THE GRAFT IS THEN BURIED BY STITCHING THE PYLORUS OVER IT.

*The Wilms Method.* By this method a ligature of kangaroo tendon or stout silk is tied around the pyloric end of the stomach, tightly



Fig. 141.—PYLORIC OCCLUSION. WILMS METHOD.  
FIRST STEP IN THE OPERATION.  
(Adapted from "The Duodenum," by Dr. Edward L. Kellogg, New York, Paul B. Hoeber, Inc., 1933.)

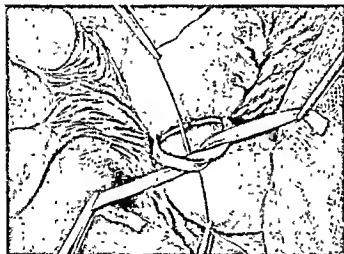


Fig. 142.—PYLORIC OCCLUSION. WILMS METHOD.  
SECOND STEP IN THE OPERATION  
(Adapted from "The Duodenum," by Dr. Edward L. Kellogg, New York, Paul B. Hoeber, Inc., 1933.)

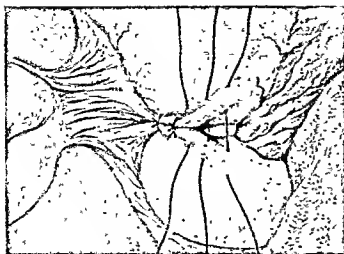


Fig. 143.—PYLORIC OCCLUSION. WILMS METHOD.  
THIRD STEP IN THE OPERATION.  
(Adapted from "The Duodenum," by Dr. Edward L. Kellogg, New York, Paul B. Hoeber, Inc., 1933.)



enough to produce occlusion without injury to the mucosa, but not so firmly as to produce blanching of the tissues.

A long strip of the anterior sheath of the rectus muscle is dissected free and tied in the groove made by the kangaroo tendon (fig. 140), the two ligatures then being buried by drawing the gut over them with a few interrupted Lembert sutures. The steps of this operation are shown in figures 141, 142 and 143.

The Wilms method of occlusion by an autoplasic graft produces a temporary complete block, but as a rule does not cause permanent stenosis. There will, however, as a result of the organisation of the graft, be sufficient narrowing to deflect the bulk of the gastric contents permanently through the new gastro-jejunal stoma (fig. 144).

Ligation of the pylorus with silk, stout catgut, kangaroo tendon, autoplasic ligatures, or ligamentum teres (Polya) without crushing the gut, all aim at producing stenosis for a long enough time to allow the ulcer to heal, i.e. 6-8 weeks.

*The Bier Method.* Bier crushes the pylorus with a powerful enterotribe and doubly ligates the crushed portion with silk, the adjacent portions of the stomach being drawn across the crushed area with two tiers of interrupted sutures.

*The Devine Method.* Here the stomach is divided proximal to the ulcer, and, after closing the pyloric end, the cut end of the stomach is anastomosed to the proximal jejunum.

*Finsterer's Operation of Partial Gastrectomy with Pyloric Exclusion.* In this operation the duodenum is avoided and a sufficient portion of stomach is removed to ensure that the acid level is kept below that at which fresh ulceration can occur. The operation is therefore planned for acid reduction, and is a very satisfactory method of dealing with certain cases of chronic duodenal ulcer without stenosis or with those cases of gastric ulcer where malignancy can be excluded. It is a comparatively simple operation, especially when a Petz clamp is used to render the resection of the stomach a bloodless and aseptic procedure. The subsequent death-rate is low, and the final results are usually excellent.

The first step in the operation is the separation of the great omentum from the greater curvature of the stomach. The arteries and veins to the greater curvature are tied between the stomach and the

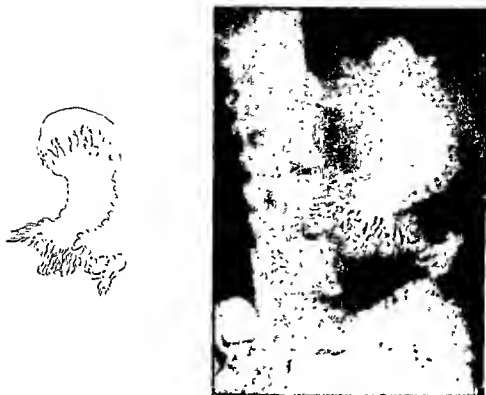


Fig. 144.—PYLORIC OCCLUSION BY WILMS' METHOD PLUS POSTERIOR GASTRO JEJUNOSTOMY FOR CHRONIC DUODENAL ULCER. SKIAGRAM TAKEN FOUR MONTHS AFTER OPERATION. NOTE ALL THE BARIUM MEAL PASSING THROUGH THE HEALTHY STOMA; NO BARIUM IS SEEN TO ENTER THE DUODENUM THROUGH THE PYLORUS. (Author's Case.)

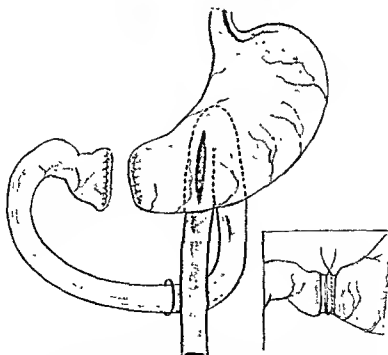


Fig. 145.—PYLORIC OCCLUSION. VON EISELSBERG'S METHOD. A BETTER METHOD, HOWEVER, IS THAT OF DEVINE, WHEREBY THE PYLORIC SEGMENT IS CLOSED AND THE CUT END OF THE STOMACH IS ANASTOMOSED TO THE PROXIMAL JEJUNUM. INSET: THE BIRK METHOD.

vascular gastro-epiploic arch, so that the whole omentum and its blood supply are detached from the stomach.

Ogilvie, in a brilliant and instructive article (*B.M.J.*, March 9, 1935), gives an excellent account of the various points in connection with the technique of this operation, and emphasises the necessity of avoiding damage to this vascular arch. The blood-vessels requiring ligature are more numerous inside the arch than below it, but they should be picked up one by one, close to the stomach, ligatured and cut, and the separation then be carried up to a point about 2 inches from the pyloric outlet and high up on the greater curvature about 2 inches or so below the lower border of the gastro-splenic omentum (fig. 146).

The gastro-hepatic omentum is then torn through with the fingers, and the sheaf of pyloric vessels (the right gastric artery) is underrun with an aneurysm needle and tied off, again about 2 inches from the pylorus.

The Petz clamp is applied to the pyloric vestibule, 2 inches from the pylorus, a Post electric cautery being used to divide the gut between the rows of clips. The pyloric stump is then oversewn.

The coronary or left gastric artery is then ligatured close to its origin. This is done by drawing the stomach upwards over the left costal margin to expose the under-surface of the stomach. The artery is put on the stretch, and an aneurysm needle threaded with a strand of No. 2 20-day chromic catgut is passed round it and tied close to the coeliac axis. The artery is divided between ligatures, and the glands and fatty tissues can then be stripped downwards towards the middle of the lesser curvature.

It will now be seen that the mobility of the stomach is much increased, and a Petz clamp can be applied precisely in the proposed line of transection, which should be oblique, running from a point in the lesser curvature for some 2 inches from the cardiac orifice, and from there obliquely across the body of the stomach towards the greater curvature, as shown in figure 146.

The stomach is then cut through the crushed groove with a fine electric cautery, and the isolated portion removed. The upper half of the crushed portion of the stomach, including an inch or so of the lesser curvature, is invaginated with a double row of closely-applied interrupted Lembert sutures.

An opening is next made in the mesocolon to the left of the middle colic artery, and the proximal jejunum is brought through this opening into the supra-colic compartment. The duodeno-jejunal flexure is identified and a portion of gut some 6-8 inches from this point is

selected for the anastomosis, the proximal end of this isolated loop being applied to the lesser curvature.

The first continuous sero-muscular suture is inserted as in the operation of gastro-jejunostomy, after which the lower half of the crushed portion of the stomach with its clips is trimmed away with scissors.

A suction tube is then introduced into the small gastric pouch to aspirate any remaining gastric contents.

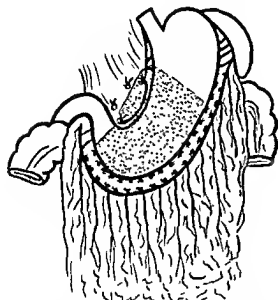


Fig. 146.—FINSTERER'S OPERATION FOR PEPTIC ULCER. THE DOTTED AREA INDICATES THE AMOUNT OF STOMACH REMOVED. THE WHOLE OF THE GREAT OMENTUM AND ITS BLOOD SUPPLY ARE DETACHED INTACT FROM THE STOMACH. (After Ogilvie, B.M.J.)

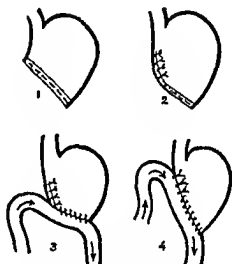


Fig. 147.—CONSTRUCTION OF THE STOMA IN THE FINSTERER NON-RETURN (VALVULAR) GASTRECTOMY.  
(After Ogilvie, modified, B.M.J.)

The jejunum is opened for a length corresponding to the gastric stoma, about one-third to half an inch in front of the continuous sero-muscular stitch. A continuous hæmostatic suture is then introduced, after which the sero-muscular suture is picked up and made to invaginate the anterior suture line. The afferent limb of the jejunum is drawn upwards and attached to the upper half of the gastric pouch with a few interrupted sutures (fig. 147).

The left-hand side of the opening in the mesocolon is sewn to the posterior surface of the stomach before the anastomosis is commenced; the right-hand leaf to the anterior wall after the anastomosis is completed.

The final step in the operation consists of attaching the gastro-epiploic loop carrying the omentum to the posterior abdominal wall above the anastomosis with a few interrupted sutures (fig. 148).

It should be remembered that where this operation is performed for chronic duodenal ulcer, as no acid is produced in the small remaining pyloric pouch and as the valvular formation of the stoma prevents any back-flow of gastric contents into the duodenum, healing of the ulcer is assured (fig. 149).

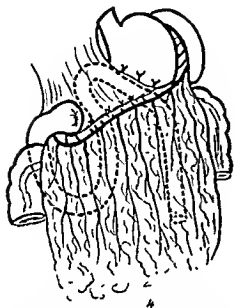


Fig. 148.—FINSTERLIN'S OPERATION OF PARTIAL GASTRECTOMY FOR ULCER COMPLETED. NOTE CONSERVATION OF THE VAGUS GASTRO-PYLOIC ARCH. (After Ogilvie, B.M.J.)

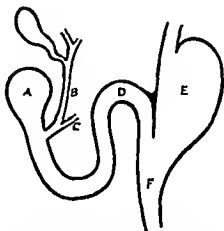


Fig. 149.—DIAGRAM OF FINSTERLIN'S PARTIAL GASTRECTOMY FOR CHRONIC PEPTIC ULCER, TO SHOW MAXIMAL ALKALI CONSERVATION AND PROTECTION OF THE AFFERENT LOOP AGAINST REGURGITATION.

- A—Pyloric Antrum.
- B—Common Bile-Duct.
- C—Pancreatic Duct.
- D—Afferent Loop.
- E—Stomach.
- F—Efferent Loop.

(After Ogilvie, modified, B.M.J.)

### *Excision of Gastric Ulcer*

By whatever method a chronic gastric ulcer is excised, whether by means of the cautery or by wedge excision, this should be followed by gastro-jejunostomy or some form of pyloroplasty, as simple removal of the ulcer has been shown beyond dispute to be followed in at least 50 per cent of cases by a bad result.

When, however, gastro-jejunostomy is performed after the excision, the results are only surpassed in their excellence by partial gastrectomy, although this latter operation carries with it a somewhat higher mortality.

Even where the ulcer has been removed together with a cylindrical portion of the organ—sleeve resection—after the two portions of the stomach have been united, a pyloroplasty by either Horsley's or Jahoulay's method should be undertaken, as a gastro-jejunostomy is often impracticable. But sleeve resection is an operation which I have performed only on the rarest of occasions, reserving it for a few cases in which the ulcer has occupied approximately the middle portion of the stomach. The addition of pyloroplasty greatly enhances the results of this procedure.

### *Cautery Excision (Balfour's Operation)*

Balfour's operation is reserved for those cases in which a *small* ulcer is found in the region of the lesser curvature near the cardia, on the posterior wall where V-excision may prove difficult to perform, or where partial gastrectomy may be contra-indicated. In such cases the destruction of the ulcer by the cautery is a simpler, quicker, and safer method of treatment than wedge excision.

The portion of the stomach in which the ulcer is situated is mobilised, and its covering pad of fat is dissected free until the base of the ulcer can be *clearly and indisputably identified*. The crater of the ulcer is then completely destroyed by a Post or other suitable electric cautery.

The cautery pierces the entire thickness of the stomach wall in its process of destruction of the ulcer crater, producing a perforation which is closed with a few through-and-through interrupted sutures which in turn are hurried by a continuous sero-muscular suture of catgut.

The flap of fat which was dissected off the stomach wall over the ulcer and folded back is then replaced so as to cover the sutured area and is stitched into position in order to afford an efficient barrier against the formation of crippling adhesions.

Gastro-jejunostomy completes the operation.

Balfour's method has much to commend it, especially for small, somewhat inaccessible, chronic gastric ulcers. The cautery sears a minimal amount of stomach wall and so does not in any appreciable measure interfere with the functional capacity of the stomach. In addition to this, the heat destroys any cancer cells which may be present in the ulcer margins.

During the past twenty years Balfour has employed this method in a large number of cases with an operative mortality of only 2-3 per cent. He claims over 80 per cent of cures, while recurrence of ulceration followed in only some 2 per cent of all his cases.

### *Wedge Excision*

When an ulcer is discovered on or in the region of the lesser curvature it must be very carefully examined to determine its size, shape, fixation, and other characteristics before deciding that wedge excision is, in fact, the best procedure to adopt under the circumstances.

If, for instance, the ulcer is large, if penetration of the pancreas has occurred, or if the possibility of malignancy cannot be definitely excluded, it is wiser to perform partial gastrectomy than to be content with the less radical procedure of V-excision of the ulcer.

Again, even where the ulcer is small and easily accessible, partial gastrectomy is to be preferred if fractional test meals show that there is a *very high acid curve*, as under these circumstances ulceration or dyspeptic symptoms are more likely to recur.

By Walton's method, after the ulcer has been excised, the pylorus is occluded and a posterior transverse gastro-jejunostomy is performed.

There are many methods of excising an accessible chronic gastric ulcer, but the technique here described is probably the simplest while the results are very satisfactory.

The stomach is drawn well into the wound and the gastro-hepatic omentum is opened just above the ulcer so that the posterior aspect of the stomach can be thoroughly explored. If any adhesions are found here they should be separated to allow of free mobilisation of the organ, the coronary artery being then tied in two places, proximal and distal to the site of ulceration.

Occasionally it is possible to dissect the artery free from the lesser curvature, retract it, and then replace it after the ulcer has been resected. More often than not, however, the artery is so involved in adhesions that it cannot be isolated, in which case it is secured and tied on the pyloric and cardiac sides of the area to be excised.

An opening is then made in the gastro-colic omentum and one blade of a rubber-covered Sherrin clamp is made to pass below the stomach and emerge on the lesser curvature about 2 inches or so above and to the cardiac side of the ulcer; the other blade is passed anterior to the stomach, after which the two blades are clamped.

A second opening is made through the gastro-colic omentum, near the pyloric region, and a second clamp is applied in a similar manner to the first, again about 2 inches from the ulcer but below and to the pyloric side, so that the ulcer lies between the tips of the two clamps. Thus the clamps isolate the ulcer together with a cylindrical portion of

the stomach, so preventing contamination and facilitating approximation of the parts after excision of the ulcer (fig. 150). By controlling hæmorrhage, they also render suture of the resulting gap a relatively easy matter.

A large, hot, moist swab is packed into the lesser sac and covered with a mackintosh square. The rest of the abdominal cavity is packed off, and the wound margins are protected by waterproof sheeting in order to guard against the risk of infection.

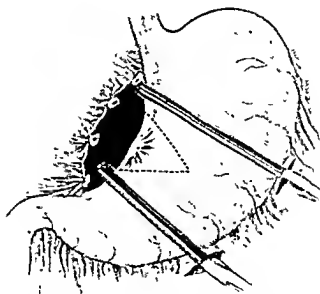


Fig. 150.—WEDGE EXCISION OF CHRONIC GASTRIC ULCER. THE CLAMPS HAVE BEEN APPLIED. THE DOTTED LINE INDICATES THE POSITION OF THE WOUND MADE IN THE ANTERIOR WALL OF THE STOMACH TO EXPOSE THE ULCER. (After Walton)

A wedge-shaped portion of the lesser curvature, including a generous rim of healthy tissue around the ulcer, is removed with scissors or an electric cauter, and the clamps are slightly rotated to bring the posterior lips of the wound into apposition (figs. 151 and 152).

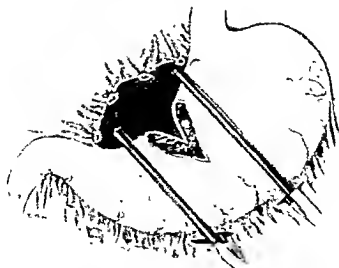
The method of suturing the wound in the stomach is shown in figures 153, 154, 155 and 156, where the position of the reinforcing sutures should be carefully noted.

The operation is completed by occluding the pylorus, usually by the Kelling-Mayo method, and by performing a posterior transverse gastro-jejunostomy, one half of the stoma being placed above and the other half below the sutured area in case there is any tendency to hour-glass constriction.





*Fig. 151.—WEDGE EXCISION OF CHRONIC GASTRIC ULCER. THE ANTERIOR WEDGE IS PULLED UP WITH ALLIS FORCEPS AND RETRACTED UPWARDS IN ORDER TO AFFORD A GOOD VIEW OF THE ULCER. (After Walton.)*



*Fig. 152.—WEDGE EXCISION OF CHRONIC GASTRIC ULCER. THE POSTERIOR WEDGE HAS BEEN REMOVED TOGETHER WITH THE ULCER. THE PARTS ARE NOW READY FOR SUTURE. (After Walton.)*

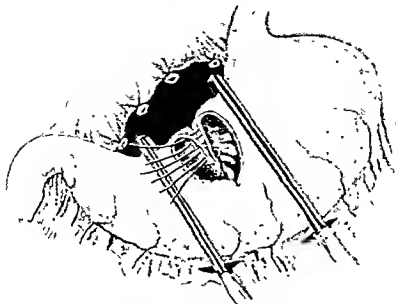


Fig. 153.—WEDGE EXCISION OF CHRONIC GASTRIC ULCER. INTRODUCTION OF THE MATTRESS SUTURES WHICH APPROXIMATE THE CUT EDGES OF THE POSTERIOR WALLS OF THE STOMACH. THE FIRST SUTURE HAS BEEN TIED.

In those cases in which the ulcer has penetrated the pancreas, the gastro-hepatic omentum should be divided, the clamps placed in position and lifted up so as to aid the passage of a finger from above around the base of the ulcer and also the separation of the ulcer itself by dissection from the pancreas.

In the process of this separation the stomach is almost invariably opened. When the organ is retracted the base of the ulcer can be seen deeply embedded in scarred sclerotic pancreatic tissue. The indurated rim of the opening in the stomach should be freely excised with scissors and the wound closed in the manner described.

It is customary to scrape the base of the ulcer or to cauterise it. This, however, is not free from the danger of causing severe hæmorrhage by inadvertently injuring a large artery which may lie exposed in the base of the ulcer. It is far better simply to mop out the cavity and thoroughly disinfect it with tinct. iodine or tinct. metaphen, plug it with an omental graft, and cover the whole area posteriorly with a portion of omentum to prevent the subsequent formation of adhesions between the stomach and the pancreas.

*Trans-gastric resection* of a gastric ulcer is now never advised, and Mayo, who originally advocated the procedure, in his latest papers recommends wedge excision in preference.

*Sleeve Resection—The Riedel-Rodman Operation*

This operation has been called annular gastrectomy, cuff resection of the stomach, segmental resection, sleeve resection, etc., as it involves the removal of a cylindrical portion of the stomach, including the ulcer, this procedure being followed by end-to-end anastomosis between the distal and proximal segments.

This is an operation rarely performed to-day, as the scar of the suture line is prone to contract and to produce a secondary hour-glass stricture, while the final results in no way compare with the brilliant success which follows partial gastrectomy. Nevertheless, Bastianelli, Alessandro, von Eiselsberg, Pannett, and Gordon-Taylor all express great satisfaction with the results obtained. Bumm states that the results in 22 out of 29 of his cases were highly gratifying, whilst Pannett lays stress upon the necessity of performing some form of pyloroplasty at the completion of the operation.

In certain cases of medium-sized ulcer of the body of the stomach in women, Gordon-Taylor advises and practises, where possible, sleeve resection in preference to partial gastrectomy, as he has found that the end-results of the latter operation are not so uniformly good in this sex, there being a tendency to the post-operative development of anæmia. He found that in some 50 per cent of women in a hospital series of his own gastrectomy cases there was a fairly marked anæmia.

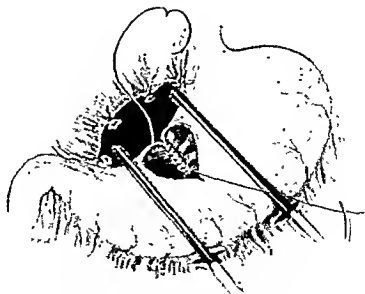


Fig. 154.—WEDGE EXCISION OF CHRONIC GASTRIC ULCER. THE INTRODUCTION OF THE POSTERIOR CONTINUOUS HÆMOSTATIC SUTURE.

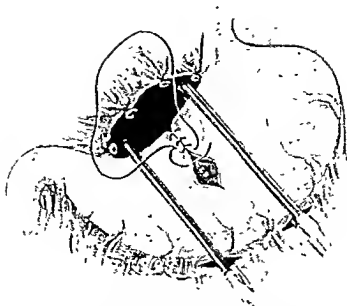


Fig. 153.—WEDGE EXCISION OF CHRONIC GASTRIC ULCER. THE POSTERIOR CONTINUOUS HÆMOSTATIC SUTURE HAS ROUNDED THE CORNER OF THE LESSER CURVATURE, AND IS BEING USED TO DRAW TOGETHER THE ANTERIOR MARGINS OF THE STOMACH WALL.

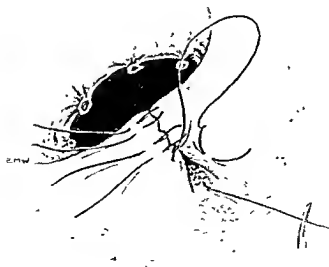


Fig. 156.—WEDGE EXCISION OF CHRONIC GASTRIC ULCER. A CONTINUOUS RIGHT-ANGLE CUSHING STITCH INVAGINATES THE SUTURE LINE. A FEW INTERRUPTED SUTURES ARE INTRODUCED IN THE REGION OF THE LESSER CURVATURE TO DIMINISH THE RISK OF TENSION ON THE SUTURE LINE AT THIS SITE.

The operation is performed as follows: The lesser and greater curvatures of the stomach are freed from the omenta as widely as seems necessary, and the coronary, pyloric, and both gastro-epiploic arteries are ligatured at the limits of the area chosen for resection. A cylindrical portion of the stomach which has been denuded of its blood supply is lifted upwards, dissected free from the pancreas should there be any attachment here, and a hot moist Cripps pad packed into the lesser sac.

At the cardiac and pyloric ends of this cylinder two pairs of clamps are placed, and between each pair the stomach is divided with an electric cauter, and the segment removed. The two outer clamps are then brought together to permit of an easy anastomosis between the two gastric segments. The divided ends of the stomach should be of about equal size. If the pyloric portion is narrow the pyloric clamps should be placed obliquely so that after the performance of the cuff resection there is a longer section of stomach from the greater to the lesser curvature.

The two parts of the stomach can usually be drawn together easily and without producing tension. If, however, difficulty is experienced in approximating these two segments or if it seems likely that tension will follow the making of the anastomosis, the operation should be abandoned, the remaining portion of the pylorus removed, and the operation completed by Polya's method.

The end-to-end anastomosis is performed in a similar manner to that of gastro-jejunostomy, two continuous sutures being used, an outer sero-muscular and an inner through-and-through hæmostatic. The suture line on the anterior aspect of the stomach should be further buried by a Cushing right-angle stitch which effectively invaginates the anterior suture line and prevents the stomach from becoming subsequently attached to the peritoneum of the anterior abdominal wall.

When the operation is completed, a pyloroplasty after the method of Horsley or Jabonlay is essential in order to aid the emptying of the stomach and to relieve any strain on the line of suture.

### *Pyloroplasty—Gastro-Duodenostomy*

*Horsley's Operation.* Horsley (*Surgery of the Stomach and Duodenum*, p. 153. Kimpton) describes the technique of his operation as follows:

"It is not necessary to mobilise the duodenum in this pyloroplasty, but the incision should never be farther than an inch into the duodenum. After packing off the

surrounding tissues, a point midway between the greater and lesser curvatures is caught with Allis forceps. The stomach and duodenum are surrounded with moist gauze and an assistant using his fingers covered with gauze as retractors draws the body of the stomach toward the left, so facilitating the exposure. The length of the incision into the stomach depends upon the location of the ulcer. The incision in the stomach must always be at least twice as long as the incision in the duodenum, and in the duodenum it should never be more than an inch (2.5 cm.). Consequently,

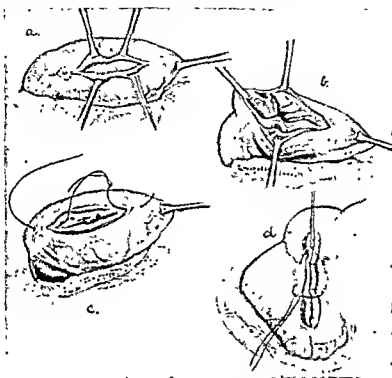


Fig 157—HORSLEY'S "PHYSIOLOGIC" PYLOROPLASTY.

- a. THE INCISION THROUGH THE PERITONEAL AND MUSCULAR COATS OF THE STOMACH HAS BEEN MADE, AND ALL BLEEDING VESSELS ARE CAUGHT. THE DOTTED LINE SURROUNDS THE ULCER WHICH IS TO BE EXCISED.
- b. THE INCISION HAS BEEN CARRIED THROUGH THE GASTRIC MUCOSA, CARE BEING TAKEN NOT TO INJURE THE GASTRIC MUCOSA. THE ULCER IS SHOWN IN THE DUODENUM.
- c. THE ULCER HAS BEEN EXCISED AND A TRACTOR SUTURE IS BEING INSERTED, CATCHING THE WHOLE WALL OF THE DUODENUM BUT ONLY THE MUSCULAR AND PERITONEAL COATS IN THE STOMACH.
- d. BOTH TRACTOR SUTURES ARE BEING TIED.

(Drawn, with slight modifications, from "Surgery of the Stomach and Duodenum," by Dr. J. Shelton Horsley, Kimpton. By kind permission.)

if an ulcer is more than  $\frac{3}{4}$  inch (2 cm.) from the pylorus this pyloroplasty should not be done. If the ulcer in the duodenum adjoins the pylorus, an incision in the duodenum of  $\frac{1}{2}$  inch (1.25 cm.) may be all that is necessary, so the incision in the stomach will be  $1\frac{1}{4}$  inches (3 cm.) in order to divide all of the fibres of the pyloric canal.

The incision is made down to the submucosa of the stomach but not through the mucosa at first. The bleeding points are carefully clamped. The mucosa of the stomach is incised. Great care is taken not to clamp the mucosa of the stomach. The gastric and duodenal contents are emptied with a suction apparatus. The incision

is then carried with scissors through the pyloric sphincter; here bleeding vessels are encountered which should be clamped. By keeping the field as dry as possible with suction apparatus, and sponging with moist gauze wrung out of salt solution, the ulcer can be readily located. If there is a tendency for the gastro-duodenal contents to regurgitate into the wound, a moist gauze pack is placed in the stomach and a small strip of moist gauze in the duodenum. However, with the frequent use of the suction apparatus and a watchful assistant this is usually not necessary.

If an ulcer is on the posterior wall of the duodenum or the pylorus, and is quite limited, it may be cauterised, excised and sutured. If extensive, however, the pyloroplasty should be abandoned and either a partial gastrectomy or a posterior gastroenterostomy should be done.

The ulcer is removed along with a small amount of surrounding healthy duodenal mucosa. Bleeding in the wall of the duodenum is controlled with hæmostatic clamps. No effort is made to separate the mucosa of the duodenum from the rest of its wall, because it would be anatomically difficult to do and because, too, it does not appear that trauma to the duodenal mucosa is so provocative of ulcer as trauma of the pyloric mucosa. A tractor suture is placed and begins at the extremity of the incision in the stomach, passing through the serous and muscular layers only (fig. 157). The pyloric mucosa should not be grasped even with forceps. At the duodenal extremity the suture penetrates the whole of the duodenal wall. About  $\frac{1}{2}$  inch (1.25 cm.) above this, another tractor suture is similarly passed, taking care to avoid injury to the pyloric mucosa. The sutures are of No. 1 tanned or chromic catgut. These sutures are gently tied and the ends are left long and held up to prevent regurgitation of the stomach and duodenal contents.

The hæmostatic clamps that have been placed on the bleeding points should remain until the suturing has begun. A row of sutures of No. 1 tanned or chromic catgut starts at the lower extremity of this wound which has been converted by the tractor sutures into a transverse instead of a longitudinal wound. It is inserted with a fine curved needle, and unites the muscular and peritoneal layers of the stomach. It is drawn snugly, for it is a hæmostatic as well as an approximating suture. The gastric mucosa is not penetrated by this suture. When the duodenum is reached the suture catches the whole of the duodenal wall. No attempt is made to invert the edges of the wound, as it would make too great a bulk, and only sufficient tissue is included in the suture to secure a firm hold. The clamped vessels are released as the suture proceeds. As the incision has been made midway between the greater and lesser curvatures, no large vessels are encountered except where the pyloric sphincter is divided. Here, if necessary, an additional backstitch from the suture will add greater security to the tissues and to the hæmostasis. If gauze has been inserted into the stomach it should be removed before the suture is completed. The suture is tied at the upper end of this transverse wound, and the original short end and this end are used as tractor sutures, in addition to the two tractors that were inserted at first. A second row of sutures of fine tanned or chromic catgut is placed, beginning at the lower end as a purse-string suture, invaginating the feat of stomach at this point, and is carried upward as a right-angle stitch with an occasional backstitch to lock it firmly. The tractor sutures are cut short as the suture reaches them. A right-angle suture is less likely to tear through the wall of the duodenum than an overhand stitch (fig. 158).

The stomach can be readily brought over and as much of it folded over the first line of sutures as desired. When the suture reaches the upper extremity of the wound it is again converted into a purse-string suture and tied. In the middle of the suture

line one or two interrupted mattress sutures of fine catgut are placed and gastro-colic omentum is brought over the line of sutures and fastened by a few interrupted sutures. A tag of peritoneal covered fat from above may also be brought over if it can be done without making traction. This serves not only to support the line of sutures, but to protect from adhesions about the gall-bladder or the liver. Then, too, the weight of the gastro-colic omentum has a slight downward traction on the line of sutures.

In the Heineke-Mikulicz pyloroplasty there is a tendency for the incision as it heals to pull up underneath the liver and make a rather sharp angle. This incision,

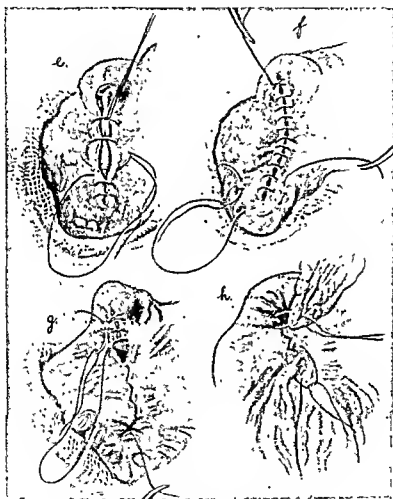


Fig. 158.—HOSSLEY'S "PHYSIOLOGIC" PYLOROPlasty.

- e. THE FIRST ROW OF SUTURES IS BEING PLACED, TAKING ONLY THE MUSCULAR AND PERITONEAL COATS ON THE STOMACH SIDE BUT THE WHOLE THICKNESS OF THE DUODENAL WALL.
- f. THE FIRST ROW OF SUTURES HAS BEEN COMPLETED AND THE SECOND ROW OF SUTURES IS BEGUN AT THE LOWER END OF THE WOUND AS A PURSE-STRING SUTURE. THE ENDS OF THE TRACTOR SUTURES SHOULD NOT BE CUT UNTIL THE SECOND ROW OF SUTURES REACHES THEM.
- g. THE SECOND ROW OF SUTURES IS BEING TERMINATED AS A PURSE-STRING SUTURE AT THE UPPER END OF THE WOUND.
- h. A FEW INTERRUPTED SUTURES BRING OVER PERITONEAL-COVERED FAT.

(Drawn, with slight modifications, from "Surgery of the Stomach and Duodenum," by Dr. J. Shelton Hossley, Kimpton. By kind permission.)



however, because most of it is on the stomach side, should be less likely to be drawn up toward the liver, but even this must be prevented. Adhesions about the pylorus or duodenum are much more prone to give symptoms than in portions of the gastrointestinal tract where the range of motion is normally greater. Adhesions about the middle of the transverse colon or about the body of the stomach are frequently symptomless, but adhesions around a viscus whose motion is normally very limited are prone to cause disagreeable and uncomfortable sensations. Adhesions about the heart, for instance, which has a limited but essential motion, are always a matter of consequence. Adhesions about the ileo-colic valve or the duodenum will frequently produce clinical disturbances because their very limited range of movement is interfered with, whereas if movement is naturally through a wide range, adjustment to adhesions may be easily made. Consequently, it is important in operations about the pylorus to make every effort to avoid adhesions as far as possible, and if adhesions must occur they should be to fatty tissue rather than to relatively solid tissue, such as the under-surface of the liver, or to the gall-bladder which is sensitive to any traction from adhesions."

*Judd's Pyloroplasty.* This operation consists of the mobilisation of the duodenum and the removal of the anterior half or two-thirds of the pyloric sphincter, the cap of the duodenum, and the ulcer (fig. 159). The union of the stomach and duodenum posteriorly is not incised. When this excision is completed the openings at the lower end of the stomach and the upper end of the duodenum appear like the two openings of a gastro-jejunostomy after the posterior sutures have been inserted (fig. 160). The aperture which results from this excision is closed in the transverse axis with two or three rows of continuous sutures (fig. 161).

Figures 159, 160 and 161 show clearly the various steps of the operation.

*Judd lays stress upon the necessity for free mobilisation of the duodenum and wide excision of all the scarred tissue of the bulb.*

If the ulcer found on the posterior wall of the duodenum is small it may be canterised, but if it is large and penetrates the pancreas to any great depth, the operation should be abandoned and the wound in the gut closed in the longitudinal axis so as to narrow or even occlude the pylorus prior to the performance of a gastro-jejunostomy, as recommended by Kellogg.

The operation must also be abandoned if all the scarred tissue in the anterior wall of the gut cannot be satisfactorily excised, for unless the parts concerned in the anastomosis are perfectly healthy, stricture formation at the line of suture or recurrence of ulceration is likely to follow.

*Gastro-duodenostomy by Finney's Method.* To my knowledge Finney's operation is not often performed in this country, as most

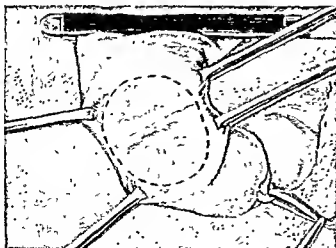


Fig. 159.—Judd's Pyloroplasty for Chronic Duodenal Ulcer. The portion of the anterior wall of the pylorus and duodenum to be excised is shown as encircled by a dotted line.  
(Adapted from "The Duodenum," by Dr. Edward L. Kellogg, New York, Paul B. Hoeber, Inc., 1933.)

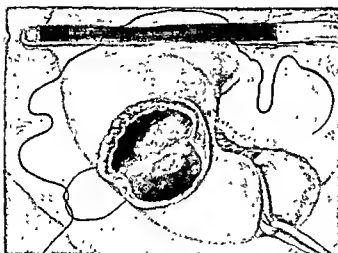


Fig. 160.—Judd's Pyloroplasty for Chronic Duodenal Ulcer. The appearance of the parts after resection of the anterior portion of the pylorus and adjacent gut. The first through and through suture being.  
(Adapted from "The Duodenum," by Dr. Edward L. Kellogg, New York, Paul B. Hoeber, Inc., 1933.)

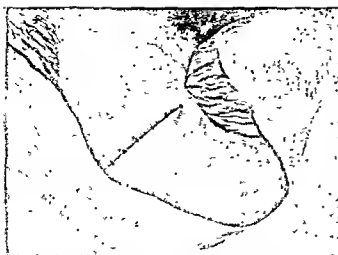


Fig. 161.—Judd's Pyloroplasty. The operation completed.  
(Adapted from "The Duodenum," by Dr. Edward L. Kellogg, New York, Paul B. Hoeber, Inc., 1933.)

surgeons prefer a gastro-jejunostomy to any form of pyloroplasty for chronic duodenal ulcer; those, however, who favour gastro-duodenostomy in certain cases usually choose the method of Jaboulay on account of its greater simplicity.

Finney's operation is only possible where surrounding adhesions are absent or slight, where mobilisation of the duodenum and pylorus presents no difficulty, and where the occlusion produced by an anteriorly situated duodenal ulcer is only partial.

It is definitely contra-indicated where extensive adhesions exist, where there is a large inflammatory mass in the duodenum associated with considerable infiltration, where the gastro-hepatic omentum is short and thick, where the gut is greatly scarred and puckered, and its lumen is almost wholly occluded, or where there is a large penetrating duodenal ulcer on the posterior wall.

The operation is started by mobilisation of the first and second parts and the commencement of the third part of the duodenum, and of the pylorus itself.

*Unless an easy mobilisation can be achieved the operation should be abandoned, as recommended by Finney himself.*

When the pylorus and duodenum have been freed, three sutures are introduced to serve as guides and retractors. One is passed at the upper border of the pylorus, another in the anterior wall of the stomach near the greater curvature, about 3-3½ inches below the pylorus, whilst the third is inserted in the anterior border of the duodenum at a point exactly opposite to the guide suture in the stomach.

The stomach and duodenum are brought together for the introduction of the first sero-muscular suture by making downward traction upon the two lower tractor sutures, while at the same time the guide in the pylorus is pulled upwards.

It is best to dispense with clamps, although there can be no doubt that they simplify the operation and diminish the dangers of contamination. If clamps are used they should be placed in the manner depicted in figure 162, where it will be noted that, when accurately applied, they meet at the pylorus but do not cross or control the convex portion of the horseshoe, i.e. the pyloric canal and the duodenal bulb.

The first posterior sero-muscular suture is then introduced, commencing at the pylorus and ending just above the two lower tractor sutures. An inverted U or a horseshoe-shaped incision is made parallel with the posterior line of sutures through all the coats of the stomach, the pylorus, and the duodenum (fig. 162).

If a chronic ulcer is found on the anterior wall of the duodenum

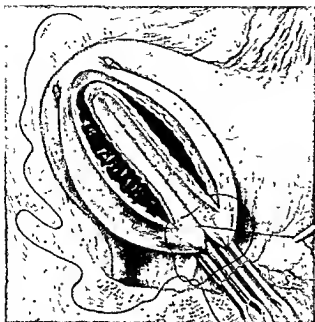


Fig. 162.—FINNEY'S OPERATION. THE STOMACH AND DUODENUM HAVE BEEN CLAMPED AND AN OPENING MADE IN THE MANNER DEPICTED. A TRACTOR SUTURE HAS BEEN INTRODUCED AND IS ABOUT TO BE TIED.

(Adapted from "The Duodenum," by Dr. Edward L. Kellogg, New York, Paul B. Hoeber, Inc., 1933.)

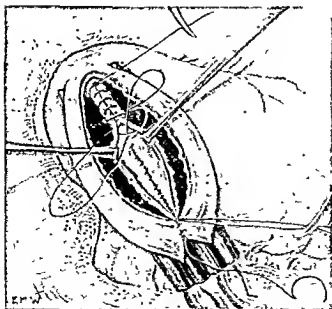


Fig. 163.—FINNEY'S OPERATION. THE INNER SUTURE COMMENCED.

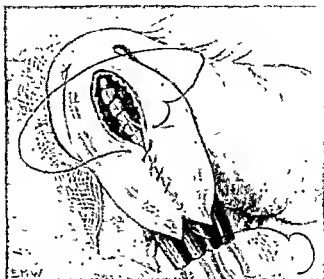


Fig. 164.—FINNEY'S OPERATION. THE THROUGH-AND-THROUGH SUTURE IS NEARING COMPLETION.

it can be excised easily with scissors, as much scar tissue as possible being removed with it. If, however, there is an ulcer on the posterior wall, although it may be difficult to excise it, this is nevertheless possible by trimming away a triangular portion of the duodenal wall in which the ulcer is embedded. If the ulcer found in the posterior wall is small, it may be destroyed with an electric cantery.

The posterior through-and-through hæmostatic suture is then

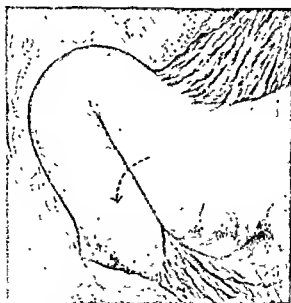


Fig. 165.—FINNEY'S OPERATION COMPLETED.

introduced, starting at the divided pylorus and proceeding downwards to the lower angle of the incision (fig. 163). From this point it is carried upwards without interruption, uniting the anterior edges of the stomach and duodenum and the divided pylorus, beyond which point it is tied (fig. 164).

The posterior sero-muscular suture is next picked up and continued anteriorly to reinforce and invaginate the first row of sutures. A few Halsted sutures are inserted here and there where the anterior suture line requires strengthening, and adjacent tags of omentum are drawn across to prevent the gut from adhering to the parietal peritoneum, gall-bladder, or liver.

Some surgeons make three suture lines, the second of which unites the cut edges of the serosa and muscularis posteriorly and is continued anteriorly after the mucosal stitch has been inserted.

When the operation is completed it will be seen that the duodenum resumes its original position, dragging the stomach with it (fig. 165).

### *The Operation of Gastro-Duodenostomy by Jaboulay's Method*

Jaboulay united the anterior wall of the duodenum to the anterior wall of the stomach, folding the duodenum forward over the hinge



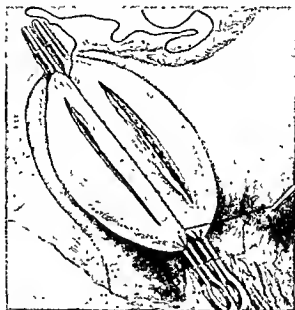
Fig 166.—A METHOD OF INFOLDING THE SCARRED ANTERIOR WALL OF THE DUODENUM.

formed by the pylorus. The operation here described is a slight modification of this method, in that the duodenum is first mobilised, and an ulcer, if found on the anterior wall of the duodenum, is excised, destroyed with a cautery, or infolded prior to the making of the anastomosis (fig. 166).

Those who favour this type of operation for chronic duodenal ulcer consider it to be contra-indicated under the following circumstances:

(1) When the ulcer in the duodenum is large and associated with a considerable degree of acute inflammation. In such cases the surrounding inflammation may predispose to further ulceration at the suture line.

(2) When the duodenum cannot be sufficiently mobilised. This may be due to extensive adhesions in the vicinity of the ulcer which bind the gut to the under-surface of the liver or to the posterior



*Fig. 107.*—JACQUELYN'S OPERATION OF GASTRO-DUODENOSTOMY. THE FIRST SUTURE HAS BEEN INTRODUCED. THE INCISIONS IN THE STOMACH AND DUODENUM ARE SHOWN. THE OPERATION PROCEEDS AS IN GASTRO-JEJUNOSTOMY.

abdominal wall. Again, it may be due to deep erosion of the pancreas by a large chronic ulcer. In certain cases Kocher's mobilisation may be impossible owing to the presence of many abnormally placed vessels at the outer side of the duodenum or to the gut being in an aberrant position.

When the duodenum is obstructed in its third part, the operation is still feasible if supplemented by duodeno-jejunostomy.

The first step in the operation is the free and complete mobilisation of the duodenum by Kocher's method. Into the raw space which results from this process a moist hot Cripps pad is placed. The ulcer in the duodenum is then excised with a knife or cautery, the wound edges being infolded and protected with an omental pad.

The descending portion of the duodenum is clamped for a length of about  $2\frac{1}{2}$  inches, after which some  $2\frac{1}{2}$  inches of the anterior stomach wall in the pyloric segment near the greater curvature are clamped up to the pylorus. The operative field is then carefully isolated and packed off, and the operation is conducted in a similar manner to a gastro-jejunostomy (fig. 167).

This operation may be, and often is, performed without the aid of clamps.

At the completion of the operation it is important to protect the anterior suture line with adjacent tags of omentum to prevent the formation of adhesions which may subsequently prove crippling.

If at the completion of this type of gastro-duodenostomy the stoma appears to be unduly small (a common occurrence), or if, owing to inadequate mobilisation, there is compression, rotation, or other evident mechanical defect at the site of anastomosis, it is good practice to supplement the procedure by performing a gastro-jejunostomy.

The immediate post-operative stage is usually stormy. There is often much epigastric pain and disturbance, sometimes associated with severe vomiting due to the fact that on account of spasm and œdema of the stoma the stomach is unable for the time to pass an adequate amount of its contents into the duodenum. Instantaneous relief may be afforded by aspiration of the stomach contents and intravenous injections of saline. The late results, however, are on the whole gratifying and a barium meal examination at a later date will prove the emptying rate of the stomach to be efficient—at times almost too rapid, and the gastric tone good.

### *Gastro-Jejunostomy*

For cases where it is indicated, there is no operation in abdominal surgery which yields such satisfactory results as a well-performed gastro-jejunostomy.

Since its introduction by Wölfler, in 1881, this operation has passed through many variations in the method of its performance, until now it has become more or less standardised. Although the fundamental principles underlying its performance are still adhered to, many surgeons have introduced numerous minor differences in the technique which, however, do not appear to affect the final results.

Wölfler's first operation was an *anterior* long-loop anti-peristaltic gastro-jejunostomy, and this, in 1885, gave place to von Hacker's *posterior* long-loop anti-peristaltic gastro-jejunostomy.



As obstructive symptoms were common after von Hacker's operation, Braun introduced the addition of a lateral entero-anastomosis to overcome the regurgitant vomiting which was such a common sequela of the long-loop method, and he later obtained even better results by performing an anterior long-loop iso-peristaltic gastro-jejuno-stomy plus an entero-anastomosis between the proximal and distal jejunal loops.

Roux's anastomosis in Y was a method by which it was hoped that obstruction would be overcome, but unfortunately this operation was followed by jejunal ulceration in a large number of cases.

Brenner (1892) was the first to perform a *retro-colic anterior* long-loop iso-peristaltic gastro-jejuno-stomy. The loop proximal jejunal loop was brought through an opening in the mesocolon and gastro-colic omentum, and the jejunum was anastomosed to the pyloric segment of the stomach.

Stiles and Sherren obtained better results than Brenner by performing the operation without a loop, making a junction either to the anterior wall of the stomach or to its posterior surface. This operation still has its indications, as we shall later show.

Petersen (1900) laid the foundations of the modern operation of posterior no-loop gastro-jejuno-stomy. Moynihan and the Mayos were quick to observe the enormous advantages of this method which overcame the great drawback of the long-loop operation—vicious circle vomiting—and they, by their masterly skill and judgment, evolved a technique which was widely adopted and still remains unsurpassed.

Moynihan made the *opening in the stomach* vertical, extending from the lesser to the greater curvature, and insisted that a portion of the opening should be placed at the greater curvature to prevent the formation of a pool of stagnant fluid in the stomach.

W. J. Mayo anastomosed the jejunum to the stomach in an anti-peristaltic line so that there should be no interruption in the line of the duodenum at the flexure.

In practice the majority of cases show but little if any difference in the results of the operation, whether the opening is made vertical or transverse, provided that some portion of the opening is placed as near to the greater curvature as possible. In certain cases, however, a transverse opening is to be preferred to a vertical. For instance, if the stomach is very small and contracted, the distance from the lesser to the greater curvature is often not sufficient to allow of an adequate stoma being made, and in such cases the stoma should be made transverse or slightly oblique.

Again, after V-excision of a gastric ulcer or destruction of an ulcer by Balfour's cautery method, a transverse opening placed close to the greater curvature ensures that the anastomosis is far enough away from the suture line in the stomach or from the cauterised area.

A vertical opening is usually made when the stomach is greatly dilated as the result of pyloric stenosis, or when a gastro-jejunostomy is indicated for an ulcer situated high up on the lesser curvature near the cardia. The position, direction, and size of the stoma are all important factors in the production of a successful issue.

Moynihan's vertical stoma is made in the posterior wall of the stomach in line with the vertical portion of the lesser curvature and with the right margin of the oesophagus. It stretches from just below the lesser curvature to the lowest point of the greater curvature. The jejunum is usually brought directly downwards in a straight line from the flexure without a loop, and anastomosed to the under-surface of the stomach, thus eliminating all possibility of regurgitant vomiting.

In cases of marked visceroptosis or where the stomach is unusually small and contracted, it is advisable to leave a small loop of jejunum, say 2-3 inches, in order to give a certain amount of play at the anastomotic line and thus avoid tension and kinking.

Where V-excision of a gastric ulcer has been performed it is my practice to make the transverse opening as near the greater curvature as possible, half this opening being above and half below the sutured area in the stomach, the jejunum being attached to the stomach in an iso-peristaltic rather than in an anti-peristaltic direction.

In making an anterior gastro-jejunostomy the stomach clamp should be applied somewhat obliquely so that the direction of the stoma runs from above downwards towards the greater curvature, ending about 2 inches below the pylorus. This is well shown in figure 185.

The *size of the stoma* varies in individual cases; for instance, when the stomach is markedly dilated the stoma must be correspondingly large, i.e. about  $3\frac{1}{2}$  inches. Whilst the usual size of the opening is about  $2\frac{1}{2}$  inches, it should never be less than 2 inches or more than  $3\frac{1}{2}$  inches.

In view of the subsequent contraction of the stomach and of the stoma itself which follows gastro-jejunostomy in cases of dilatation of the stomach due to pyloric stenosis, the anastomotic opening in such cases must always be of generous proportions. When the stomach is normal in size, however, no appreciable contraction of the stoma occurs; in fact, when such does take place it is generally due to the original opening having been made either too small or at too high a level, or to

the fact that a fresh ulcer has developed at the junction. Such contraction, which may be clearly demonstrated at a barium meal examination, is an ominous sign.

As previously stated, no useful purpose can be served by making the anastomotic opening unduly large, as this may result in too rapid emptying of the stomach—"dumping" stomach—accompanied by epigastric distress, flatulence, and diarrhoea.

### *The Effects of Gastro-Jejunostomy*

#### (1) Relief of Obstruction.

(a) Mechanical, e.g. pyloric stenosis.

(b) Physiological, e.g. pyloric spasm.

#### (2) Relief of pain by diminishing intra-gastric tension.

#### (3) Neutralisation of the acid gastric contents by the regurgitation into the stomach of the alkaline pancreatic juices.

Paterson states that the total acid is diminished by 30 per cent after gastro-jejunosomy, and that persistence of acidity at or about the pre-operative level often heralds a recurrence of symptoms or of ulceration. In a communication to the *B.M.J.* (p. 676, March 30, 1935) he writes as follows:

"The acidity can be, and is, reduced by gastro-jejunosomy, and, if necessary, it can be further reduced by a suitable diet. Nature's method of reducing the gastric acidity is by allowing the regurgitation of pancreatic juice and bile through the pylorus. Gastro-jejunosomy aids Nature in this endeavour by allowing a slight backflow of pancreatic juice and bile through the stoma into the stomach, and therefore is a rational physiological, and I may add, effective operation."

Ogilvie (*Lancet*, p. 421, Feb. 23, 1935), although holding a different view from Paterson, makes some interesting observations on this subject. He writes:

"The claims for gastro-jejunosomy are twofold—that it overcomes stasis and that it allows neutralisation of the acid gastric contents. These two claims must be considered separately. A fresh opening, if correctly placed and efficiently fashioned, will most assuredly empty the stomach and relieve stasis if this is present. But such an opening will not necessarily reduce the acidity of the gastric contents, and in fact it does not do so. High acid and rapid emptying go together, and when an artificial opening has been made, the emptying of the stomach is still more rapid and neutralisation less complete. The claims for the neutralising action of gastro-jejunosomy were based upon test-meals performed shortly after the operation; but later investigations of the same patients have shown that the acid curve has returned to its pre-operative level, or even higher. The low value obtained at the first test-meal was due, not to neutralisation, but to the effect of the operation itself upon gastric secretion."

## (4) Improvement in nutrition.

*Posterior Gastro-Jejunostomy.* Many surgeons have introduced some slight variation in the technique of this operation, but the method I am about to describe, which is based on Moynihan's, is the one I have found to be the most satisfactory in my own cases.

The abdomen is opened through a right paramedian or mid-line incision of ample length to permit of an easy exploration of the abdominal viscera. Tetra-cloths are fixed to the skin margins, and hæmostasis of the wound is ensured. The ulcer is located and demonstrated to the assistant. The entire stomach, the duodenum, and the duodeno-jejunal flexure are methodically scrutinised for evidence of any other pathological lesion. The abdominal viscera are then examined in the order suggested on page 332.

The stomach, the transverse colon, and the great omentum are drawn through the wound and held up by the assistant to allow of an examination of the under-surface of the mesocolon and the position of the middle colic artery and its branches.

The surgeon then passes his right hand along the mesocolon towards the left of the spine to pick up and identify the duodeno-jejunal flexure, after which he withdraws some 6-8 inches of the proximal jejunum through the wound, wraps it in a small abdominal towel soaked in warm saline, and lays it on the abdominal wall.

Any pathological or physiological adhesions which are found attaching the jejunum to the under-surface of the mesocolon are divided.

The arrangement of the blood-vessels in the mesocolon is carefully noted, as if these are numerous, aberrant, or obscured in fat, if an adequate bloodless space cannot be found in which to make the opening into the lesser sac, if the mesocolon is very short or is fused to the peritoneum of the stomach bed, or if a large area of the stomach is fixed posteriorly by a penetrating ulcer, the posterior operation will have to be abandoned.

If, however, conditions permit of the performance of the posterior operation, the assistant holds the transverse colon upwards with both hands, while the surgeon presses the posterior wall of the stomach downwards against the mesocolon to put it on the stretch.

An opening is then made through the mesocolon into the lesser sac in the bloodless space which exists to the left of the middle colic artery and beneath its anastomotic arch (fig. 168). This opening is started by working a small hole through the taut mesocolon with dissecting forceps, the fingers then being used to enlarge it by gentle stretching

and tearing until three or four fingers can be introduced without difficulty.

By another method the mesocolon is lifted up away from the stomach and clipped with a hæmostat at a bloodless spot. A snip is then made with scissors into the lesser sac, after which the opening can be further enlarged.

This opening must not be too niggardly, never less than 3 inches long, and should always permit of a free exploration of the posterior aspect of the stomach. If any adhesions exist between the stomach and its bed these should be freed and the organ adequately mobilised before proceeding with the anastomosis.

In order to make a vertical stoma a vertical fold of the posterior wall of the stomach, in line with the lesser curvature and with the right margin of the œsophagus, is picked up and drawn through the rent in the mesocolon, and Allis forceps applied at each extremity of this fold, i.e. one pair at the greater curvature and one pair at the lesser curvature.

As this fold, which should be 3-4 inches in length, is held up with the two pairs of Allis forceps by the assistant, a Sherren clamp with rubber-covered blades is applied vertically, the tips of the blades pointing towards the patient's chin and the handles towards the pubes (fig. 169). The clamp is then rotated so that its tips point to the patient's right side, the handles being held on the left side by the assistant.

The coil of jejunum which was withdrawn through the wound at the commencement of the operation and which was covered with a towel and laid on the abdominal wall is now picked up and put on the stretch, to identify clearly the duodeno-jejunal flexure and to determine the exact distance from the flexure at which the clamp should be placed.

In the majority of cases the operation is performed without a loop, i.e. the clamp is applied as close to the duodeno-jejunal flexure as possible; in other cases, however, where there is marked visceroptosis or the stomach is tubular and contracted, a small loop of jejunum must be allowed between the clamped portion of gut and the flexure to permit of a certain amount of play when the patient stands erect, when the stomach will tend either to fall downwards towards the pelvis or retract upwards under the liver.

The anti-mesenteric border of the portion of jejunum selected for the anastomosis is picked up with Allis forceps which are placed at a distance of 3-4 inches apart. Whilst the surgeon lifts up these forceps,

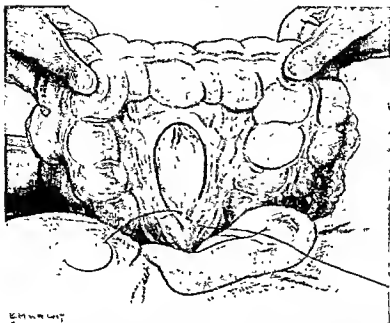


Fig. 168.—POSTERIOR GASTRO-JEJUNOSTOMY. THE TRANSVERSE MESOCOLON IS DRAWN THROUGH THE WOUND AND IS LIFTED UPWARDS BY THE ASSISTANT. AN OPENING IS MADE THROUGH AN AVASCULAR AREA OF THE MESOCOLON. THE POSTERIOR WALL OF THE STOMACH IS FORCED THROUGH THE RENT IN THE MESOCOLON, AND A STITCH IS INTRODUCED THROUGH THE MESOCOLON TO ANCHOR THE STOMACH TO IT, AS SHOWN IN THE ILLUSTRATION. THE PROXIMAL JEJUNAL LOOP HAS BEEN DRAWN THROUGH THE ABDOMINAL INCISION, READY FOR ANASTOMOSES.

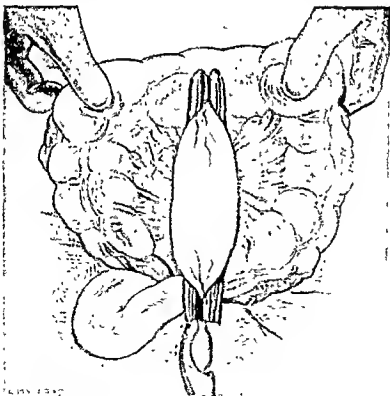


Fig. 169.—POSTERIOR GASTRO-JEJUNOSTOMY. METHOD NO. 1. APPLICATION OF THE CLAMP TO THE STOMACH.

thus rendering the gut taut, the clamp is lightly applied by the assistant (fig. 170).

The Allis forceps are then removed and the surgeon takes the clamp from the assistant and locks it, as he himself is the best judge of the degree of tightness required. This important step in the operation, i.e. the locking of the clamps, should never be assigned to the assistant, as if the clamps are applied with undue force the friable mucosa may be damaged. The two clamps are then placed side by side, the handles being held by the assistant and the points directed to the right side of the patient.

There are other methods and directions in which the clamps may be applied, a good alternative being to place them in the reverse direction to that just described, i.e. the gastric clamp is applied from above downwards with the handles pointing towards the chin and the tips towards the pubes (fig. 171), whilst the tips of the jejunal clamp, when in position, point towards the patient's left shoulder. The handles of both clamps are then turned transversely so that they come to lie on the patient's left side where they are held by the assistant (fig. 172).

When the two clamps are in position it will be seen that the uppermost one embraces a portion of the stomach 3-4 inches in length, whilst the lower one holds a portion of jejunum of about the same length. The clamps are then rotated outwards and a long strip of gauze is placed between them (fig. 173), after which they are brought close together so that the portions of stomach and jejunum about to be anastomosed lie in easy apposition.

The transverse colon, the omentum, and any intestines which have prolapsed through the wound are now replaced into the abdominal cavity and are prevented from protruding into the field of operation by means of suitably placed abdominal packs. The only portions of the viscera which are allowed to remain outside the abdomen are those about to be anastomosed.

The operative field is isolated with hot moist Cripps pads or turkish towels wrung out in hot saline, over which mackintosh squares are placed. The tips of the clamps and as much as possible of the handles should be covered and bidden from view by the towels.

Any large blood-vessels which are seen on the wall of the stomach are under-run with an eyeless needle which carries a length of fine cat-gut. It is important to pass the needle sufficiently deep to the blood-vessels to prevent the possibility of their being punctured with the consequent formation of a hæmatoma. The sutures are of No. 0 or

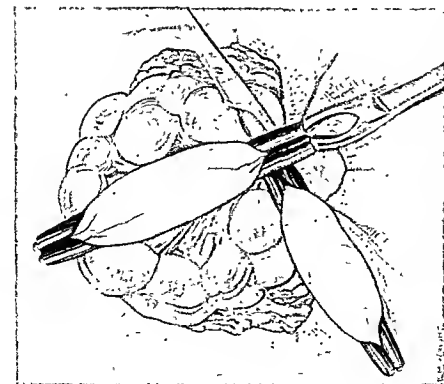


Fig. 170.—POSTERIOR GASTRO-JEJUNOSTOMY, METHOD NO. 1. BOTH CLAMPS HAVE BEEN APPLIED. (After McFarlane.)

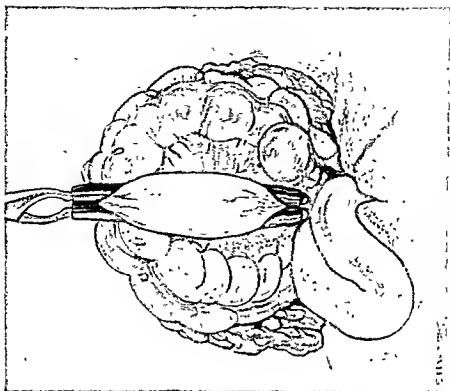
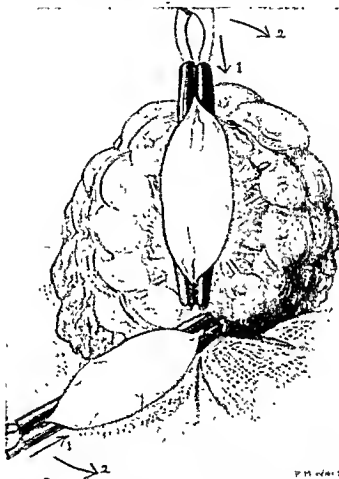


Fig. 171.—POSTERIOR GASTRO-JEJUNOSTOMY, METHOD NO. 2. APPLICATION OF CLAMP TO THE STOMACH.



No. 00 20-day chromic catgut, and are introduced on eyeless atraumatic needles. Usually only two continuous sutures are used—a sero-muscular and a through-and-through hæmostatic. Neither of these is knotted except at the starting and finishing points.

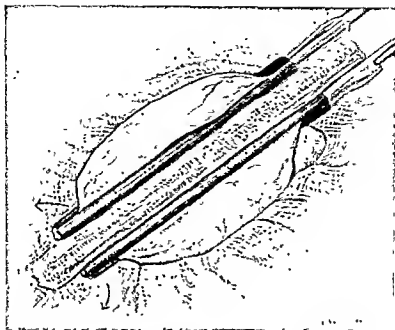
The first stitch, which is a continuous Lembert, may begin either at the lower (the patient's right-hand side) or, more often, at the upper



*Fig. 172.*—POSTERIOR GASTRO-JEJUNOSTOMY, METHOD NO. 2. BOTH CLAMPS HAVE BEEN APPLIED. *Arrows 1* INDICATE THE ORIGINAL DIRECTION OF THE CLAMPS BEFORE THE HANDLES ARE ROTATED TOWARDS THE PATIENT'S LEFT SIDE AS SHOWN BY *Arrow 2*.

extremity (the patient's left-hand side) of the clamped portions of the stomach and jejunum.

Assuming that the stitch starts at the upper end, it picks up the sero-muscular coats of the stomach and jejunum at this point and is then knotted and tied, one end which is left long being clipped with a hæmostat (fig. 174). The length of the suture line is usually between  $2\frac{1}{2}$  and 3 inches, and should never be less than 2 inches. The individual

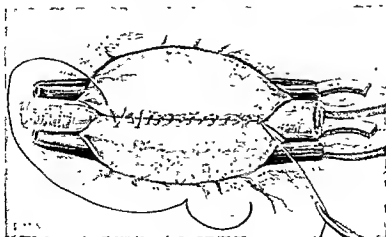


*Fig. 173.—POSTERIOR GASTRO-JEJUNOSTOMY. BEFORE THE SUTURING IS COMMENCED A ROLL OF GAUZE IS PLACED BETWEEN THE PORTIONS OF STOMACH AND DUODENUM EMBRACED BY THE CLAMPS.*

stitches are placed very close to one another, approximately  $\frac{1}{8}$  inch apart, each one being drawn tight as it is introduced, producing a small fold which indicates where the next stitch should be inserted.

When the suture reaches the opposite end it is locked once or twice and the suture and needle are wrapped in a sterile gauze swab and laid aside for use at a later stage in the operation.

Two longitudinal incisions through the sero-muscular coats of the stomach and jejunum are now made with a knife, parallel with and  $\frac{1}{4}$ – $\frac{1}{3}$  inch from the posterior suture line. The mucous membrane of the



*Fig. 174.—POSTERIOR GASTRO-JEJUNOSTOMY. THE FIRST SUTURE IS BEING INTRODUCED.*

stomach and jejunum will pout through these incisions as the sero-muscular coats retract. Any large blood-vessels which can be seen coursing over the mucous membrane should be undermined and ligatured on each side of the incision (fig. 175).

The mucous membrane of the stomach is picked up with forceps and incised with an electric cautery or seissors for the full length of the sero-muscular incision. Any gastric contents are rapidly mopped up and the interior of the gastric pouch is cleansed with moist gauze. The jejunal mucous membrane is similarly picked up, incised, and the

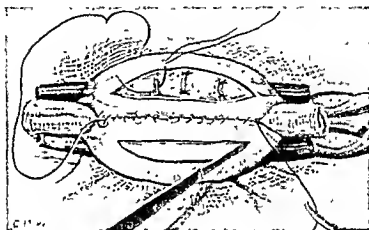


Fig. 175.—POSTERIOR GASTRO-JEJUNOSTOMY. THE FIRST SERO-MUCOSAL SUTURE HAS BEEN INTRODUCED AND LOCKED. THE STOMACH AND JEJUNUM HAVE BEEN INCISED DOWN TO THEIR MUCOUS MEMBRANES. THE LARGE BLOOD-VESSELS WHICH LIE OVER THE NOW EXPOSED MUCOUS MEMBRANE OF THE STOMACH ARE UNDERMINED AND TIED SECURELY TO DIMINISH THE POSSIBILITY OF POST-OPERATIVE BLEEDING.

gut sponged. Redundant mucous membrane of the stomach or jejunum is not excised, as such trimming may predispose to the formation of stomal ulceration.

The posterior margins of the wound in the jejunum and stomach are separately picked up with Allis forceps and held apart to facilitate the introduction of the posterior continuous through-and-through hæmostatic suture. This suture may start at the lower, or more often at the upper, end of the clamped portions of the stomach and jejunum.

Using a curved atraumatic intestinal needle carrying a suture of No. 0 or No. 00 chromic catgut, all the coats at the uppermost extremity of the wound in the jejunum are pierced from within outwards, the needle then being carried across to the corresponding point in the stomach which it pierces from without inwards, so that when the suture is tied the knot lies on the mucous surface (fig. 176).

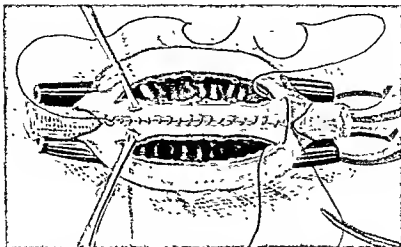


Fig. 170.—POSTERIOR GASTRO-JEJUNOSTOMY. THE COMMENCEMENT OF THE SECOND SUTURE.

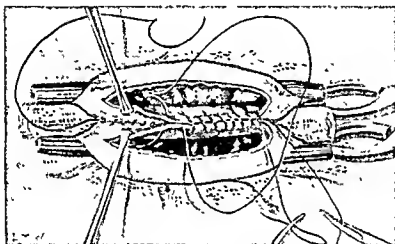


Fig. 177.—POSTERIOR GASTRO-JEJUNOSTOMY. THE SECOND SUTURE EMBRACING ALL THE COATS.

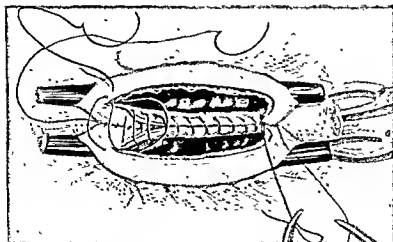


Fig. 178.—POSTERIOR GASTRO-JEJUNOSTOMY. THE METHOD ADVISED FOR TURNING THE CORNER WITH THE CONTINUOUS THROUGH-AND-THROUGH HEMOSTATIC SUTURE.

This suture, as it proceeds towards the lower end (the patient's right-hand side), picks up all the coats of the posterior margins of the jejunum and stomach, the individual stitches being vertical and almost side by side, and being drawn sufficiently tight to prevent bleeding (fig. 177). Before it reaches the corner, the clamps are momentarily loosened to detect any bleeding point in the suture line which, if found, must be underrun with a mattress suture and carefully tied off.

The usual method of turning the corner with this suture is shown in figure 178. Occasionally, however, a loop-on-the-mucosa stitch is used, but it is not free from danger as, unless the individual loops are placed very close to one another and drawn fairly tight, hæmorrhage may occur at this site (fig. 179).

When the stitching has been completed along the posterior margin of the incision it turns the corner and returns along the anterior margin without interruption, locking, or knotting, until the end which was left long is reached, when a double knot is tied and the ends are cut short (fig. 180).

Just before this suture is completed the clamps are once more loosened and any bleeding point which is seen is oversewn.

The parts are then gently mopped with gauze soaked in warm saline, the stomach clamp is loosened, the jejunal clamp and mackintosh squares are removed, and all instruments which have been used are discarded. The surgeon and his assistants then change their gloves.

The sero-muscular stitch is picked up once more and introduced anteriorly as a continuous Lembert stitch to strengthen and invaginate the anterior suture line (fig. 181). It is carried slightly beyond where it originally started so as to add extra protection to a point in the suture line which might otherwise be weak. This plan of Moynihan's adds greatly to the finish and neatness of the operation (fig. 182).

The stomach clamp should be removed when the anterior sero-muscular suture is half completed, to permit the adjacent portions of the stomach and jejunum to be drawn together more easily and without tension. The suture line is again gently mopped, and if any weak point is detected a Halsted stitch is inserted.

The roll of gauze which was placed between the clamps before the suture was started is drawn upwards and rotated, first to the right and then to the left, to allow of inspection of the posterior lines of suture, and here again a stitch or two may be inserted if and where necessary. The roll of gauze is then removed.

The stomach, transverse colon, and omentum are once more drawn through the wound and the assistant picks up the omentum and colon

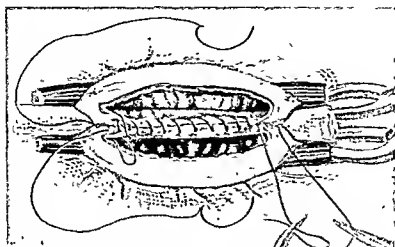


Fig. 179.—POSTERIOR GASTRO-JEJUNOSTOMY. THE SECOND CONTINUOUS SUTURE TURNING THE CORNER BY CHANGING INTO A CONNELL STITCH. THE LOOPS OF THE CONNELL STITCH SHOULD BE PLACED VERY CLOSE TO ONE ANOTHER AND DRAWN TIGHT TO PREVENT THE POSSIBILITY OF HÆMORRHAGE.

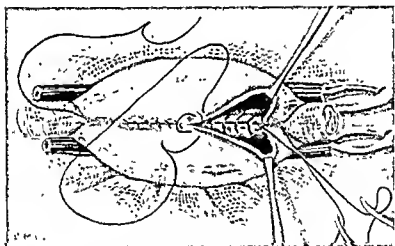


Fig. 180.—POSTERIOR GASTRO-JEJUNOSTOMY. THE SECOND (THROUGH AND-THROUGH) SUTURE NEARLY COMPLETED. (After Moynihan)

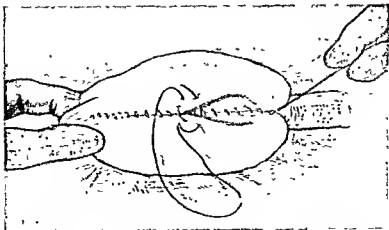


Fig. 181.—POSTERIOR GASTRO-JEJUNOSTOMY. THE OUTER SERO-MUSCULAR SUTURE IS NEARING COMPLETION.

and holds them upwards, whilst the surgeon gently draws the anastomosed portion well through the opening in the mesocolon and applies a clip to each margin. The edges of the mesocolon are sutured to the stomach on the right and left sides, about  $\frac{1}{4}$ – $\frac{1}{2}$  inch away from

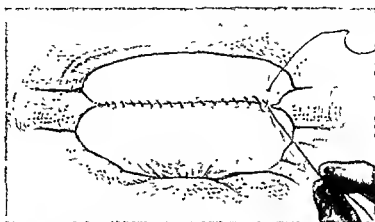


Fig. 182.—POSTERIOR GASTRO-JEJUNOSTOMY. NOTE THE METHOD OF COMPLETING THE SERO-MUSCULAR STITCH. (After Moynihan.)

the line of suture, the stitches being inserted in such a way that when the sutures are tied the edges of the mesocolon are turned inwards towards the lesser sac, leaving no rough surface to which coils of intestine might become subsequently attached (figs. 183 and 184).

#### *Causes of Unsatisfactory Results following Posterior Gastro-Jejunostomy*

These may be summarised as follows :

- (1) Faulty pre-operative treatment.
  - (a) Failure to eradicate all accessible foci of infection.
  - (b) Omission to treat chronic gastritis or chronic duodenitis when it is present to any marked degree.
  - (c) Not resorting to such pre-operative measures as washing out the stomach and prescribing the special diet and medicines required in cases of dilatation of the stomach.
  - (d) Failure to investigate the chemistry of the blood or to correct any alkalosis which may be present.
- (2) Faulty selection of cases for operation.

(3) Undertaking gastro-jejunostomy for disorders other than those of chronic gastric ulcer or chronic duodenal ulcer, e.g. gastro-ptosis, the gastric crises of tabes, etc.

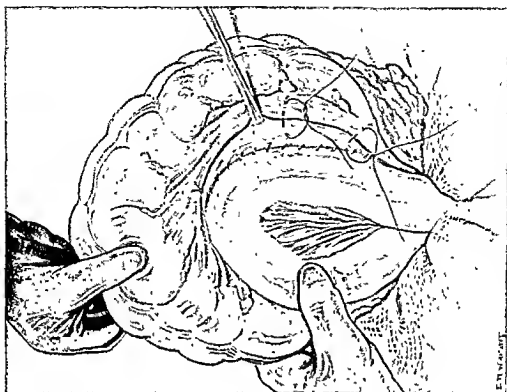


Fig. 181.—POSTERIOR GASTRO-JEJUNOSTOMY. SUTURES ATTACHING THE MESOCOLON TO THE STOMACH ON THE LEFT SIDE.

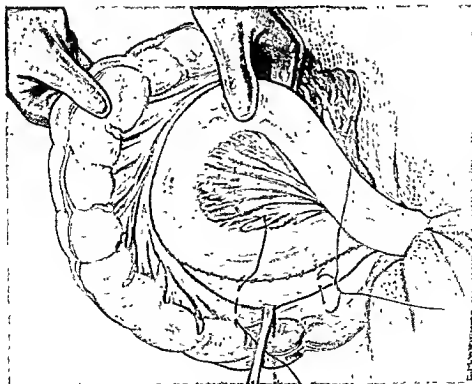


Fig. 182.—POSTERIOR GASTRO-JEJUNOSTOMY. FIXATION OF THE MESOCOLON TO THE STOMACH ON THE RIGHT SIDE.



(4) Lack of anatomical knowledge. There are cases on record where the stomach has been anastomosed to the transverse colon or to the ileum.

(5) Errors in operative technique :

- (a) Failure to excise, cauterise, or otherwise destroy a chronic gastric or chronic duodenal ulcer where this is possible.
- (b) Failure to remove an intra-abdominal septic focus when present, e.g. chronically inflamed appendix or gall-bladder.
- (c) Performing the operation with a jejunal loop which is either : (i) too long ; (ii) too short ; or (iii) rotated on its long axis. Any of these errors may predispose to regurgitant vomiting.
- (d) A badly planned opening. This may be : (i) too small ; (ii) too high ; (iii) too large ; (iv) not close enough to the greater curvature ; (v) made to the distal gastric pouch in cases of hour-glass stomach ; (vi) made with a junction which is too short ; or (vii) carelessly sutured, the anterior wall being stitched to the posterior wall.
- (e) An opening in the mesocolon which is : (i) too small ; (ii) left unsutured ; (iii) stitched to the jejunum or line of anastomosis, and not to the stomach as recommended ; (iv) sutured to the stomach in such a way that the margins are not inverted ; (v) carried too close to the colon ; or (vi) narrowed by the formation of a large hæmatoma which has resulted from one of the blood-vessels being punctured during the process of suturing the stomach to the margins of the mesocolon.
- (f) Adhesions which result from : (i) not peritonising the raw surfaces which remain after the jejunum has been separated from the mesocolon on account of physiological or pathological fusion ; (ii) infection ; (iii) the efferent loop of the jejunum becoming stuck to the abdominal scar ; or (iv) the use of irritant lotions, predisposing to plastic peritonitis.
- (g) Faulty methods of suturing ; the use of non-absorbable suture material ; making the anastomosis throughout with interrupted sutures ; tying large knots ; or drawing the sutures so tight that the tissues are strangled.
- (h) Faulty use of clamps, e.g. applying them too firmly.
- (i) Failing to detect duodenal ileus when present.

(6) Faulty post-operative management.

(7) The late development of anæmia, gastro-jejunitis, gastro-jejunal ulceration, carcinoma, or retrograde jejuno-gastric intussusception.

*Anterior Gastro-Jejunostomy.* In this operation a portion of the proximal jejunum, some 16-18 inches from the flexure, is chosen for the anastomosis, a long loop being necessary to prevent the transverse colon from being subsequently compressed by the loop.

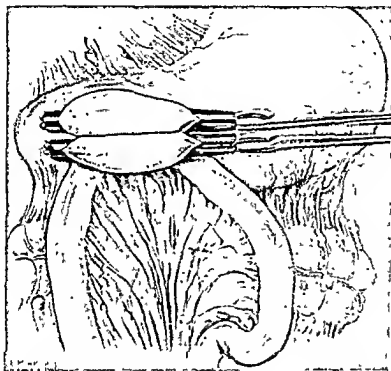


Fig. 185.—ANTERIOR GASTRO-JEJUNOSTOMY. THE CLAMPS IN POSITION PRIOR TO MAKING THE ANASTOMOSIS.

The duodeno-jejunal flexure is identified and the selected portion of the jejunum is brought over the transverse colon, clamped, and approximated to the stomach. The stomach clamp is applied somewhat obliquely so that the stoma in the stomach runs from above downwards, ending at the greater curvature at a point about 2 inches or so below the pylorus.

The clamped portions of the stomach and jejunum should never be less than 4 inches in length, as a wide area of attachment between the two organs is necessary to prevent kinking of the afferent or efferent limb of the jejunum (fig. 185). The size of the stoma itself, however, should not exceed 3 inches.

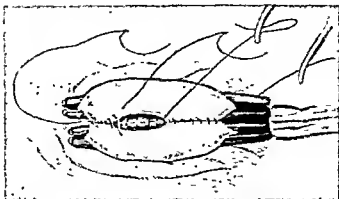


Fig. 186.—ANTERIOR GASTRO-JEJUNOSTOMY. NOTE THE OUTER SUTURE EMBRACING A MUCH LONGER PORTION OF STOMACH AND JEJUNUM THAN THE INNER. THE CONTINUOUS THROUGH AND THROUGH SUTURE IS NEARLY COMPLETED. X—THE GREATER CURVATURE. (After Mayo-Robson.)

The method of performing the anastomosis is the same as in the posterior operation, except that at the completion of the operation the jejunal loop must be anchored to the stomach in the manner shown in figures 186 and 187.

Those surgeons who content themselves with the performance of an anterior gastro-jejunostomy may take encouragement from the excellent results reported by Paterson in his per-

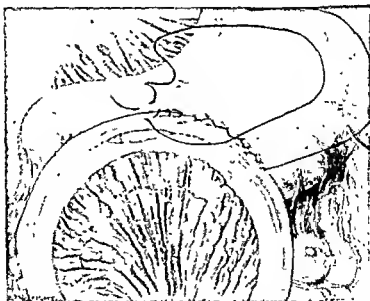


Fig. 187.—ANTERIOR GASTRO-JEJUNOSTOMY. THE OPERATION IS NEARLY COMPLETED AND THE JEJUNUM IS BEING SOWN TO THE STOMACH OVER A WIDE AREA TO PREVENT ANGIULATION OF THE JEJUNAL LOOPS. IN THIS ILLUSTRATION THE ANASTOMOTIC STOMA IS, FOR PURPOSES OF EXPLANATION, DEPICTED AS BEING TRANSPARENT. THE DOTTED LINES INDICATE THE POSITION AND EXTENT OF THE OUTER SERO-MUSCULAR SUTURE.

formance of this operation. He writes (*B.M.J.*, p. 676, March 30, 1935):

"I know . . . that 83 per cent of my patients are quite well, and this assuming that the untraced cases are not cured. If, as is done by some writers, the untraced cases are disregarded, the percentage of cures would be over 89 per cent.

Some years ago I analysed my results in 500 consecutive cases after gastro-jejunostomy performed more than six years previously. In that series three patients died after the operation and 89 per cent were quite well; in 8 per cent of the cases the results were unsatisfactory. Of this 8 per cent of unsatisfactory results 2.6 per cent were the result of jejunal or gastro-jejunal ulcer, 2.5 per cent of chronic diseases such as phthisis, chronic nephritis, or cancer elsewhere than in the stomach, and 3 per cent were due to hyperacidity."

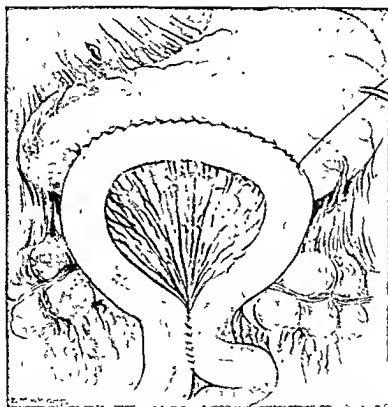


Fig. 188—ANTERIOR GASTRO JEJUNOSTOMY WITH ENTERO ANASTOMOSIS. THE OPERATION IS COMPLETED BY ANCHORING THE JEJUNUM TO THE STOMACH IN THE MANNER SHOWN, TO PREVENT ANY ANGULATION OF THE PROXIMAL OR DISTAL LOOPS OF JEJUNUM OR TRACTION ON THE SUTURE LINE OF THE GASTRO-JEJUNOSTOMY.

At the completion of this operation some surgeons perform an entero-anastomosis between the afferent and efferent limbs of the jejunum to prevent the possibility of regurgitant vomiting, but I consider that this added procedure is wholly unnecessary if the jejunum is widely attached to the stomach, as depicted in figure 188.

Again, this method is to be condemned, on the grounds that it unquestionably favours the development of secondary peptic ulceration.

*Retro-Colic Anterior or Posterior Gastro-Jejunostomy.* In certain cases, particularly in obese patients, where the stomach is small, or where numerous adhesions exist between its posterior surface and the pancreas; where, in fact, a posterior gastro-jejunostomy would be

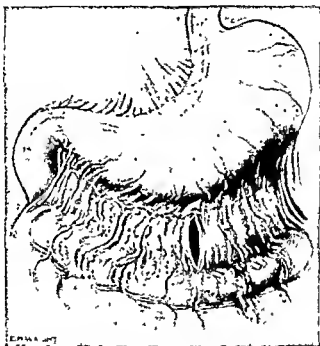


Fig. 189.—RETRO-COLIC POSTERIOR GASTRO-JEJUNOSTOMY. AN OPENING IS MADE IN THE GASTRO-COLIC OMENTUM IN A LINE WITH THE INCISURA. CARE MUST BE TAKEN NOT TO INJURE THE LARGE ARTERIES IN THE IMMEDIATE VICINITY OF THE GREATER CURVATURE. THROUGH THIS OPENING THE INTERIOR OF THE LESSER SAC AND THE POSTERIOR ASPECT OF THE STOMACH ARE EXAMINED.

difficult to perform owing to limited mobility of the stomach, a retro-colic gastro-jejunostomy may sometimes be preferable to an anterior gastro-jejunostomy.

The steps of the operation are as follows :

After the routine abdominal examination has been conducted and it is decided that the case requires a gastro-jejunostomy, the gastro-colic omentum is picked up at an avascular point, 1 inch or so below the greater curvature and in a line with the incisura, and is opened (fig. 189). The omentum in this region is very thin and the incision, which is



Fig. 190.—RETRO COLIC POSTERIOR GASTRO-JEJUNOSTOMY. THE OPENING IN THE GASTRO-COLIC OMENTUM IS WIDELY STRETCHED WITH THE FINGERS.

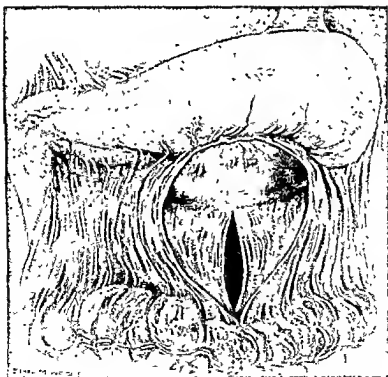


Fig. 191.—RETRO-COLIC POSTERIOR GASTRO-JEJUNOSTOMY. THE OPENINGS IN THE GASTRO-COLIC OMENTUM AND THE MESOCOLON ARE SHOWN.

readily enlarged by stretching with the fingers, gives free access to the lesser sac so that through it the whole of the posterior surface of the stomach can be methodically examined (fig. 190).

By the posterior method—which I would recommend in preference to the anterior—a portion of the most dependent part of the posterior wall of the stomach (usually opposite the incisura), about  $1\frac{1}{2}$  inches from the greater curvature, is picked up with two pairs of Allis forceps placed about 4 inches apart, thus producing a longitudinal fold which facilitates the application of the gastric clamp.

The transverse colon is then held upwards by the assistant, and an opening in the mesocolon is made in a bloodless area to the left of the middle colic artery (fig. 191). Through this opening a coil of the proximal jejunum is drawn, this being carried straight up without a loop and clamped at the site selected for the anastomosis, after making quite sure that there will be no undue tension either at the stoma or at the flexure (fig. 192).

The clamps are placed side by side, the operative field is packed off, and the operation is carried out as already described under gastro-jejunostomy (fig. 193).

Finally the anastomosis is drawn through the rent in the mesocolon and its margins are stitched to the stomach above the suture line.

By the anterior method the selected portion of the jejunum is anastomosed to the anterior surface of the stomach in an iso-peristaltic direction and as close to the greater curvature as possible. When the anastomosis is completed the openings in the gastro-colic omentum and mesocolon are stitched to the afferent and efferent loops of the jejunum to prevent herniation.

The many advantages claimed in connection with this operation may be briefly summarised as follows:

(1) The free opening in the gastro-colic omentum enables the whole of the posterior surface of the stomach to be carefully examined, and greatly facilitates the separation of adhesions.

(2) The making of the anastomosis is a simple matter, as all traction on the stomach is avoided and the more mobile organ, i.e. the jejunum, is brought up to the comparatively immobile organ, i.e. the stomach, instead of vice versa.

(3) The absence of pulling diminishes shock.

(4) In patients who are fat or where the stomach is retracted, an operation which would otherwise present considerable difficulty is rendered simple and straightforward.

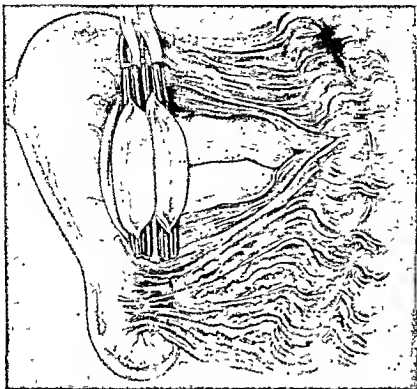


Fig. 103.—RETRO COLIC POSTERIOR GASTRO-JEJUNOSTOMY. THE CLAMPS HAVE BEEN APPLIED AND THE TWO POUCHES ARE READY TO BE ANASTOMOSED IN THE USUAL MANNER. AT THE COMPLETION OF THE ANASTOMOSIS THE OPENING IN THE GASTRO-COLIC OMENTUM IS CLOSED WITH A FEW INTERRUPTED SUTURES, AND THE ANASTOMOSED PORTION IS THEN GENTLY DRAWN THROUGH THE RENT IN THE MESOCOLON TO THE EDGE OF WHICH THE STOMACH IS LIVED BY A FEW INTERRUPTED SUTURES TO PREVENT HERNIATION.

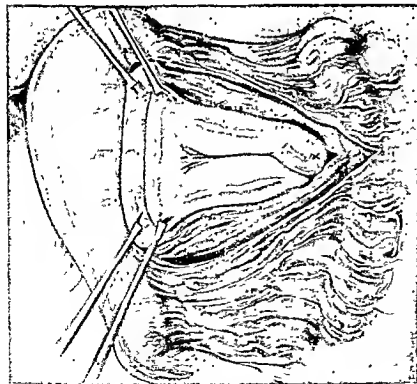


Fig. 102.—RETRO COLIC POSTERIOR GASTRO-JEJUNOSTOMY. X—DUODENO-JEJUNAL FLEXURE. THE PROXIMAL JEJUNAL LOOP HAS BEEN DRAWN THROUGH THE RENT IN THE MESOCOLON. THE ALLIS FORCEPS ARE GRASPING THE SELECTED PORTIONS OF THE POSTERIOR WALL OF THE STOMACH AND OF THE JEJUNUM TO FACILITATE THE APPLICATION OF THE CLAMPS.



Sherren and Stiles, the great exponents of this operation, preferred an anterior to a posterior anastomosis. They both performed the operation without a loop and attached the jejunum in an iso-peristaltic direction.

Ivor Back, however, speaks very favourably of the results he has obtained by the posterior method, and offers many valuable suggestions with regard to the performance of this operation (*Lancet*, 802, Oct. 7, 1933).

### *Partial Gastrectomy*

All partial gastrectomies are based on the principle of the Billroth methods.

There are many variations of the original Billroth I technique, three of which will now be described.

(1) Where the Billroth I operation is indicated, I would favour *Finochietto's method*, a detailed account of which is given on page 526.

(2) In the *Haberer-Finney* operation the first part of the duodenum, the pyloric segment, and a variable amount of the body of the stomach are removed. The duodenal stump is securely closed and the cut end of the stomach is anastomosed to the anterior aspect of the second part of the mobilised duodenum (fig. 194).

This operation possesses no advantages over *Finochietto's method*, is more difficult to perform, and, as far as I am aware, is very rarely, if ever, practised in this country.

(3) In *Schoemaker's* operation the first portion of the duodenum, the pyloric segment, and the greater portion or the whole of the lesser curvature are excised, often with the aid of special clamps such as those designed by Schoemaker, Morley, or Souttar.

After the lesser curvature has been reconstructed, an end-to-end anastomosis is performed between the cut ends of the stomach and duodenum (fig. 195).

Morley performed this operation for ulcer of the body of the stomach in 119 cases with 3 deaths, a mortality of 2.5 per cent, these 3 deaths all being due to pulmonary complications.

According to Morley, it is a safe and neither a lengthy nor a difficult operation to perform, the clinical results being remarkably good. It is also claimed that *Schoemaker's* operation does not abolish gastric digestion as the more radical *Polya* operations tend to do, and that it does not give rise to the post-operative secondary anaemia which is sometimes seen after the more extensive gastric resections.

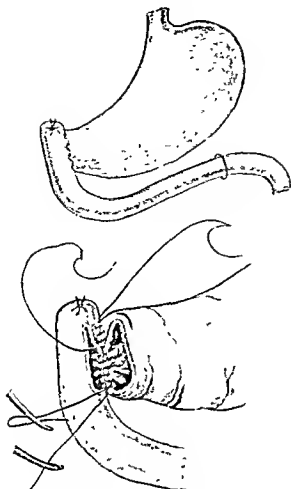


Fig. 104.—PARTIAL GASTRECTOMY BY THE HAREKER FINNEY METHOD.

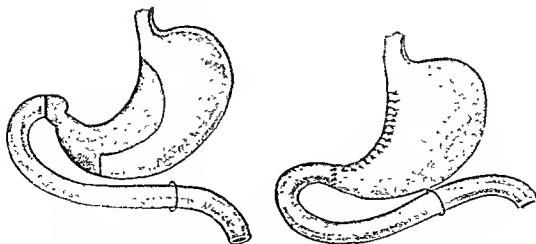


Fig. 105.—PARTIAL GASTRECTOMY BY SCHOEMAKER'S METHOD. THE SHADED AREA INDICATES THE PORTION OF STOMACH REMOVED. ON THE RIGHT, THE OPERATION COMPLETED.

But in spite of the many advantages claimed by the advocates of this operation, it is not likely to take the place of the more widely practised Polya methods.

The operations which are most frequently performed for chronic gastric ulcer are the anterior and posterior Polya types of gastrectomy.

In the *anterior Polya* type the proximal jejunum is brought in front of the transverse colon and anastomosed to the cut end of the stomach, the most popular method being Moynihan's modification, a detailed description of which is given on page 468. By this method the proximal jejunum is laid from left to right against

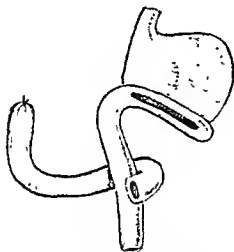


Fig. 196.—PARTIAL GASTRECTOMY BY ROUX'S METHOD OF ANASTOMOSIS IN Y.

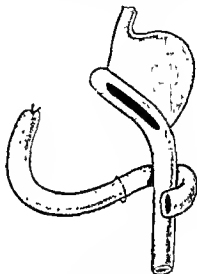


Fig. 197.—PARTIAL GASTRECTOMY. ROUX'S METHOD

the stomach, the distance between the duodeno-jejunal flexure and the proximal part of the anastomosis being usually not more than 4 inches, i.e. no loop is employed.

A number of surgeons, however, prefer Balfour's method (see page 491) whereby the proximal jejunum is laid in the opposite direction so that the afferent end of the jejunum is brought over to the lesser curvature.

When this operation is undertaken for chronic gastric ulcer, the making of the entero-anastomosis between the afferent and efferent limbs of the jejunum may predispose to the formation of jejunal ulceration. This operation, although sometimes performed for the cure of this condition, holds a more definite place in the treatment of cancer of the stomach.

Partial gastrectomy with anastomosis by *Roux's method in Y*

(figs. 196 and 197) is, to my mind, a most unsatisfactory operation and one which I now never practise, as in each of the three cases in which it appeared to be clearly indicated and in which I performed it for gastro-jejunal ulcer, it was subsequently followed by the development of secondary peptic ulceration.

Where, for one reason or another, a *posterior Polya* operation is indicated, I prefer the *Lahey* method of dealing with the proximal jejunal loop (see page 495), or *Finsterer's* physiological gastrectomy (see page 336).

### (G) POST-OPERATIVE TREATMENT

There are many schemes practised by surgeons in the post-operative management of cases after the performance of gastric operations, although the main principles are usually the same in each.

The following is a brief sketch of the methods which I have myself adopted with satisfactory results :

During the *first 24 hours* after operation no fluids are permitted by mouth, although the patient is encouraged to wash out his mouth frequently with some soothing antiseptic lotion. During this period water, salts, and glucose are introduced into the circulation by proctoclysis, intermittent intravenous injections, or by means of the intravenous continuous drip method.

Pain is relieved by one or more intramuscular injections of morphia, heroin, or omnopon.

Body heat is maintained by means of applications of mild radiant heat, special electrically heated blankets, hot-water bottles, etc.

Cardiac stimulants, such as coramine or digitalis, may be required.

The following mixture, given hypodermically 4-hourly for six doses, may be found useful :

R/	
Liq. adrenalin hydrochlor. (1 in 1000)	10 minims.
Strychnine hydrochlor.	$\frac{1}{50}$ gr.
Atropine sulph.	$\frac{1}{200}$ gr.

If there is much vomiting or distension a small stomach tube should be passed through the nostril into the stomach and the gastric contents aspirated, this being followed by very gentle irrigation with small quantities of warm normal saline. If vomiting is persistent the tube

should be passed at frequent intervals or left *in situ* so that the aspirations and irrigations can be carried out as required.

By gradual degrees the patient is placed in the Fowler position and maintained thus until he is fit to get out of bed.

Inhalations of  $\text{CO}_2$  and oxygen may be necessary during the first few hours after operation to ventilate the lungs thoroughly and to encourage deep breathing.

During the *second 24 hours* frequent mouth-washes are continued, but all fluids by mouth are still withheld.

The treatment during this stage is usually the same as that outlined above, with the exception that a sleeping draught is prescribed and a rectal flatus tube is passed from time to time.

During the *third day after operation* the patient is allowed small frequent sips of warm water to which glucose may be added.

During the *first three post-operative days* no purgatives are on any account administered, but on the morning of the fourth day an enema is given and this is repeated on subsequent days until the bowels are working normally, after which a mild laxative such as cascara evacuant may be given at night when required.

On the *fourth day* Lénhartz diet (see page 388) is prescribed and this is continued until the patient is fit to be discharged. He is also given 6-hourly doses of *cremor alkalinus* or one of the alkaline powders usually prescribed in the treatment of chronic peptic ulceration.

During the convalescent stage it is often advantageous to give intramuscular injections of iron and arsenic, such as Fraiese's mixture. After a wide gastric resection has been performed, liver and stomach extracts as well as ferrous salts may be found beneficial in preventing or combating post-operative anaemia.

Before the patient goes home he is given detailed instructions as to medicines and diet and a copy of Hurst's post-ulcer regime (see page 376).

An alternative scheme of diet, as advocated by Professor John Fraser, is given (see page 389).

## (II) POST-OPERATIVE COMPLICATIONS

"An adventure is a sign of incompetence. . . . If everything is well managed, if there are no miscalculations or mistakes, then the things that happen are only the things you expected to happen and for which you are ready and with which you can therefore deal." (Vilhjalmur Stefansson.)

The main post-operative complications in peptic ulcer cases may be grouped as follows :

- (1) *Hæmorrhage.*
- (2) *Vomiting.*
- (3) *Anæmia.*
- (4) *Anastomotic ulcer.*

#### HÆMORRHAGE

(a) *Immediate.*

- (i) *From the suture line.*
- (ii) *From the ulcer bed.*

(b) *Late.*

- (i) *From the ulcer bed.*
- (ii) *Due to a gastro-jejunal ulcer.*
- (iii) *Due to carcinoma of the stomach.*
- (iv) *Due to acute retrograde jejuno-gastric intussusception.*
- (v) *Due to blood disorders or to diseases of the spleen or liver.*
- (vi) *Due to gastro-jejunitis.*

Following most gastric operations the first few vomits are usually blood-stained, this being due in most cases to a slight oozing of blood from the suture line. Sometimes, however, it may be caused by rough handling of a gastric or duodenal ulcer which has proved irremovable. Should the patient continue to vomit bright blood, a serious view of the case must be taken.

Post-operative hæmatemesis is nowadays a very rare occurrence, as it is customary to excise or cauterise the ulcer, and special precautions are taken in suturing to prevent the danger of hæmorrhage at the anastomotic line.

Hæmorrhage is prevented by :

- (1) The use of continuous rather than interrupted sutures.
- (2) The use of an inner suture which embraces all the coats of the stomach and jejunum.
- (3) The introduction of the inner suture so that the individual stitches are placed very close to one another and are drawn together firmly and evenly.

(4) Inspection of the posterior suture line after the clamps have been temporarily released, and inspection of the anterior suture line just before the completion of the through-and-through suture for which the clamps are again momentarily loosened.

(5) The underrunning and ligature of any large blood-vessels near the margins of the anastomosis.

### *Immediate Treatment*

If the patient continues to bleed, the following measures are necessary :

(1) The pulse-rate is recorded every  $\frac{1}{4}$  hour on a special chart. A rising pulse-rate is a bad omen.

(2) The stomach is washed out through a large stomach tube with warm normal saline.

(3) Morphia,  $\frac{1}{4}$  gr., is given, with a further injection of  $\frac{1}{8}$ – $\frac{1}{4}$  gr. if required.

(4) Blood-transfusions are given and often prove successful either in arresting the hæmorrhage or in rendering the patient fit to stand a second operation should this prove necessary.

(5) Calcium gluconate (Sandoz) 10 cc. is injected intravenously at intervals of one hour up to three doses.

(6) No fluids or nourishment are permitted by mouth, but salines are introduced into the circulation by proctoclysis.

(7) The end of the bed is raised on blocks.

### *Operative Treatment*

If in spite of the above measures the pulse-rate is rapidly rising and or the patient *continues* to vomit bright blood, the abdomen should be opened.

The abdominal wall is thoroughly disinfected with tinct. metaphen. or with surgical spirit followed by dettol. The sutures or clips of the original skin incision are removed, the wound edges are separated, and tetra-cloths are affixed to its margins. The sutures of the abdominal wall are then snipped with scissors and removed, and the abdominal cavity is opened.

If the previous operation has been a *posterior gastro-jejunostomy*, one of the two following procedures may be adopted :

(1) The anterior wall of the stomach opposite the gastro-jejunostomy is opened and held apart with Allis forceps to permit of an inspection of the interior of the stomach and the anastomotic line. The gastric contents are mopped up or aspirated.

A stay suture is inserted at each end of the anastomotic opening and the gastro-jejunostomy is pulled up through the anterior wound in the stomach so that it can be thoroughly inspected. Any portion of the suture line which is bleeding briskly should be underrun and tied, and the parts then gently sponged to see that there is no oozing elsewhere along the suture line. If the oozing appears to be fairly general it is better to oversee the entire circumference of the gastro-jejunostomy with No. 0 20-day chromic catgut. Should oozing still persist at any particular spot, it must be controlled by the insertion of one or two interrupted sutures.

The traction sutures are then removed and the anastomosis is allowed to fall back, after which the anterior wound in the stomach is closed with a two- or three-tier suture.

(2) By the alternative method the transverse colon and omentum are drawn through the wound and lifted upwards, and the stitches in the mesocolon are snipped. Sherren clamps are applied to the stomach and jejunum and the anterior row of sutures in the anastomosis are cut through and withdrawn.

The outer margins of the wounds in the stomach and jejunum are held apart, the area is thoroughly cleansed, and the clamps released while the posterior suture line is inspected.

A new through-and-through suture is inserted, starting at one end of the posterior suture line and proceeding to the opposite end ; it then continues anteriorly, uniting the anterior margins of the stomach and jejunum firmly and evenly and in such a way that the risk of further hæmorrhage is practically eliminated. It is important to loosen the clamps just before the anterior row of sutures is completed. This anterior suture line is further reinforced and invaginated by a continuous Lembert suture, after which the margins of the opening in the mesocolon are stitched to the stomach on each side.

If in the previous operation the original ulcer was not dealt with, and if after inspection of the posterior row of sutures it is obvious that hæmorrhage is not arising from the suture line, a determined effort should be made either to excise the ulcer or to cauterise it ; if both



these procedures are impracticable, the blood-vessels in the vicinity of the ulcer should be underrun and ligated and an attempt made to oversew the ulcer.

During the operation all means of resuscitation should be at hand in case of need.

It should be remembered that post-operative hæmorrhage from a chronic peptic ulcer is in general more likely to occur in those patients who suffered from hæmorrhage before operation.

If the previous operation has been an *anterior gastro-jejunostomy*, clamps should be applied and the anterior row of sutures removed to permit of inspection of the posterior suture line.

A new through-and-through hæmostatic suture should be introduced, embracing all the coats of the stomach and jejunum, after which the anterior row of sutures is further reinforced and invaginated by a continuous Lembert suture.

Here, as in the posterior operation, the clamps should be loosened to permit of inspection when the suturing is half completed and again when it is almost finished.

If the previous operation has been a *gastro-duodenostomy*, the anterior row of sutures is unpicked, any bleeding point is underrun, and the posterior layer is reinforced. From this point onwards the steps of the operation are exactly similar to those of an ordinary gastro-duodenostomy.

Severe hæmatemesis immediately following *partial gastrectomy* is very rare, but when it occurs a secondary operation for its control is a hazardous undertaking and cannot as a rule be recommended. It is far better in such cases to rely upon repeated blood-transfusions, which often prove successful.

### VOMITING

Vomiting occurring during the first day or two after gastric operations is almost invariable and is due in most instances to delay in emptying of the stomach, to œdema of the stoma, or perhaps sometimes to a mild degree of acute dilatation of the stomach.

As the œdema subsides and the ileus becomes corrected, normal function is restored.

Persistent vomiting, severe enough to cause anxiety or to necessitate a secondary operation, is to-day very rare.

If the vomits are copious, the pulse-rate rapid, and the patient collapsed, and particularly if grave symptoms arise suddenly after a

period of apparently satisfactory progress, there is every possibility of acute dilatation of the stomach being present, and appropriate treatment should accordingly be instituted without delay (see page 165).

### *Palliative Measures*

In suspected cases of obstruction following gastric operations, the following palliative measures should always be given a trial for a period of 24-36 hours :

(1) Morphia,  $\frac{1}{4}$  gr., is injected and the dose repeated as required.

(2) No fluids are allowed by mouth.

(3) A stomach tube is passed through the nostril into the stomach and the gastric contents are aspirated, after which warm normal saline is used for irrigation. The amounts of fluid introduced and the amounts withdrawn from the stomach should be very carefully measured, as if the quantity withdrawn continually exceeds that which has been introduced it is almost surely an indication that there is some mechanical blockage.

(4) Water, salines, and glucose are introduced into the circulation by the continuous drip method, by occasional intravenous injections, or by proctoclysis.

(5) The vomited material is diluted with normal saline and introduced per rectum in small quantities at frequent intervals.

(6) The intake and output of fluids is very carefully noted on a special chart.

It is sometimes difficult to estimate the amount of urine passed, as some may be voided during the giving of an enema or the patient may suffer from a certain degree of incontinence. A decreasing urinary output, however, is always an ominous sign and may be the first indication of the onset of obstruction.

(7) 1 cc. of pitressin injected intramuscularly at hourly intervals up to four doses may be effectual in increasing peristaltic movement.

(8) A human-bile or ox-gall enema should be given to aid the passage of flatus and relieve distension.

It is always a very difficult matter to decide whether obstruction is serious enough to necessitate a secondary operation. Secondary operations should, as far as possible, be avoided as there are very few

experienced surgeons who have not on some occasion undertaken them with subsequent regret. If, however, it is clear that more fluid is being withdrawn from the stomach than is being introduced, if vomiting proves intractable, or if the amount vomited in 24 hours exceeds 50 oz., it is obvious that the stoma is not functioning and that some secondary operation must be carried out without further delay.

### *Secondary Operative Measures*

(1) Where obstruction follows any of the *Billroth I* types of operation, the operation recommended is a gastro-jejunostomy. If, however, the patient is very collapsed a Witzel jejunostomy performed under a local anæsthetic is the only procedure advisable.

Jejunostomy, in addition to supplying a means of feeding the patient, is also very useful for the re-introduction of the gastric contents which have been withdrawn through the stomach tube.

The surgeon should never be in haste to perform a secondary operation following the *Billroth I* types of partial gastrectomy, as gastric retention, vomiting, rapid pulse-rate, and considerable general disturbance are almost invariable for the first few post-operative days; in fact, intermittent vomiting may continue for several days. When once the œdema of the stoma subsides and the gastric function becomes normal, however, subsequent progress is usually uninterrupted and most gratifying.

(2) If the primary operation has been a *pyloroplasty* or *gastro-duodenostomy*, the secondary operation for relief of obstruction should be a gastro-jejunostomy.

(3) After the *Polya* types of operation obstruction is very rare. When it does occur it may be due to one of the following factors:

(a) Retraction of the anastomosis into the lesser peritoneal cavity.

(b) Adhesions or deformity of the distal loop of bowel which may become adherent to the anterior abdominal wall or to the under-surface of the abdominal incision.

(c) The use of too short or too long a jejunal loop. If the former, angulation may result or the taut gut may become compressed by the distended transverse colon; if the latter, the regurgitant vomiting may be due to waterlogging of the afferent limb.

Obstructive symptoms are much commoner where too short a loop has been used, as after the operation the stomach contracts upwards more than might be expected, dragging the already taut

loop with it and further flattening out the jejunum, kinking it at its line of attachment.

(d) Obstruction of the upper part of the jejunum proximal to the gastro-jejunostomy anastomosis may occur after excision of a large jejunal ulcer and is due in some instances to the invagination of too much of the bowel wall into the lumen of the jejunum while repairing an extensive defect following gastric resection for a jejunal ulcer.

(e) Obstruction may result at the stoma through too much of the anterior margin of the stomach having been turned in, thus leading to the formation of a valve. A similar condition may also be produced by invaginating too much of the stomach wall into the lumen of the jejunum in the region of the greater curvature of the stomach.

Regurgitant vomiting following the Polya types of partial gastrectomy should be treated by palliative measures, as outlined above, until the stoma commences to function normally or until it is obvious that a mechanical obstruction exists, requiring a secondary operation.

If the patient is *in extremis* the surgeon will have to be content with performing a Witzel jejunostomy; if, on the other hand, the patient appears to have sufficient strength to stand an exploratory operation the abdomen should be re-opened through the original incision, the wound edges well retracted, and the duodenum and the parts concerned in the anastomosis very carefully inspected.

If either the afferent or the efferent limb is adherent to some neighbouring viscus or to the parietes, it should be gently separated and prevented from contracting further adhesions by wrapping a portion of omentum around the affected segment of gut.

If kinking has occurred as a result of upward retraction of the small portion of stomach which remains after a posterior Polya gastrectomy, the sutures which anchor the gut to the rent in the mesocolon should be snipped through and the opening arranged and closed snugly in such a way that further kinking is rendered impossible.

If the obstruction is due to flattening and kinking after the use of too short a jejunal loop, or if there is any obstruction of the afferent or efferent limb or of the stoma itself, an entero-anastomosis between the two limbs or between the efferent loop and the duodenum itself is necessary.

In cases where the efferent limb is obstructed in the region of the greater curvature of the stomach or slightly beyond this, an alternative procedure to entero-anastomosis is to instruct the anaesthetist to pass an Einhorn tube through the nostril into the gastric pouch and for the surgeon to manipulate the end of this tube through the stoma into the

effluent loop well beyond the site of obstruction. This indwelling tube is used for feeding purposes after the operation, and also helps to dilate the obstructed portion of gut. It should be left *in situ* until normal function is restored.

In cases where obstruction occurs at the stoma itself due to the invagination of too much of the walls of the stomach and jejunum, in addition to passing this first tube into the effluent limb, a second tube should be passed by way of the other nostril, through the stoma, down the afferent limb into the duodenum. The duodenal contents are aspirated at frequent intervals and re-introduced through the tube which passes into the effluent jejunal loop.

This method worked very successfully with one case of mine in which the obstruction was undoubtedly due to the cause mentioned above.

(4) If the primary operation has been an *anterior gastro-jejunostomy* and this is followed by regurgitant vomiting due to the anastomosis becoming stuck to the under-surface of the abdominal wound or to the abdominal parietes itself, to the afferent portion of the long loop becoming waterlogged, or to the long loop itself becoming compressed by the colon, the secondary operation for relief of the condition should be a small entero-anastomosis between the afferent and effluent loops of the jejunum.

(5) Vicious circle vomiting following *posterior gastro-jejunostomy* may be due to one of many causes, but the following are probably the commonest :

- (a) The use of too long or too short a proximal loop of jejunum.
- (b) Insecure suturing of the anastomosis to the rent in the meso-colon, or such technical defects as making the opening in the mesocolon too small or suturing it to the jejunum.
- (c) Making the anastomosis at an unsuitable site.
- (d) Atony of the stomach.

It should be remembered that complete inability of the stomach to empty itself after gastro-jejunostomy may be due to atony of the stomach apart from any mechanical operative defects.

If the regurgitant vomiting is due :

- (i) To the use of too long a loop, this may be rectified by anastomosing the proximal and distal segments of the jejunum.
- (ii) To the anastomosis being too small, being improperly placed, or to the proximal loop being too short, more often than not it is best

to undo the anastomosis and make a new one rather than to attempt other secondary procedures, the success of which may be very doubtful.

After the tenth post-operative day disconnecting a gastro-jejunostomy may present considerable technical difficulties, and if these appear to be insuperable the surgeon is well advised to be content merely with performing a jejunostomy.

(iii) To œdema of the stoma when the mechanics of the operation appear to be satisfactory. In such cases a stomach tube should be passed through the nostril into the stomach and guided through the stoma into the efferent limb. Fluid nourishment and the gastric contents which have been either vomited or aspirated are introduced through this tube at frequent intervals.

Waltman Walters recommends the performance of a double jejunostomy in such cases, one tube being passed upwards along the efferent jejunal loop and through the stoma into the stomach, while the other is made to pass downwards into the lower reaches of the jejunum. The uppermost jejunostomy tube collects fluid from the stomach and this can be re-introduced into the lower jejunostomy tube which is also used for feeding purposes.

(e) Kinking or adhesions of the distal loop of the jejunum. Here again a tube passed through the nostril is manipulated through the stoma into the distal loop of the jejunum.

Walters recommends that a gastrostomy be performed and the tube led through the stoma into the efferent limb; but I consider my method to be less complicated and equally efficacious.

### *The Late Causes of Vomiting following Gastric Operations*

Vomiting occurring months or even years after gastric operations is always a very grave symptom and may be due to one of the following causes:

- (1) The formation of a new ulcer in the stomach or duodenum.
- (2) Gastro-jejunal ulceration.
- (3) The onset of cancer of the stomach.
- (4) Adhesions.
- (5) Contraction of the aperture in the mesocolon.
- (6) Retrograde jejuno-gastric intussusception.

*Retrograde jejuno-gastric intussusception* (fig. 198). The intussusception may be acute or chronic.

During the past 20 years some 36 cases of *acute retrograde jejuno-gastric intussusception* have been recorded. Adams (*B.M.J.*, No. 3866, p. 248, Feb. 9, 1935) states that the condition is commoner in women than in men, that cases have been reported as occurring from six days to sixteen years after operation, and



Fig. 198. -RETROGRADE JEJUNO-GASTRIC INTUSSUSCEPTION. GASTRO JEJUNO-STOMY HAD BEEN PERFORMED FIVE YEARS BEFORE DEATH. THE STOMACH HAD BEEN LAID OPEN TO SHOW A LARGE INTUSSUSCEPTION WHICH TOOK PLACE AS A RESULT OF THE STRAIN OF LABOUR AND ATTENDANT VOMITING.

(Museum, St Bartholomew's Hospital.)

that a correct pre-operative diagnosis is very rarely made, the usual being one of acute intestinal obstruction or of bleeding peptic ulcer.

There are two clinical types: One in which the patient is suddenly seized with an acute attack of epigastric pain followed by severe vomiting. On examination there is visible peristalsis, the waves passing from left to right, and a tumour can often be palpated in the epigastrium. These cases, being diagnosed as acute intestinal obstruction,

are usually operated upon immediately, thus rendering the prognosis favourable.

In the other type, which closely resembles a bleeding peptic ulcer, vomiting is frequent and becomes first blood-stained and then definitely hæmorrhagic. A provisional diagnosis of bleeding ulcer is usually made, and the patient receives treatment by medical measures accordingly, thereby leading to delay in operation with a consequent high death-rate.

As spontaneous reduction never occurs, all such cases upon which no operation is performed will die within a few days. If, however, operation is undertaken at once the outlook is good and some 90 per cent of cases will recover.

From literature Adams reports 30 cases which were subjected to operation, of which 9 died and 21 recovered. He also reported one successful case of his own.

The *chronic* variety is characterised by intermittent and sometimes severe vomiting occurring at a remote date after gastro-jejunostomy. X-rays are useful in forming a diagnosis, as the intussusception displaces the barium meal and so produces a filling defect. The rounded shadow produced by the ectopic coils of jejunum in the stomach tends to change its position on palpation and is marked with striations due to the folds of jejunal mucosa.

Adams emphasises the following points in connection with this rare complication after gastro-jejunostomy :

(1) Jejunogastric intussusception is a well-established late complication of gastro-jejunostomy for peptic ulcer. It occurs in acute or chronic forms, the former being fatal apart from operation.

(2) Diagnosis is possible, depending upon the rule—"where hæmatemesis or obstructive symptoms appear after gastro-jejunostomy, think of intussusception."

(3) If in sudden acute intestinal obstruction the following triad is present, then acute retrograde intussusception should be diagnosed :

(a) Epigastric scar.

(b) Visible peristalsis, the waves passing from left to right.

(c) A palpable mobile swelling about the mid-abdomen.

(4) The chronic form is to be remembered as one of the causes of recurrent vomiting after gastro-jejunostomy. Early radiography is demanded in such cases and will reveal the intussusception if present and pave the way for a curative operation.



The following operative procedures have been practised :

- (1) Reduction of the intussusception.
- (2) Resection of the anastomosis.
- (3) Suturing the afferent and efferent loops together after reduction of the intussusception.
- (4) Entero-anastomosis, the afferent loop being anastomosed to the efferent loop.

Debenham (*B.M.J.*, p. 250, Feb. 9, 1935) reported an interesting case of retrograde intussusception of the jejunum following gastro-jejunostomy, and offers some useful suggestions with regard to operative treatment.

#### ANÆMIA

Anæmia may occur after gastric operations. *It is usually of the hypochromic microcytic type and responds very rapidly to treatment with adequate doses of ferrous salts.* A few cases of pernicious anæmia have been recorded after gastric operations and particularly after partial gastrectomy. From literature Rowlands and Simpson (*Lancet*, p. 1202, Dec. 3, 1932) were able to collect 15 cases of Addison's anæmia, plus two cases suffering also from sub-acute combined degeneration of the cord. Five cases occurred after total gastrectomy, eight after partial gastrectomy, and four after gastro-jejunostomy, these operations being performed for carcinoma, for gastric ulcer, and (in one case) for duodenal ulcer. The period between the operation and development of the anæmia varied from 2-15 years, the average being 6 years.

Gordon-Taylor investigated the results of 52 cases of partial gastrectomy and found that marked anæmia was present in 8 and severe anæmia in 15. But in no instance did the anæmia resemble that of the Addisonian type.

Morley quotes very similar figures and is of the opinion that the amount of stomach removed is an important ætiological factor and that following the Billroth I types of operation, and particularly after Schoemaker's method, anæmia is very rare.

Hartfall (*Guy's Hospital Rep.*, lxxxiv, 448, 1934) in a study of 40 cases of anæmia following gastric operations states that the anæmia may be as severe after gastro-jejunostomy as after partial gastrectomy. He considers that the amount of stomach removed has no constant relation to the development of anæmia and that the anæmia is dependent

rather upon the functional disturbance produced by the operation in the particular case; but from his figures it would appear possible that anæmia is more common after the Polya types of operation. He also adds that the presence or absence of free hydrochloric acid does not seem to affect the development of this condition.

Gordon-Taylor stresses the fact that anæmia is much more likely to follow wide resections in women than in men, but that it does not appear to influence the final results as regards appetite or capacity for work, and that a large number of patients, although developing anæmia, are very little affected by it.

Lake, on the other hand, investigated 51 cases of partial gastrectomy and found the blood counts to be approximately normal in all. Achlorhydria was present in all but one case. Similar results have been obtained by Ogilvie and myself.

In my series I have had only one case of severe anæmia, which occurred after total gastrectomy for leather-bottle stomach. This patient lived for ten months after operation.

The sum total of evidence would seem to indicate that Addisonian anæmia is extremely rare after partial gastrectomy or gastro-jejunostomy. It is, nevertheless, a wise prophylactic measure to prescribe iron, arsenic, and copper salts, as well as liver and stomach extracts, in all cases after extensive gastric resections.

#### ANASTOMOTIC ULCER

Garnett Wright suggests that a good title for this condition is secondary peptic ulcer, and that the individual ulcers should be called jejunal, anastomotic (when occurring at the line of anastomosis), or duodenal (when occurring after gastro-duodenostomy). Other familiar titles are marginal ulceration, stomal ulcer, and recurrent ulcer.

Paterson distinguished two kinds of these ulcers—jejunal (located in the jejunum) and gastro-jejunal (when the ulcer was situated at the anastomotic line but extended to the gastric or jejunal mucosa or to both).

#### *Ætiology and Pathology*

(1) *Incidence.* It is difficult to assess correctly the incidence of secondary peptic ulceration, as the figures given by various authorities range from 1·6 to 34 per cent, and a number of authors in their analyses

of cases do not specify whether the ulcers occurred after operations for gastric, pyloric, or duodenal ulcers.

It is universally agreed that gastro-jejunostomy for duodenal ulcer is the operation most frequently followed by anastomotic ulcer. Moynihan (1919) gave the incidence as occurring in 1.6 per cent of the cases analysed by him; Paterson (1909) 2.4 per cent; Luff (1929) out of 744 cases was able to trace 21 cases—2.8 per cent; Balfour (1930) 3 per cent; and Walton (1935) 3.9 per cent after all gastro-jejunostomies performed by him for duodenal ulcer and watched for 10 years. Garnett Wright (1935) places the incidence at 8.75 per cent, whilst Ogilvie (1935) assesses the total incidence at 20 per cent. Friedman (1928) 25 per cent, and Lewisohn (1925) in a series of 68 cases of gastro-jejunostomy for duodenal ulcer re-examined from 4-9 years after operation found anastomotic ulcer in 34 per cent. My own estimate of the occurrence of secondary peptic ulceration following gastro-jejunostomy for duodenal ulcer *without stenosis* is from 6-8 per cent.

Hurst considers that secondary peptic ulcer is a dangerous and frequent sequel of gastro-jejunostomy, and claims that the number of cases in which the complication occurs after this operation is found to multiply slowly and steadily as such patients are watched over increasing periods. This view is supported by Ogilvie and a number of other surgeons.

Although it is generally claimed that stomal ulceration is more rare after gastro-duodenostomy than it is after gastro-jejunostomy for simple duodenal ulcer, the incidence is probably about the same. Grey Turner (1921) found recurrence in 4 out of 43 cases, and very similar figures are quoted by others.

The condition seldom occurs after gastro-jejunostomy for pyloric stenosis—about 1 per cent, or after physiological gastrectomy for simple duodenal ulcer provided that an ample portion of the acid-bearing area of the stomach is excised. As I have emphasised elsewhere, when undertaking this latter operation in the treatment of such cases it is essential that at least three-quarters of the stomach should be removed, as the results of a pylorotomy and of a timid resection are found to be equally poor.

Gastro-jejunostomy performed at the time of an acute perforation is especially liable to be followed by secondary ulceration, some authors placing the incidence as high as 10-15 per cent.

*A particularly pernicious operation is Roux's method in Y or a combination of anterior gastro-jejunostomy with entero-anastomosis, and*

*neither of these operations should in any circumstances be advised as a primary operative procedure for primary peptic ulceration.*

The incidence of marginal ulceration after gastro-jejunostomy for gastric ulcer is comparatively rare, probably not higher than 5 per cent. When this operation is combined with V-excision of the ulcer or with its destruction with the cantery after Balfour's method, the likelihood of ulceration is still further diminished, i.e. to about 2 per cent. When, however, partial gastrectomy is undertaken for gastric ulcer, provided a sufficient amount of stomach is removed to produce achlorhydria, the complication is almost unknown.

Anastomotic ulcer may, for all practical purposes, be said never to occur after operations for carcinoma of the stomach. Judd reported one case and other instances may be found in literature; but Garnett Wright (*Br. Jl. Surg.*, Vol. xxii, p. 433, Jan. 1935) was unable to find a single case out of 436 which were traced and offers the following possible explanations for the absence of this complication:

- (a) That carcinoma patients are immune from secondary ulceration.
- (b) That these patients do not live long enough for secondary ulcers to develop; but this is only true in part as secondary ulcers may appear very quickly.
- (c) That symptoms of secondary ulcer are likely to be masked by those of carcinoma.

It is probable that the infrequency of secondary ulceration after operations for carcinoma is due to the low acidity and possibly also to the diminished peptic activity of the gastric juice.

(2) *Age.* The commonest period of life at which stomal ulceration occurs is between the ages of 30 and 60, with the highest incidence between 40 and 50.

(3) *Sex.* Recurrent peptic ulceration after gastro-jejunostomy for duodenal ulcer is twenty times commoner in males than in females, whereas after operations for gastric ulcer the ratio is approximately 3 males to 1 female.

(4) *Family History.* The tendency for duodenal ulcer to occur in families has already been mentioned, but, as Hurst has said, a family history is still more commonly obtained in cases of secondary peptic ulceration.

(5) *Site of Ulceration.* These ulcers may occur at the line of anastomosis and extend from this point into the stomach or jejunum or

into both. When occurring here they cause considerable scarring and eventually lead to partial or complete stenosis of the stoma. They are not so prone to perforate as true jejunal ulcers, but may be associated with severe bleeding. Jejunal ulcers, so-called when the ulcer lies within the jejunum, are usually situated from  $\frac{1}{2}$ –1 inch away from the stoma and, like those in the anterior wall of the duodenum, are located in a thin unsupported wall and consequently tend to perforate early, though they rarely give rise to any marked degree of stenosis (fig. 199).



Fig 199.—JEJUNAL ULCER (Reproduced from "Surgery of the Stomach and Duodenum," by Dr. J. Shelton Horsley, Kingston. By kind permission.)

Jejunal ulcer occurring in the afferent limb is extremely rare owing to this loop of gut being bathed in alkaline juice. Stomal ulcers usually occur singly but in certain cases they may be multiple.

(6) *Healing.* Spontaneous healing of secondary peptic ulcers is very rare, much more so than in the case of chronic gastric or duodenal ulcer. Hurst states that if the condition is recognised early and strictly treated by medical measures, healing may be brought about successfully in some cases. Such measures, however, should not be persisted with unless there is convincing evidence of rapid and favourable response to the treatment, as complications, such as perforation or hæmorrhage, are very prone to occur.

*Pathogenesis*

(1) *The Acid Factor.* It is agreed by everyone that the production of these ulcers depends in some way upon the action of the gastric juice and that the true cause is closely bound up with the cause of primary gastric or duodenal ulcer.

Most authors maintain that the presence of free hydrochloric acid is an important factor, and explain its absence in certain cases by the fact that chronic gastritis may develop as the result of the secondary peptic ulceration itself.

Roscoe, Graham, and Lewis (*Jl. Amer. Med. Assoc.*, p. 386, Feb. 2, 1935) suggest that, except in cases of chronic gastritis, hyperchlorhydria is always present in cases of stomal ulcer and that an error may arise through not ensuring that the tube used for aspiration is actually in the stomach itself; it may inadvertently be passed through the stoma into the duodenum or into the efferent limb of the jejunum, and thus withdraw intestinal instead of gastric contents.

(2) *The Infective Factor.* Probably one of the most important causes of secondary peptic ulcer is the presence of infective foci in connection with the teeth, tonsils, nasal sinuses, appendix, or gall-bladder. Infection may also occur from an unhealed chronic peptic ulcer.

Bohmansson (1926) drew attention to the possible influence of gastritis in the development of stomal ulceration, and Konjetzny has shown how frequently gastritis is associated with gastric and duodenal ulcer. An anastomosis between the inflamed mucous membrane of the stomach and the healthy mucous membrane of the jejunum may heal imperfectly and thus predispose to the formation of ulceration. The importance of postponing operation until gastritis, when present, has been successfully dealt with, has already been stressed.

(3) *Technical Errors.* Technical errors in the performance of the original operation may slightly increase the frequency of the lesion but are not the primary cause.

It was at one time thought that the use of *non-absorbable suture material* accounted for a large number of cases, but there is not sufficient proof that the use of an absorbable suture is any safeguard against secondary ulceration, as the complication appears to be equally common after the use of silk or catgut.

It has been suggested that the crushing effect of *intestinal clamps* used during the performance of the anastomosis may be responsible

for trauma of the mucous membrane which, under the influence of the gastric juice, may, in turn, initiate and perpetuate the process of ulceration. Clamps, however, when employed with ordinary care, cannot be said to play an important part in the production of the lesion, as ulceration sometimes occurs apart from their use (fig. 200).

*Excision of the redundant gastric and jejunal mucosa* before proceeding with the continuous hæmostatic suture has been blamed by some for the occurrence of secondary ulceration (Grey Turner). It was thought that certain raw surfaces which resulted at the line of anastomosis, being unprotected by mucosa, came freely into contact with the

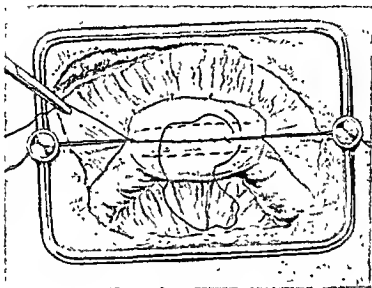


Fig. 200.—POSTERIOR GASTRO-JEJUNOSTOMY WITHOUT THE AID OF CLAMPS.  
LANG'S FRAME IS BEING USED.

eroding influence of the acid gastric juice to which they were unaccustomed, with the consequent production of ulceration. But as ulceration appears to be equally common when the redundant mucosa is not excised, this cannot be said to be an important factor.

At one time it was thought that *temporary occlusion of the pylorus* following gastro-jejunostomy was a factor which increased the incidence of stomal ulceration. There is ample evidence to show that temporary occlusion with a silk ligature has very little bearing upon the increased incidence. Walton frequently employs this method, but the incidence of marginal ulceration in his cases would seem to be, if anything, lower than in those reported by other surgeons who do not adopt this procedure.

An argument in favour of the view that technical errors are largely

responsible for recurrent peptic ulcer was that these ulcers usually seemed to occur within the first year or two after operation. Wright, in his series, however, found that in only 284 out of 458 cases, i.e. just over 62 per cent, did the symptoms appear within two years.

To me this does not seem to favour the view that technique plays an important part in the production of secondary ulcer, as in such a large proportion of cases the onset is so delayed that it is difficult to believe that technical errors could have had any influence.

When the complication arises shortly after operation—within a month or two—it would appear that, in some cases at least, technique is at fault, although no one particular error seems to characterise these failures in any large series of cases investigated.

### *Signs and Symptoms*

(1) *Pain.* Pain is the most prominent symptom and is more intense and persistent than in cases of gastric or duodenal ulcer. Although it is usually confined to the epigastric region, it may be diffused over the whole abdomen, or be localised to the lower abdomen or more frequently to an area slightly below and to the left of the umbilicus. Unlike the periodic pain which is so typical of gastric and duodenal ulcer, it is generally continuous and is less influenced by the intake of food.

In certain cases of gastro-colic fistula pain may be absent owing to healing of the ulcer.

(2) *Vomiting.* Vomiting, which varies in intensity and frequency, takes place in some 50 per cent of cases. It is often self-induced and is the most speedy method of obtaining relief from pain.

(3) *Condition of the Bowels.* Constipation is the rule, but when a gastro-colic or gastro-jejuno-colic fistula develops there is usually very persistent diarrhoea.

(4) *Wasting.* This is generally slight at first, but becomes more marked in those cases in which there is fistula formation.

(5) *Hæmorrhage.* Hæmorrhage—hæmatemesis, or severe melaena—is a common complication of gastro-jejunal ulceration, often demanding urgent surgical measures. A grave form of anæmia may result from the persistent oozing of blood from the ulcer bed.

On examination there is usually tenderness on palpation, especially marked in the epigastrium, but occasionally all over the abdomen and



particularly on the left side. The pain and tenderness seem to be distributed over a much wider area than is the case with gastric or duodenal ulcer. In rare instances, where recurrent ulceration occurs after anterior gastro-jejunostomy, the ulcer may become fixed to and actually erode the abdominal wall, producing a palpable mass.

### *Radiological Findings*

It is difficult to diagnose secondary peptic ulceration by means of X-rays. Usually, however, a positive diagnosis can be made on one or a combination of the following findings :

- (1) Tenderness over the stoma.
- (2) Deformity of the stoma.
- (3) Stenosis of the stoma.
- (4) Residue in the region of the stoma.
- (5) Delay in emptying of the stomach.
- (6) Ulcer crater (fig. 201).
- (7) Gastro-colic or gastro-jejuno-colic fistula.
- (8) Deformity of the efferent loop of the jejunum.
- (9) Ulcer of the stomach situated near the stoma.

### *Complications*

The following complications may occur :

- (1) Perforation.
- (2) Hæmorrhage.
- (3) Fistula.
- (4) Duodenal obstruction.
- (5) Adhesions.

*Perforation* is probably the commonest complication of secondary peptic ulcer, and is estimated to occur in approximately 15 per cent of all cases. After anterior gastro-jejunostomy perforation may occur into the general peritoneal cavity when the signs and symptoms will be indistinguishable from those of perforation of a primary peptic ulcer, or the ulcer may become stuck to the anterior abdominal wall, perforate it, and slowly give rise to an external fistula.

After posterior operations there is a tendency to chronic perforation into the mesocolon which becomes œdematous, thickened, and later

contracted, a state of affairs which is very prone to produce distortion of the jejunal loop and chronic duodenal ileus.

Perforation may also occur into a neighbouring viscus, such as the transverse colon, giving rise to gastro-colic fistula.

*The treatment recommended is simple closure of the perforation. If there is much contamination of the peritoneal cavity, drainage also will be required.*



**Fig. 201.—GASTRO JEJUNAL ULCER.** THE ULCER HAS FORMED A LARGE CRATER IN THE DISTAL LOOP OF THE JEJUNUM ABOUT ONE INCH BELOW THE GASTRO-JEJUNOSTOMY. NOTE THE SPASTIC CONTRACTION OF THE STOMACH AND OF THE JEJUNUM AT THE GASTRO-JEJUNOSTOMY. THE PATIENT, A THIN SCROFULOUS MAN, HAD A POSTERIOR GASTRO-JEJUNOSTOMY PERFORMED FOR A CHRONIC DUODENAL ULCER. AT A SECOND OPERATION, PERFORMED BY THE AUTHOR THREE YEARS LATER, THERE WAS NO EVIDENCE OF A DUODENAL ULCER AND THE ANASTOMOSIS WAS THEREFORE DISCONNECTED. AFTER EXCISING THE ULCER WITH A PORTION OF THE JEJUNAL WALL, THE PARTS WERE RECONSTRUCTED AND THE CONTINUITY OF THE ALIMENTARY CANAL RE-ESTABLISHED. THE PATIENT HAS SUBSEQUENTLY REMAINED IN GOOD HEALTH.

Following the operation the patient should be strictly treated by medical measures, but if no improvement results secondary operation, often amounting to partial gastrectomy, will be necessary.

The operative mortality of simple suture of a perforated jejunal ulcer is about 25 per cent.

*Hæmorrhage*, as stated above, may be so severe as to necessitate immediate surgical interference, but the majority of cases will show at any rate a temporary response to medical treatment.

*Fistula* occurs in some 10 per cent of cases of anastomotic ulcer. The fistula may be jejuno-colic, gastro-colic, or gastro-jejuno-colic. In very rare instances an external fistula may occur, and this is most

commonly seen where secondary peptic ulceration has followed anterior gastro-jejunostomy.

Following the formation of a gastro-colic or gastro-jejuno-colic fistula, rapid healing of the stomal ulcer may result owing to the neutralising effect of the colonic contents, or possibly to the effect of a superadded gastritis which frequently follows the perforation of the stomach into the colon.

The fistulous tract often becomes lined with mucosa, and when this takes place pain arising from the ulcer may cease.

As soon as the fistula becomes established, vomiting is frequent and faecal. There is often belching of foul-smelling gas. This belching of gas with a faecal odour, which occasionally occurs in cases of obstruction of the stomach due to a necrotic fungating growth, is a very important diagnostic sign. Diarrhoea is often intractable, and undigested food may be detected in the stools. There is often marked emaciation.

An X-ray examination after the administration of a barium meal or barium enema confirms the presence of a fistula, and on gastric analysis faecal material may be found in the stomach contents.

*Treatment.* The following operations have been practised:

(1) Restoration of the normal alimentary passage.

(2) Wilkie's two-stage operation. Wilkie considers that this is the method of choice for the larger gastro-jejunal fistulae where marked faecal regurgitation is present, where the patient is anæmic, and where all the tissues around the fistula are oedematous, friable, and infected. By excluding the portion of the colon involved in the fistula and allowing a period of some weeks or months to elapse, a relatively clean field can be obtained for the second and major stage of the operation (Wilkie, *Annals of Surgery*, Vol. 99, No. 3, p. 401, March, 1934).

(3) A one-stage radical operation which will consist of:

- (a) Resection with end-to-end union of the jejunum.
- (b) Resection with end-to-end union of the colon, or resection followed by Paul's operation.
- (c) Resection of the stomach; and
- (d) Anastomosis of the cut end of the stomach to the jejunum.

The various operations for the cure of these fistulae are similar to those practised for stomal ulcer, but there is the added procedure of separation of the portions of gut involved in the fistula, followed by

either (i) closure of the hole in the colon ; (ii) partial colectomy followed by end-to-end union ; or (iii) Paul's operation.

*Duodenal Obstruction.* Owing to the formation of adhesions and to the fibrosis and thickening of the mesocolon which so frequently accompanies gastro-jejunal ulceration, producing secondary duodenal ileus, the necessity of performing duodeno-jejunostomy at the completion of the main operative procedure should be borne in mind.

### *Treatment of Anastomotic Ulcer*

" If gastro-jejunostomy were only performed when there is a visible and palpable ulcer causing obstruction of the pylorus or duodenum ; if before the operation every possible source of infection in the mouth and pharynx were removed ; if at the operation any associated disease of the appendix or gall-bladder were dealt with ; if after the operation the patient were given adequate instructions with regard to diet and told to take additional feeds between meals ; and if moderation in smoking and the taking of olive oil before meals were also enjoined ; then the chances that a gastro-jejunal or jejunal ulcer might develop would be reduced to a minimum. Under such conditions these complications would rarely, if ever, arise in the absence of some gross error in technique." (Hurst & Stewart, *Gastric and Duodenal Ulcer*, p. 491, Oxf. Med. Publ.)

Whether operation is performed or not, the mortality of this complication is in the region of 22 per cent. Operation is recommended for perforation, hæmorrhage, fistula, duodenal obstruction, or for the relief of intractable pain.

The following operations have at one time or another been performed for anastomotic ulcer :

- (1) Local operation, e.g. excision of ulcer.
- (2) Reconstruction of the normal alimentary passage.
- (3) Excision of gastro-jejunostomy followed by gastro-duodeno-stomy or pyloroplasty.
- (4) The construction of a new gastro-jejunostomy.
- (5) Entero-anastomosis.
- (6) Partial gastrectomy.

*Local Operation.* This usually consists of excision of the ulcer with or without some plastic procedure upon the stoma, but is a most unsatisfactory undertaking and is followed by a very high percentage of recurrences.

*Reconstruction of the normal alimentary passage* involves disconnecting the gastro-jejunostomy, excision of the ulcer, and closure of

the openings in the stomach and jejunum. This is always a severe measure and includes what is, in fact, the hardest step in any operation for anastomotic ulcer, i.e. the freeing of the anastomosis from the mesocolon. The mortality of 20 per cent is high, and not more than 20 per cent of the cases are cured. The failures are due to the recurrence of the original duodenal ulcer.

The operation of *restitutio ad integrum* is indicated only where there is no evidence of duodenal ulcer, it being apparent that the primary operation was needlessly undertaken, or where the pylorus and duodenum are widely patent and there is either no visible scar or only a very slight one. When, however, there is an active duodenal ulcer or pyloric stenosis is present, *excision of the gastro-jejunostomy* may be followed by *gastro-duodenostomy* or *pyloroplasty*; but in the presence of such conditions I prefer partial gastrectomy to either of these procedures.

The construction of a new gastro-jejunostomy may entail:

- (a) The making of a new anastomosis, leaving the old one intact.
- (b) Resection of the old gastro-jejunostomy followed by re-suture; or
- (c) Resection and closure of the old gastro-jejunostomy followed by the establishment of a new one in another position.

All these procedures are to be deprecated, as fully one-third of the cases thus treated develop recurrent ulceration at the site of the new anastomosis.

*Entero-anastomosis*—a rare procedure—can only be summarily condemned, as it contravenes all the principles of surgery for peptic ulcer.

*Partial gastrectomy* offers by far the best chance of cure for secondary peptic ulceration—60 per cent. In such cases the posterior Polya operation has an operative mortality of about 20 per cent; the anterior operation 15 per cent.

By this method at least three-quarters of the stomach is removed to ensure alkalinisation and to guard against recurrence of ulceration, for relief of which any subsequent operative procedure is fraught with considerable danger and infinite difficulty.

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## CHAPTER VII

### NEW GROWTHS OF THE STOMACH

(A) CARCINOMA OF THE STOMACH

(B) SARCOMA OF THE STOMACH

(C) INNOCENT NEW GROWTHS OF THE STOMACH

#### (A) CARCINOMA OF THE STOMACH

CANCER of the stomach can be cured by operation, and by this method alone can the patient be saved. All non-surgical methods of treatment have a mortality of 100 per cent. As fully 70 per cent of patients suffering from cancer of the stomach are beyond the reach of radical surgery when seen by the surgeon for the first time, it is obvious that no improvement is possible in the present situation unless these cases are referred to him at a much earlier period in their illness than they have been hitherto. The fight against cancer is a fight for earlier diagnosis, and thus for earlier treatment.

#### ÆTIOLOGY

(1) *Frequency.* The stomach is the commonest site of malignant disease.

Häberlein found that of 27,511 cases of fatal carcinoma examined at autopsy 41·5 per cent were situated in the stomach.

D'Espère calculated that 44·3 per cent of all cancers occurred in the stomach.

Rowlands (*B.M.J.*, p. 905, May 21, 1933) estimated that for England and Wales there was in 1933 a death-roll of 19,000 from this disease.

Graham (*Canad. M. Ass. J.*, 18:25, 1928) considers that the economic seriousness of this disease can best be appreciated by the fact that in the U.S.A. 90,000 people die of carcinoma each year. Of this number, 34,000 die of carcinoma which originates in the stomach.

Balfour considers that the stomach is the most frequent site of malignant disease in *both sexes*, and that in some countries the statistics appear to show that cancer occurs in this organ as frequently as it does in all other situations put together.

The general conservative estimate is that one-third of all cancers in men and one-fifth in women are primarily situated in the stomach. It is probably correct to say that even after making all allowances for the increased expectancy of life and for more accurate diagnosis, cancer in general, and cancer of the stomach in particular, is becoming yearly more frequent.

(2) *Age Incidence.* Cancer of the stomach occurs most commonly between the ages of 40 and 70, but more especially between 55 and 65. Fully 80 per cent of cases occur within this ten-year period. Cases have been recorded as occurring in adolescence, but only 10 per cent are met with before the age of 40. The disease is very rare after 70.

(3) *Sex Incidence.* The ratio of males to females is about 3 to 1.

(4) *Heredity.* Gatewood found a positive family history in 11 per cent of his cases; Balfour in 16 per cent. In 100 consecutive cases I have found a definite family history in 26, and it would appear that, in certain families at any rate, there is an hereditary tendency to the disease.

(5) *Chronic Gastritis.* Konjetzny considers this to be an important predisposing factor, and he is supported in his view by Hurst. As some degree of chronic gastritis is found in all gastrectomy specimens this view is difficult to disprove. It is possible, nevertheless, that the gastritis may be a secondary manifestation resulting from the presence of the growth.

(6) *Chronic Gastric Ulcer.* In a certain proportion of cases cancer may arise in a chronic gastric ulcer. This subject is discussed on page 561.

The position of an ulcer in the stomach is of the greatest significance as regards its possible malignancy. Thus a chronic ulcer situated on or within half an inch of the *greater curvature*, even when it possesses all the radiological appearances of a benign ulcer, should be regarded and treated as malignant. Ulcers occurring at the inlet or outlet of the stomach are often malignant, whilst those situated in the pyloric canal should be regarded with suspicion, as well over 30 per cent of



these are primarily malignant growths. The *large*, indolent, penetrating ulcer, occurring on the posterior wall, half an inch or more from the lesser curvature, should in most instances be treated by partial gastrectomy, as fully 20 per cent of these will show malignant changes when submitted to microscopical investigation.

(7) *Benign Growths of the Stomach.* All benign growths of the stomach are liable to undergo malignant degeneration but this applies particularly to adenomata (see page 521).

(8) *Other Factors.* Constant and intermittent trauma or irritation of the mucous membrane, as may be produced by unsuitable articles of food, very hot or iced drinks, over-smoking, excessive consumption of alcohol, certain proprietary medicines, constant nervous strain, a lack of vitamins, severe chronic toxæmias, the effect of the ravages of acute specific or infectious diseases, or a congenital absence of free hydrochloric acid in the gastric juice have at times been thought to predispose to cancer of the stomach. In many cases, however, as in growths situated elsewhere, the exciting factors are completely obscure.

#### PATHOLOGY

The naked-eye appearances of the disease may show wide variations, but there are three recognised types :

- (1) *The malignant ulcer.*
- (2) *The fungating, polypoid, or cauliflower tumour.*
- (3) *The leather-bottle stomach or linitis plastica.*

The malignant ulcer or polypoid growth may undergo colloid degeneration, and when this occurs to a marked degree a fourth type is sometimes described—*colloid cancer of the stomach*.

A growth situated at the lower end of the œsophagus may also invade the stomach and project into its lumen.

*In a general way growths of the pylorus and cardia are fibrous, whilst those of the body of the stomach are soft, fungating, and luxuriant.*

*Malignant Ulcer.* This sessile or ulcerated form of cancer of the stomach is the most malignant and is the commonest type seen. Malignant ulcers most frequently occur in the pyloric vestibule or in the region of the lesser curvature, although no portion of the stomach is

immune (fig. 202). They infiltrate widely and soon give rise to metastatic deposits in the lymph glands and liver. They may be symptomless for a considerable period when situated on the lesser curvature or in any portion of the body of the stomach. When they occur in the pyloric canal, however, they produce obstructive symptoms early (fig. 203), and this region of the stomach soon becomes converted into a hard rigid tumour. The growth is scirrhus in nature and is composed of *spheroidal or columnar cells in an abundant matrix of connective tissue*. The ulcer is usually oval or circular in shape, has firm, raised, rampart-like or rolled-over edges, and a shallow crater, the floor of

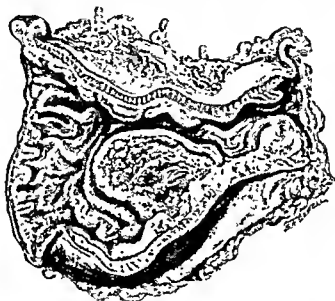


Fig. 202.—MALIGNANT ULCER OF THE PYLORIC PORTION OF THE STOMACH. ONLY THE PYLORIC PORTION OF THIS GASTRECTOMY SPECIMEN IS SHOWN (Author's case.)

which is often superficially ulcerated (fig. 204). At times the punched-out appearance of a chronic gastric ulcer may be closely mimicked, and the gastric ulcer which becomes malignant is of this variety—the sessile type.

When a section is made through the ulcerated or sessile type of cancer of the stomach and the cut surface is examined, it will often be possible to trace the muscular layers across the base of the ulcer, even in those cases where the muscle and serosa have been extensively invaded by growth. On the other hand, a section through a gastric ulcer which is undergoing malignant change will show a complete breach or obliteration of the muscular layers, the floor of the crater being composed of dense white fibrous tissue which forms an effective and insuperable barrier against invasion by cancer cells. The growth spreads rapidly in the submucosa, away from the pylorus

towards the cardia, and outlying islets can often be detected at least half an inch in advance of the growing edge.

When the growth extends to the peritoneal coat of the stomach the overlying surface of the organ may become studded with minute opaque white seedlings, wrinkled or puckered, greatly thickened and opalescent from gelatinous degeneration, or converted into a disc-like plaque of metallic hardness, from the vicinity of which may arise rigid filamentous strands or knotted cords of permeated lymphatic vessels.

*Fungating and Polypoid Tumours.* These form bulky, soft, friable, cauliflower-like masses which project into the lumen of the stomach

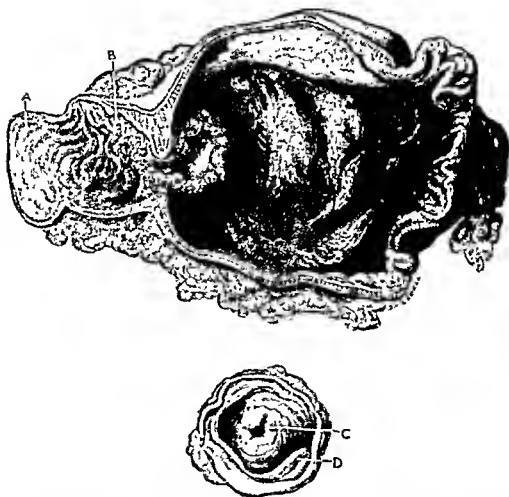


Fig. 203.—Malignant Ulcer of the Pylorus invading the first portion of the Duodenum at B. The Pyloric Orifice is markedly stenosed and the stomach dilated, the rugae being smoothed out except towards the Fundus. Microscopic Examination shows a Carcinomatous Invasiveness of the Tissue at the Margins of the Ulcer with very slight Extension into the Park. The Tumor consists of Low Columnar Cells, surrounding Irregular Spaces of Considerable Size. A and B—Duodenum. C—Growth invading Duodenum. D—Pyloric Outlet.  
(From a specimen removed by the Author at operation, and now preserved in the Museum of the R.C.S.)

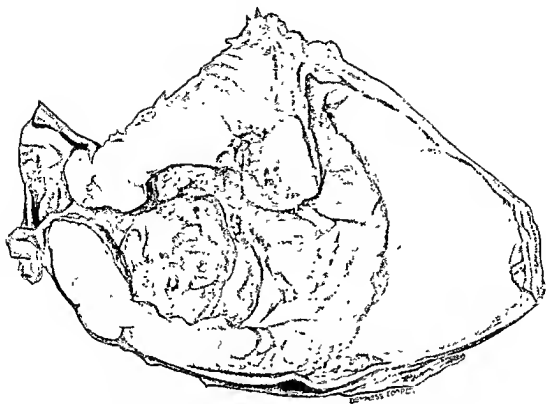


Fig. 204.—AN ULCERATED CARCINOMA OF THE STOMACH AT AN ADVANCED STAGE.  
(Museum, St. Bartholomew's Hospital.)

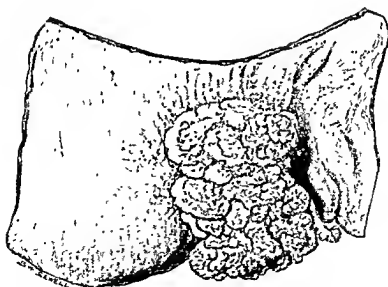


Fig. 205.—CAULIFLOWER LIKE GROWTH OF THE STOMACH.  
(Museum, R.C.S.)

(fig. 205). They usually arise in the body of the stomach in the region of the greater curvature, posterior wall, or fundus, have a comparatively narrow base, and infiltrate only a small area of stomach wall. They give rise to few symptoms during their early stages of growth, but may, by their bulk, plug the outlet of the stomach, thus causing pyloric obstruction.

The fungating tumours are adeno-carcinomata and are composed of columnar epithelial cells. The regional lymph glands are affected late in the course of the disease, and infiltration is confined to a limited area of the stomach wall. Some of these cancers probably originate in innocent tumours—polypi.

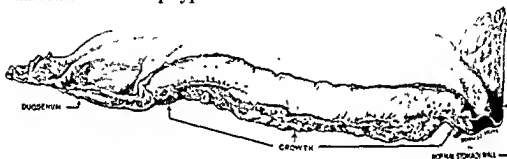


Fig. 206.—LOCALISED FORM OF LEATHER-BOTTLE STOMACH. SIDE VIEW OF SECTION MADE THROUGH THE GREATEST CURVATURE OF THE STOMACH. DRAWN FROM A SPECIMEN REMOVED BY TOTAL GASTRECTOMY. THE PATIENT DIED OF A GRAVE FORM OF ANEMIA 10 MONTHS AFTER THE OPERATION, HAVING ENJOYED GOOD HEALTH FOR THE FIRST 6 MONTHS. (Author's coll.)

*Leather-bottle Stomach or Linitis Plastica.* These terms are interchangeable, but should never be applied to the analogous condition known as fibromatosis of the stomach. Leather-bottle stomach is due to an infiltrating carcinoma, and two types are described :

- (1) Local.
- (2) Diffuse.

The *local* form of the disease starts at the pylorus, spreads very slowly in the direction of the cardia, and is associated with a great deal of fibrosis (fig. 206). The pyloric canal may, in certain cases, become constricted by the enormous overgrowth of fibrous tissue which occurs in the submucous coat, and by the accompanying hypertrophy of the overlying mucous membrane, resulting in marked dilatation of the stomach. Whilst such a condition may occur, it is more usual to find the uninvaded portion of the viscus collapsed and the pylorus patent.

On examination the pyloric portion of the stomach is found to be pearly white in colour, thickened and rigid, and densely hard and inelastic. Section of the stomach wall shows that the mucous membrane is cedematous and thrown into prominent folds, that all the coats of the

stomach are enlarged, and that the muscular layer is hypertrophied, pale, and fragmented. It will be seen that the thickening affects chiefly the submucosa, which is replaced by a hard mass of white fibrous tissue. In this tissue lurk a few sparsely scattered clumps of malignant spheroidal epithelial cells, which prove very difficult to detect on microscopical examination of serial sections of the growth.

The following conditions may be mistaken for, and may, in fact, be indistinguishable from, the localised form of leather-bottle stomach:

- (1) Hypertrophic tuberculous disease of the pylorus.
- (2) Syphilitic gummatous infiltration of the pylorus.
- (3) The so-called localised form of fibromatosis.
- (4) Scirrhus cancer of the pylorus.
- (5) Chronic gastric ulcer situated in the pyloric portion of the stomach and associated with extensive scarring of the submucous coat.

As it is often impossible to make any distinction between localised leather-bottle stomach and one or any of the above diseases when encountered at exploratory laparotomy, it is a safe rule to perform partial gastrectomy in all suspicious cases on the assumption that the leathery pyloric tumour is malignant.

The *diffuse* form of leather-bottle stomach was first described by Brinton in 1854, and is a comparatively rare type of cancer. He considered that the name *linitis plastica* was apt and very descriptive of the thick-walled, contracted organ. The disease starts at or near the pyloric orifice, infiltrating the submucosa (principally) and subserosa, and grows slowly around the circumference and along the axis of the stomach towards the cardia. The growth usually ends abruptly at the pylorus and rarely spreads into the walls of the duodenum. In an advanced case the cardia becomes involved and the infiltration extends upwards into the lower reaches of the œsophagus. The stomach eventually becomes steer-horn in shape, and is shortened and contracted by several inches (fig. 207). It is transformed into a densely hard, leathery, rigid tube, incapable of being distended, so that its capacity is reduced to as little as four ounces. The mucous membrane is swollen, congested, and markedly rugose, these hypertrophied rugæ appearing to be cemented to the underlying submucous coat. A few shallow ulcers, irregular in shape, may be present. The pyloric and cardiac orifices become fixed and patulous, and all sphincteric control is lost. The serosal aspect of the stomach is usually greyish in colour, although occasionally it may present a normal appearance.

A section of the stomach wall shows that the thickening consists mainly of white fibrous tissue and involves chiefly the submucosa and subserosa. The walls of the stomach may be as much as one inch thick. The segmentation or fragmentation of the muscle layer is clearly apparent, being produced by fibrous septa which extend outwards from the submucosa to the subserosa and divide up the hypertrophied circular muscular coat into little segmented bundles (see fig. 207). A specimen of linitis plastica may sometimes present the appearance given by a normal stomach which has been fixed in a strong formalin solution for a considerable period.

Metastases occur in the adjacent lymph nodes late in the course of the disease, and as this type of cancer is slow-growing, and the

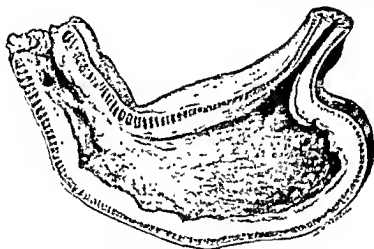


Fig. 207—LEATHER-BOTTLE STOMACH OR LINITIS PLASTICA.  
(Museum, R.C.S.)

growth is confined to the walls of the stomach itself for a long time, it might be thought that such a radical procedure as total gastrectomy would yield a high percentage of cures. Unfortunately, however, this is not so, for although this operation has now been performed for a number of such cases the late results have been most disappointing, the majority of patients being dead within one year of operation.

The diffuse form of leather-bottle stomach will have to be distinguished from :

- (1) Diffuse syphilitic infiltration of the stomach (see page 210).
- (2) Diffuse hyperplastic tuberculous disease of the stomach (see page 211).
- (3) The so-called diffuse form of fibromatosis of the stomach.

In *fibromatosis of the stomach*, which is claimed to be a pathological entity, an extreme fibrous thickening of the submucosa occurs, often in association with a chronic gastric ulcer. The disease is said to be localised when it affects the pylorus only, and generalised when the whole stomach becomes involved.

The *generalised* variety is indistinguishable in its naked-eye features from malignant diffuse leather-bottle stomach, the characteristics being the same except that no cancer cells are found on microscopy. On examination of a specimen there is the same leathery hardness, the same diffuse formation of new fibrous tissue in the submucosa (which may be half an inch thick), the same hypertrophy and fragmentation of the muscular coats, and, on section, the same abnormal distinctness of the individual coats of the stomach as occurs in the case of malignant linitis plastica.

The *localised* form, which is often associated with a deeply excavating chronic gastric ulcer, or even multiple chronic ulcers, is again similar in appearance to the localised form of leather-bottle stomach. When a section is made through the margin of the ulcer (when present), or through a portion of the involved wall of the stomach, the outstanding feature—fibrosis—is very evident, and malignant cells are nowhere to be found, even on numerous serial sections. The clinical features are indistinguishable from those of chronic peptic ulcer. The condition is probably either syphilitic or carcinomatous in origin.

*Modes of Extension of Gastric Carcinoma.* The five principal routes of spread are :

- (1) In the stomach wall.
- (2) To neighbouring lymph glands via the lymphatic vessels.
- (3) To adjacent organs or via adhesions to neighbouring viscera or the abdominal parietes.
- (4) To distant organs via the blood stream.
- (5) By the peritoneal cavity.

*In the Stomach Wall.* The main line of spread is upwards along the lesser curvature towards the cardiac orifice. A growth in the pyloric portion of the stomach rarely transgresses into the duodenal lumen, although figure 203, depicting a specimen I removed at operation, shows that the invasion of the duodenum was fairly extensive in this particular case. The growth spreads mainly in the submucous coat, and in the infiltrating types of cancer of the stomach such spread is rapid and very diffuse. The infiltration in this coat is usually one inch or more in advance of the growing edge of the ulcer. The microscopical extension of the growth, therefore, may be extensive and some distance away from the visible and palpable edge (Verbrugghen).

In the ulcerated types, outlying nodules or numerous small crops may be seen scattered in irregular arrangement just beyond the edge of the tumour. The cancer involves the muscular coats lying deep to its site of origin and splits up the muscle into pale pink blocks.



This is well seen when a section is made through the growth. The segmented muscular bundles are separated by strands of white fibrous tissue and carcinoma which run at right angles to the serosa. Occasionally the septa are arranged longitudinally, when the alternating layers of pale pink muscle and white growth can be seen.

In the late cases the whole of the lesser curvature, and even the lower end of the œsophagus, may become involved in growth.

*To the Lymph Glands.* In about 80 per cent of cases of cancer of the stomach metastases occur in the lymph glands, either at an early or a late stage in the disease; *but in some 20 per cent of cases the lymph glands do not at any stage become involved in growth.*

The position and extent of the glandular involvement is in part dependent upon the size, position, and nature of the growth, but a large tumour may sometimes be present with little or no metastatic deposit in the glands, while a small sessile tumour may be associated with wide-spread implants in the lymph glands. Again, in diffuse leather-bottle stomach the lymph glands may not be involved, or only at a late stage in the disease.

In scirrhus growths of the pylorus the adjacent group of glands—the supra-, the retro-, and the infra-pyloric—and even the lower coronary glands situated on the lesser curvature, very rapidly become involved, making the prognosis of these growths poor in comparison with those situated in the body of the stomach. Again, when the carcinoma is situated high up on the lesser curvature the glands in this region—the upper coronary groups and para-cardial glands—are invaded at an early date; whilst in cancer of the middle third of the lesser curvature, although the lower and upper coronary groups may show early metastases, provided the tumour is mobile and the glands easily accessible, gastrectomy is followed by a high percentage of good results.

The lymph glandular involvement is least when the growth is situated on the anterior wall of the body of the stomach, on the greater curvature, or in the region of the fundus. The sessile ulcerating form metastasises rapidly and extensively, whereas the cauliflower-like growth and the leather-bottle stomach do not readily involve the regional lymph nodes, and when they do their spread is more limited and less rapid.

If the glands in the portal fissure become invaded with carcinoma they may press upon and occlude the bile passages, giving rise to jaundice. Growth occasionally spreads along the ligamentum teres to

the umbilicus, where it may form a hard tumour. Invasion of Virchow's sentinel gland in the posterior triangle of the neck on the left side, immediately above the clavicle, takes place via the thoracic duct in some 2-3 per cent of late cases, and in probably less than 1 per cent of such cases the glands in the left axilla also show evidences of metastatic deposits.

Enlarged glands found at operation for malignant disease of the stomach are not necessarily cancerous, and in a number of cases they are simply inflammatory. Inflammatory glands are often enlarged and disc-like, soft, elastic, pink in colour, and discrete; whereas malignant glands may or may not be enlarged, and are often irregular in shape, hard and "shotty," whitish and sometimes matted together. Permeated lymphatic vessels often stand out as small knotted white cords.

*To Adjacent Organs.* Growth in the stomach may involve neighbouring viscera by direct spread. The organs most frequently invaded are those which lie in close proximity to the stomach, such as the transverse colon, the pancreas, the liver, the gall-bladder, the duodenum, and the upper coils of jejunum. The spleen is but rarely affected. Spread to these viscera may also occur along adhesions or through the medium of the omenta. The great omentum, when extensively infiltrated with growth, sometimes forms a huge abdominal tumour which may confuse the diagnosis.

*To Distant Organs via the Blood Stream.* When malignant cells enter the blood stream, metastases occur in the liver, lungs, pleura and bones, under the skin as subcutaneous nodules, or in other parts of the body. Metastases in the liver form large, white, hard tumours, accompanied by enlargement of the organ, and later by jaundice and ascites. They may closely simulate multiple gummata of the liver.

*By the Peritoneal Cavity.* When cancer has reached the peritoneal surface of the stomach it generally means that the case is inoperable, as malignant cells are soon freely discharged into the general peritoneal cavity and give rise to carcinomatosis (a condition which superficially resembles tuberculous peritonitis with ascites) and tumours in the pelvis. The pelvic peritoneum may become studded with growth, or large masses may form here owing to cells gravitating downwards. It is these large deposits in the pouch of Douglas which can be felt on rectal examination in cases of inoperable cancer of the stomach.

The stomach is one of the commonest organs to give rise to a general dissemination of growth over the peritoneum.

Ovarian metastases occur in 2-3 per cent of cases. These ovarian tumours may be mistaken for primary growths of the ovary and may, on microscopical examination, closely resemble a Krukenberg tumour (fibro-sarcoma muco-cellulare). In every case of bilateral malignant disease of the ovaries, therefore, the stomach should, at exploratory laparotomy, be carefully examined for any evidence of primary growth in this organ.

### DIAGNOSIS

In certain cases the clinical evidence of growth in the stomach and of the inoperability of the case is so irrefutable that ancillary methods of investigation are unnecessary. In the majority of cases, however, our methods of inquiry are conducted in the following order:

- (1) History of the case.
- (2) Physical examination of the patient.
- (3) X-ray examination of the stomach after the administration of a barium meal.
- (4) Laboratory examinations.
  - (a) Test meal.
  - (b) Occult blood test.
  - (c) Blood examination.
  - (d) Wassermann reaction.
- (5) Exploratory laparotomy.

A complete and detailed history and a painstaking physical examination—clinical methods—still remain of supreme importance in the diagnosis of this disease. Apart from positive radiological and laboratory findings, clinical evidence alone of the possible presence of cancer of the stomach is amply sufficient to warrant exploration of the abdomen.

As previously noted, there is a *short* history in 70 per cent of cases of cancer of the stomach, and a *long* history in 30 per cent.

It is significant that some 95 per cent of cases which give a long history have had intermittent dyspeptic symptoms suggestive of or even indistinguishable from those of chronic peptic ulcer, and that about half of these cases have received medical treatment for supposed gastric ulcer for periods often extending over many years. Thus



Fig. 203.—CANCER OF THE STOMACH. "MENISCUS" TYPE OF MALIGNANT ULCER AT THE PYLORIC END OF THE STOMACH. THERE IS CONSIDERABLE SPASM, BUT BARIUM IS RETAINED IN THE ULCER CRATER WHICH IS IRREGULAR IN OUTLINE. (H. Cecil Ball)



Fig. 202.—CANCER OF THE PYLORIC HALF OF THE STOMACH SHOWING THE "FINGER-PRINT" TYPE OF FILLING-DEFECT. (H. Cecil Ball)

clinical evidence would suggest that some 12-14 per cent of chronic gastric ulcers sooner or later develop into cancer.

We describe elsewhere how the symptoms in cases of chronic peptic ulcer show a complete and recognisable change of character with the onset of malignant degeneration. (See page 436.) The periodicity of the attacks and of the pain is altered. The pain, although varying in intensity, becomes continuous, and there are now no remissions and no respite.

Patients giving a *short* history may be grouped into two categories: those *with* obstructive symptoms in whom a correct early diagnosis is often possible, and those *without* obstructive symptoms in whom a diagnosis is so often delayed. The severity of the symptoms is largely dependent upon the degree of obstruction present, and therefore more upon the part of the stomach involved than upon the actual size of the carcinoma. Large palpable growths of the body of the stomach may be, and often are, associated with few symptoms, whilst as a rule small early growths of the pylorus soon proclaim their presence by the severity of the disturbance caused by the narrowing or occlusion of the outlet of the stomach. But in a general way the first manifestation of malignancy of the stomach is slight disturbance of function.

All the objective signs of cancer of the stomach—emaciation, anaemia, tumour mass, etc.—appear at a late stage in the disease.

The presence or absence of free hydrochloric acid is dependent upon the amount of gastritis and the extent of stomach wall involved by growth. Some degree of gastritis is always present. Whilst achlorhydria almost invariably characterises advanced cases, free hydrochloric acid is frequently found in early cases.

There is probably less than 2-3 per cent of error in X-ray diagnosis in cases which show some evidence of the presence of cancer, and the more advanced the case the less likelihood there is of error. X-rays are now capable of detecting comparatively early cases by the demonstration of:

- (1) Irregularity in the contour of some portion of the stomach.
- (2) Loss of peristaltic waves over a localised area of the involved stomach wall.
- (3) Filling defects. (Figs. 208 and 209.)

Most of the cases which are at first misdiagnosed are those which are seen in the early stages of the disease. The tumour may, for instance, be so small or located in such a position that, even on careful X-ray examination, it is invisible, and this particularly applies to growths

situated on the anterior wall of the stomach. Again, the growth may be solely confined to the mucous membrane, and, the muscular coat being thus uninvolved, the typical peristaltic waves may be seen to sweep unchecked across the malignant zone.

Early cases of leather-bottle stomach, growths of the greater curvature, and cancers of the fundus are elusive, and by presenting indeterminate and atypical appearances lead to misinterpretation and delay in diagnosis.

*Negative X-ray findings do not prove the absence of malignant growth, nor should a decision as to operability be based solely upon radiological findings.*

Have we no means of proving, apart from operation, whether a chronic ulcer of the stomach is simple or malignant?

There is the method of trial by medical treatment—the therapeutic test. This consists in putting the patient to bed to ensure complete rest, and in instituting a strict and careful regime of medical treatment such as that detailed by Hurst. The patient is X-rayed at the end of the first week, and again at the end of the second, third, and possibly the fourth, to ascertain the condition of the ulcer. Operation is deferred *sine die* if the lesion continues to diminish in size, if occult blood ceases to be present in the stools, and if all the symptoms disappear. When a symptomatic cure has been effected, and when there is no further X-ray evidence of ulcer, the patient is ordered Hurst's post-ulcer diet (see page 576) and discharged; but he remains a suspect for many weeks or months. Further X-ray examinations will be necessary six weeks or longer after his discharge to make quite sure that there is no return of the ulceration.

#### CLINICAL FEATURES

The *clinical features*—the symptoms, the signs, and the course of the disease—are mainly dependent upon the situation, the type, the extent, and the rigidity of the growth. Cancers situated at the inlet or outlet of the stomach declare themselves early in their course by producing obstructive symptoms and a great deal of disturbance; whereas growths occurring in the body of the stomach, and more especially in the region of the greater curvature, may be clinically silent and symptomless to the end, or be marked only by vague symptoms until a late stage in the disease has been reached.

A large fungating polypoid tumour arising from the greater curvature may grow luxuriantly in the stomach for a long time without giving warning of its presence until it declares itself with dramatic

suddenness by bleeding profusely or by occluding the pylorus with its fleshy bulk.

There is also a lethal type of cancer of the stomach which may masquerade as a chronic peptic ulcer for many months.

The facts that one type of lesion will often produce different signs and symptoms in different persons, and that various dissimilar lesions can cause similar symptoms, add greatly to the difficulties encountered in diagnosis. There are no known pathognomonic symptoms of early cancer of the body of the stomach, and the so-called classical signs and symptoms are usually those of the inoperable stage. There is not one symptom which may be said to be *always* present or *always* absent.

Cancers situated at the pyloric or cardiac orifice can be diagnosed early; those of the body of the stomach often pass undetected until too late. Even to-day ulcer-cancer is too frequently treated medically as "chronic indigestion" or "gastric ulcer" until the condition is past all hope of radical cure.

Whilst it is agreed that certain cancers of the stomach may be associated with anomalous, vague, and bizarre clinical phenomena, it is nevertheless possible to recognise *three common clinical types*:

- (1) The insidious type.
- (2) The obstructive type.
- (3) The peptic ulcer type.

*The Insidious Type.* Cancers in this group cause much difficulty in diagnosis on account of the vagueness of the inaugural symptoms. This is chiefly due to the position of the growth. Growths situated anywhere in the body of the stomach—on the lesser curvature, on the greater curvature, on the anterior or posterior wall, in the fundus; in fact, in any portion of the stomach except the pyloric vestibule, the pyloric canal, and in the immediate precincts of the cardiac orifice—are usually insidious in onset and may not give rise to any significant symptoms until the disease is already well established. The first manifestations of such growths may be hæmatemesis, melaena, or acute perforation.

The early symptoms in this type, when grouped together, are not very significant, and at the most they may only suggest that the patient is suffering from debility or from a mild form of indigestion. These *early symptoms* usually comprise:

- (1) Lassitude.
- (2) Loss of interest; lack of energy; mental apathy.

- (3) Dislike of food, especially meat.
- (4) Nausea.
- (5) A vague feeling of discomfort after food—a *mild* but persistent dyspepsia.
- (6) Flatulence.
- (7) A feeling of weight in the abdomen.
- (8) Heartburn, eructation.

The *late symptoms* may include :

- (1) Regurgitation.
- (2) Vomiting.
- (3) Constipation or diarrhoea.
- (4) Anorexia.
- (5) Loss of weight, emaciation. (It is not safe to assume that cancer of the stomach is absent because the patient's weight is stationary.)
- (6) Anæmia.
- (7) Constant epigastric pain not relieved by food or vomiting.

In the *early* cases nothing abnormal may be detected on *physical examination*. The patient may look well, and on palpation of the abdomen there may seem to be nothing amiss. Even a large growth may be impalpable when the stomach lies collapsed or hidden under cover of the liver or ribs. On the other hand, a growth may be discovered by chance during a routine abdominal examination, and even when a large malignant mass is found to be present in the stomach the patient may appear healthy and complain of nothing worse than a few mild dyspeptic symptoms.

Physical examination of *late* cases leaves little room for doubt. The patient is pale and anæmic, lemon-tinted or yellowish in colour; the eyes are sunken, the face drawn and lined, and the nose pinched; the muscles of the neck stand out prominently, the expression is apathetic, and the general mien is one of dejection. The most striking feature is emaciation. The unhealthy paleness may be so noticeable as even to suggest the possibility of pernicious anæmia. Whilst on abdominal examination nothing abnormal may be seen or felt, it is common to find the skin wrinkled and inelastic from loss of fatty tissue, the epigastric region retracted and shelving downwards from the costal margin, and occasionally slight distension and a dome-like appearance of the lower half of the abdomen. There may be visible peristalsis, the waves coursing across the epigastrium from left to right. The whole abdomen



may be distended and tense with ascitic fluid, and a malignant tumour in the stomach may form a visible localised swelling which, on examination, is found to be hard and craggy, irregular in outline, and mobile or fixed. The enlarged liver may be felt, studded with nodular umbilicated metastatic growths. Occasionally anchoring of the umbilicus may be evident.

*The Obstructive Type.* The symptoms in this type vary according to whether the growth is situated at the cardiac orifice or in the pyloric portion of the stomach. The main symptoms common to both types are those associated with *obstruction*.

(a) *The Cardiac Orifice Type.* Growths of the cardia are comparatively rare. The earliest symptom is loss of appetite. Loss of weight is excessively rapid as soon as the neoplasm encroaches upon the narrow inlet of the stomach; but even before this channel is strictured there is a steady weekly, or even daily, reduction in weight. Early in the course of the disease epigastric pain and pain referred to the back is complained of immediately after food is taken, and there may be a sensation of food remaining lodged in the stomach or in the lower portion of the gullet. At a later date pain comes on actually during meals and persists for an indefinite period, although it diminishes in intensity just before the next meal is taken.

The pain is not due solely to the passage of food over the ulcerated surface of the growth, as it is present between meals. It is probably due to some accompanying œsophagitis, with spasm and tension of the coats of the œsophagus, and also to interference with the neuromuscular mechanism of the cardiac sphincter owing to its infiltration with growth and inflammatory exudates.

Heartburn is a common symptom of this type.

Frequent regurgitation of frothy, stringy, salivary, decomposing fluid heralds the advent of complete obstruction. Whilst at first solids can be swallowed, as the disease advances and the stricture tightens this becomes an impossibility, either on account of the accompanying inflammatory œdema or through particles of food becoming impacted in the narrow channel. In the final stages, when occlusion is complete, even water may be returned into the mouth within a few seconds. Emaciation becomes increasingly pronounced, and the patient will starve to death unless a gastrostomy or jejunostomy is performed.

Owing to the similarity of all the signs and symptoms it is often

impossible clinically to distinguish a cancer of the cardia from a growth situated in the lower end of the œsophagus.

(b) *The Pyloric Type.* When the tumour mass is situated in the pyloric vestibule and has a long pedicle, it may give rise to a "ball-valve" obstruction. Tumours of this variety are less common than the ulcerated type of gastric cancer and are not so malignant, usually spreading only to a limited area near the site of their origin. They may grow to a great size and produce a large epigastric tumour. The prognosis following a wide resection in these cases is very good.

*Large palpable gastric tumours should not be considered inoperable solely on account of their size.* The surface of the growth eventually degenerates, ulcerates, bleeds, and becomes infected. When this occurs most of the classical but late manifestations of gastric cancer are found—cachexia, anæmia, toxæmia, etc.

When the pyloric portion of the stomach is the seat of cancer, the symptoms are those of pyloric stenosis, and it is often quite impossible to determine by the symptoms alone whether the obstruction is due to growth or to benign ulcer. A barium meal examination, however, is of the greatest help in making a differential diagnosis between a malignant and a benign obstruction. In malignant stenosis of the pylorus the following features will often be noted :

(i) *A short history.* Pyloric stenosis occurring in a patient past middle life with a short history of digestive upset is very significant of growth, but it should be remembered that a sub-acute gastric ulcer situated in the pyloric canal may likewise rapidly give rise to obstructive symptoms. All ulcers situated in the pyloric canal, or in fact anywhere in this region, should be most carefully screened to determine as far as possible the nature of the lesion, and if doubt exists, or if, after a brief course of medical treatment, there is no appreciable improvement, operation should be advised. Fully one-third of the ulcers situated in this portion of the stomach are primarily malignant.

(ii) *Pain.* Pain, although varying in intensity, is never absent, and becomes more severe after food and towards evening.

(iii) *Vomiting.* At first small quantities are vomited at frequent intervals, but later on, as stenosis becomes more marked, larger quantities of decomposing fluid are voided at longer intervals. Vomiting may afford some *slight* relief from the pain.

(iv) *Nausea.* This is a constant feature.

(v) *Anorexia*. This symptom becomes more marked as the disease progresses.

(vi) *Loss of weight* is gradual.

(vii) *Constipation*. This is the rule in all cases of pyloric stenosis, whether due to benign ulcer or to malignant stricture.

On examination there may be visible peristalsis, splashing sounds may be elicited, and a hard tumour may be felt in the epigastrium.

*The Peptic Ulcer Type*. Over 25 per cent of patients with cancer of the stomach give a history of "gastric ulcer" which has existed for many years prior to the discovery of the present trouble, and many of these cases are primarily treated medically for chronic gastric ulcer.

"The point at issue, and it is a purely clinical point, is that some patients with a definite ulcer history, with a radiological diagnosis of ulcer, with laboratory findings typical of ulcer, with all the clinical and scientific evidence in favour of ulcer, prove the utter unreliability and worthlessness of that evidence by dying of cancer. When or where or how the malignancy originated may interest the pathologist, but it is not of the smallest interest to the clinician who made the mistake and to the patient who paid with his life for the error." (Mace, Boyce, and McFetridge. *Ann. Surg.*, p. 619, Oct. 1933.)

The differentiation between a gastric ulcer and the ulcerated form of cancer of the stomach is impossible in certain cases, short of the method of direct inquiry—exploratory laparotomy. Even at operation it is never easy nor always possible to make such a differentiation. One of the factors concerned in the death-rate of gastric cancer is persistence with medical treatment for *supposed* gastric ulcer, particularly when, as so often happens, this treatment has afforded temporary relief. It is a safe rule to regard as malignant all chronic ulcers of the stomach with a diameter of one inch or more.

In a patient known to be suffering from a chronic gastric ulcer of some duration, the following symptoms would indicate, or at least suggest, the possibility that the ulcer has become malignant :

(i) *Loss of the periodicity of the attacks and of the symptoms*. With the onset of cancer there is a loss of the periodicity of the attacks and there is also a loss of the regularity of the occurrence of the pain during the day. The present attack is different from the previous

ones and appears to drag on interminably. The interval, too, between the present attack and the last one is very much shorter than the intervals between the earlier attacks.

(ii) *Pain*. The pain is different in character. It is mild but continuous, and there is now no longer the interval of relief after meals. The sharp burning ulcer pain is replaced by a dull ache which is intensified by eating. Where, however, an ulcer has eroded the pancreas, whether it be benign or malignant, pain is always severe, continuous, and often referred to the back or left shoulder region. In the average case the severity of the pain is not such a significant feature of malignant degeneration as the continuity of the pain.

(iii) *Anorexia*. Anorexia usually increases when a cancer becomes grafted on to an innocent ulcer.

(iv) *Nausea*. This is generally a common feature of malignant growths of the stomach.

(v) *Vomiting*. Whereas vomiting has previously given relief, with the onset of cancer this may no longer be the case. The vomiting of "coffee grounds" is often seen with large fungating or necrotic growths.

(vi) *Loss of weight*. At first this is slight, but it increases as the disease advances.

Periodicity of attacks and of symptoms characterises peptic ulcer. With the onset of malignant degeneration this periodicity is lost and the pain, although varying in severity, is constant and unremitting.

#### ANALYSIS OF INDIVIDUAL SYMPTOMS IN CASES OF CANCER OF THE STOMACH

(1) *Pain*. Abdominal pain is the first symptom in over 50 per cent of these cases, is present to some degree in 80 per cent, and absent in 20 per cent. The pain, when mild, amounts to nothing more than a sense of discomfort, uneasiness, upset, fullness, or of weight in the epigastrium. Although distress is a common symptom, severe pain is rare and is usually the result of extra-gastric involvement from penetration or perforation of the growth. With growths of the body of the stomach it is usual for the patient to complain of mild or moderate epigastric discomfort after meals which, as the disease progresses, develops into a continuous dull aching pain, worse after taking solid food, and only partially eased by vomiting, anodyne drugs, or gastric lavage. Vomiting, as a rule, does not completely relieve the pain in a

malignant stomach, although it gives some relief with early obstructive growths.

As pointed out above, in cases of ulcer-cancer there is no abatement of pain before the next meal. The severe pain may disappear, but it is immediately replaced by a different kind of pain—incessant discomfort. This discomfort is a characteristic feature of cancer of the stomach, whilst with chronic peptic ulcer there are intervals of freedom, and the periodicity of the attacks and the periodicity of the pain will often proclaim the benignity of the condition.

A sensation of fulness after eating is one of the most common of the early manifestations of cancer of the stomach; inability to take small quantities of food or drink generally denotes that extensive involvement of the walls of the stomach has already occurred. (Balfour, *Canad. Med. Assoc. Jl.*, Vol. 32, No. 3, p. 245, March, 1935.)

(2) *Loss of Weight.* In gastric cancer weight may be maintained in spite of the ravages of the disease. In certain cases, and particularly in those treated medically as chronic gastric ulcer, there may even be a temporary gain in weight; but the majority lose weight rapidly and steadily, especially the obstructive type, this loss of weight being accompanied by weakness, weariness, and later by all that is covered in the term cachexia.

Loss of weight is due to :

- (a) Partial or complete gastric stasis.
- (b) Chronic gastritis.
- (c) Anorexia.
- (d) Chronic toxæmia.
- (e) Vomiting.

(3) *Emaciation.* This is present to some degree in over 75 per cent of cases, and is associated with marked muscular weakness and undue fatigue. Lassitude, followed by fatigue on slight exertion, may be complained of before there is any local evidence of growth in the stomach, and symptoms of debility accompanied by vague gastric upset, occurring suddenly without any apparent cause in a patient over the age of 40, are suspicious of early growth in the stomach. Cachexia implies that there has been delay in diagnosis, and when this has reached an advanced stage it often denotes inoperability.

(4) *Nausea.* Nausea may be the first symptom of cancer of the stomach, and is present to some degree in nearly all cases.

(5) *Vomiting*. With a non-obstructive growth in the body of the stomach vomiting may be absent. When it occurs in such cases it is usually a *late* feature, whilst with obstructive growths of the cardia or pylorus it is rather an *early* manifestation. Vomiting gives little or no relief from pain in cases of cancer of the stomach, the maximum amount of relief being afforded by gastric lavage. One of the earliest indications of the onset of malignant change in a chronic gastric ulcer may be a cessation of the relief previously afforded by vomiting. Vomiting is a most noticeable and characteristic feature of pyloric obstruction due to an infiltrating scirrhus growth, the attacks taking place at longer intervals, usually towards the end of the day when large quantities of foul-smelling fluid containing particles of undigested food are brought up at each vomit. The vomit is often dark in colour from the presence of altered blood—"coffee-grounds"—evil-smelling, and contains fragments of food taken hours or even days previously. This type of vomiting and of vomited material occurs only in pyloric obstruction.

(6) *Anorexia*. There is generally some degree of loss of appetite. At first there is merely a disinclination for food; the patient does not feel hungry and nothing seems to tempt him to eat. At a later stage there is a definite dislike of or distaste for food, especially meat, until finally there may even be an abhorrence of the very sight or thought of food.

(7) *Anæmia*. Anæmia is of the secondary type, and is due to hæmorrhage as well as to the absorption of toxic and inflammatory products from the growth. Some degree of bleeding is almost invariable, and in fully 95 per cent of cases occult blood is found on repeated examinations of the feces and gastric contents, whilst frank hæmatemesis or melæna is estimated to occur in 15 per cent.

Any unexplained anæmia, with or without digestive symptoms and wasting, in a patient past middle age should immediately arouse suspicion of cancer of the stomach, and calls for a complete examination of the blood and an X-ray investigation. The anæmia is evidenced by pallor, breathlessness, and tachycardia. As in pernicious anæmia the skin may be deathly white or of the palest yellow tint. Ascites and œdema, due rather to severe anæmia than to metastases in the liver, may be present in a limited number of cases of gastric cancer in which at operation the growth proves to be resectable.

(8) *Condition of the Bowels.* Constipation is the rule with the obstructive types of cancer, whilst diarrhœa frequently accompanies growths of the body of the stomach.

(9) *Dysphagia.* This is an early symptom with growths of the cardia, and whilst it may be absent for a considerable period with growths situated elsewhere in the stomach, it is usually present when the disease is advanced. It may be an early feature of a malignant hour-glass stricture situated fairly high up in the stomach.

(10) *Tumour Mass.* Some 5 per cent of patients with cancer of the stomach notice a tumour in the abdomen before seeking medical advice, and a palpable tumour is computed to be present in 50 per cent of cases when examined by the surgeon for the first time.

(11) *Jaundice.* This is a late and ominous sign, and is due to secondary deposits in the liver or to enlarged malignant glands in the portal fissure compressing the bile-duct. Ascites is usually found in these late jaundiced cases.

(12) *Pyrexia.* A slight degree of pyrexia is common in most cases, and is rarely absent when secondaries are found in the liver. It may also be evidence of a superadded infective element in the neighbourhood of the growth.

(13) *Peripheral Venous Thrombosis.* "An apparently unexplained phlebitis in a patient over 50, especially if other thromboses occur elsewhere in the body, is sometimes evidence of cancer of the stomach." (Thomas C. Hunt.)

#### CLINICAL SIGNS OF INOPERABILITY

(1) Hard tumours of secondary growth are felt per rectum or per vaginam in the pelvic peritoneum.

(2) Virchow's gland in the neck is hard and enlarged.

(3) Nodules of growth are present at the umbilicus.

(4) Subcutaneous nodules of growth are scattered over the abdominal wall.

(5) Ascites is present. This may occur with metastases in the liver, peritoneal carcinomatosis, or obstruction of the portal vein.

(6) The liver is enlarged and **nodular**, or there is evidence of secondary growth in distant organs, e.g. lungs, long bones, spine, etc.

(7) There is X-ray evidence of involvement of an *extensive area* of the stomach by growth and *wide fixation* of the organ to neighbouring structures.

It is good practice to follow a rule that in cases of cancer of the stomach exploratory laparotomy should be undertaken unless recognisable, irremovable, metastatic deposits can be demonstrated. (Balfour.) The operability of a case cannot be judged merely by the size of the tumour present. A large number of cases in which a mass can be felt prove to be operable, and a freely movable tumour, whether large or small, is often more likely to be resectable than one which cannot be palpated.

*Provided that no metastases are found on examination, the patient should always be given the benefit of the doubt, even where the disease appears to be advanced.*

With large, freely movable, palpable growths, where resection is often possible, the late results are usually good. The massive proliferative growths of the body of the stomach which so frequently produce large epigastric tumours metastasise late and sparingly, are locally malignant for long periods, and often lend themselves to partial gastrectomy.

#### DIFFERENTIAL DIAGNOSIS

Cancer of the stomach must be distinguished from :

- (1) Chronic gastritis.
- (2) Chronic gastric ulcer.
- (3) Innocent new growths of the stomach.
- (4) Pernicious anæmia.
- (5) The so-called nervous dyspepsias, e.g. anorexia nervosa.
- (6) Any condition which causes debility, loss of weight, anæmia, or chronic dyspepsia.

#### CAUSES OF DELAY IN DIAGNOSIS

Why is there such a heavy death-rate from cancer of the stomach ? Why is it that such a large number of cases are inoperable when seen for the first time, and in only some 30 per cent can any attempt at cure be made ? Wherein lies the blame for such a lamentable state of affairs ? What are the causes of delay in diagnosis ?



It may be the fault of :

- (1) The patient.
- (2) The medical attendant.
- (3) The radiologist.
- (4) The nature of the disease.
- (5) Undue persistence with medical treatment.

The *patient* is to blame when he is aware that he is suffering from a serious gastric malady, but clings to the hope that with the aid of self-prescribed anti-dyspeptic remedies the condition will eventually right itself; when he is conscious of the persistence of abdominal symptoms, but dreads to seek advice, fearing to be told that he is suffering from cancer and that an operation is necessary; when, ill as he is, he fails to attend to his health for business or other reasons; when he considers that the present "gastric ulcer" attack, although more stubborn and differing in many respects from previous attacks, will, by the old, tried methods of dieting and alkaline powders, eventually subside as previous attacks have done; when he can feel a tumour in his abdomen but keeps this sinister fact to himself; whereas, had he sought medical advice at an early stage in his disease, radical operation and hopes of cure might have been possible.

No blame for delay can be attributed to him, however, when, although harbouring a cancer in his stomach, he feels well and continues in his usual health until smitten suddenly and unexpectedly by a copious hæmorrhage or acute perforation, or when, in the absence of gastric symptoms, or in the presence of only slight discomfort, a sense of weight or uneasiness in the abdomen, or the mildest symptoms of indigestion, he immediately reports to his medical attendant, only to discover that, unknown to him, there is a cancer in his stomach which has already reached an inoperable stage.

The *medical attendant* is responsible when he does not fully investigate at the earliest possible moment every case of chronic dyspepsia which comes to him for treatment; when he fails to bear in mind that there is a potential cancer in the stomach of every patient, especially after the age of 40, who, for the first time in his life, complains of indigestion; when he treats cases of large chronic gastric ulcers by medical measures indefinitely without submitting them to periodic radiological examinations and tests for occult blood in the stools; and when he overlooks the possibility of gastric cancer in all anomalous chronic abdominal conditions.

The *radiologist* is occasionally at fault for delay in diagnosis when

he does not, in spite of his penetrating means of vision, detect, or at least suspect, a growth of the stomach when it is present, or when he mistakenly suggests that the niche of a malignant ulcer is that of a simple gastric ulcer. It is, however, to be admitted that it is only on rare occasions that a competent radiologist will make a mistake in diagnosis in the presence of this disease, especially if he frequently re-examines all cases which are radiologically or clinically suspicious.

We have already described how the *nature of the disease* may, in itself, be a potent factor in causing delay in diagnosis. Certain growths in the stomach are symptomless, insidious in onset, or, by being situated in a capacious portion of the stomach, give rise to few symptoms until the disease is advanced.

### CURABILITY

Cancer of the stomach can only be cured by operation. What percentage of the cases who have sought surgical aid are alive and well after ten years? Possibly not more than 10 per cent; but this depends upon several factors, such as the ability of the surgeon, the position, the extent and the nature of the growth, and the age and general physique of the patient.

A study of literature shows that there is no uniformity of opinion with regard to operability-rate, resection-rate, and final results. But here is a brief résumé of the probably unsurpassed results given by three leading surgeons.

Balfour (*Coll. P. Mayo Clin.*, p. 80, 1931) states that resection was possible in 43.16 per cent of his cases in which exploration was undertaken, and that this represents 22.62 per cent of the patients who were seen with gastric cancer. He considers that the present situation is that in about 50 per cent of cases of cancer of the stomach exploration is warranted, and in about 50 per cent of these cases the growth can be removed. His figures thus prove that in less than 25 per cent of cases of cancer of the stomach can any attempt at cure by radical operation be made. He is also of the opinion that the mortality for partial gastrectomy should be less than 10 per cent. In the last 200 cases in which he performed partial gastrectomy or total gastrectomy for cancer of the stomach there were only 10 deaths—a mortality of 5 per cent. He reported a group of 278 patients who had lived five years or more following the removal of cancer of the stomach, stating that five-year cures could be obtained in 50 per cent of cases in which the lymphatic glands were not involved. No fewer than

128 patients with gastric cancer, operated upon between 1910 and 1926 at the Mayo Clinic, lived ten years or more, and this figure represents about 20 per cent of the total number of patients upon whom resection was performed.

Gatewood (*Surg., Gynec., Obstet.*, lvi, 2142, 1933) had a mortality of 18 per cent following partial gastrectomy, and of those who survived 46.1 per cent lived over three years and 36 per cent were alive at the end of five years. Of the operations performed by Gatewood for cancer of the stomach :

- 24 per cent were exploratory only ;
- 7 per cent were palliative for relief of starvation (gastrostomy or jejunostomy) ;
- 41 per cent were gastro-jejunostomies ; and
- 28 per cent were gastric resections.

The average length of life following exploration was 6.1 months ; after gastro-jejunostomy 8.17 months (2.65 months longer) ; whilst following resection the patients who left hospital have lived on an average four years and nine months.

Finsterer's figures (*Wien. Klin. Woch.*, Aug. 1929, p. 1125 ; *ibid.*, Sept., 1929, p. 1157) are interesting and may be grouped as follows :

- (1) Simple Resection—211 cases.
  - 6.1 per cent died.
  - 31 per cent lived over five years.
- (2) Complicated Resection—129 cases.
  - 41 per cent died.
  - 30.4 per cent lived over five years.

#### TREATMENT OF GASTRIC CANCER

- (1) Management of the Inoperable Case.
- (2) Pre-operative Treatment.
- (3) Operative Treatment.

- (a) Palliative Operations.
  - (i) Gastro-jejunostomy.
  - (ii) Exclusion of the growth.
  - (iii) Resection of the growth.
  - (iv) Gastrostomy.
  - (v) Jejunostomy.

- (b) Radical Operations.
  - (i) Partial gastrectomy.
  - (ii) Total gastrectomy.

(4) Post-operative Treatment.

*Management of the Inoperable Case.*

This chiefly concerns the relief of pain, and of obstruction if present. The pain may be due to pressure of the growth upon neighbouring structures, to obstruction, to gastritis, or to a combination of all three.

The following measures are advocated :

(1) *Diet.* In the late stages no solid food should be taken. Nutrition should be maintained by the administration of fluids and semi-solids by mouth, and these may include soup, junket, jelly, Ovaltine, citrated milk, alcohol, barley-water to which is added 10 per cent glucose, etc.

(2) *Medicines.* Aspirin and potassium bromide, luminal, tinct. chlorof. et morph. co., tinct. opii, nepenthe, or tinct. belladonna may prove useful in alleviating pain and vomiting. Where the drugs mentioned above prove ineffective, and the patient is unable to bear the intolerable and unremitting pain with which he is tormented, the inestimable benefits of lavish doses of morphia, omnopon, or heroin should not be denied.

(3) *Gastric Lavage.* This is urgently indicated in cases of pyloric stenosis to rid the stomach of its decomposing stagnant fluids and mucus, to relieve pain, and to improve the general condition of the patient. It is also a very useful measure in relieving pain, even when no obstruction is present.

A small stomach tube should be passed daily and the gastric contents aspirated. The stomach is then gently irrigated with normal saline, hydrogen peroxide (one drachm to the pint), or with a weak solution of hydrochloric acid. The irrigation should be continued until the contents are returned clear.

*Pre-operative Treatment.*

(1) *Intravenous Salines.* These are introduced into the circulation to combat the dehydration which is so often present, and to stimulate hepatic and renal function.

(2) *Blood-Transfusions.* These may be necessary where the patient is anæmic, especially if the hæmoglobin is below 60 per cent, or where he is in a very low state.

(3) *Gastric Lavage.* The contents of the stomach are aspirated, and lavage is performed with a weak solution of hydrogen peroxide, with normal saline, or with 0.25 per cent hydrochloric acid, once or twice a day, and always one hour before operation is undertaken.

(4) *Care of the Mouth.* The teeth are examined and receive such treatment as is necessary for the eradication of sepsis. Frequent mouth-washes are prescribed in all cases.

(5) *Medicines.* Prior to operation in all cases of cancer of the stomach I make a practice of prescribing an acid tonic mixture containing dilute hydrochloric acid. The hydrochloric acid does much to diminish the sepsis which is so often present with growths in the stomach, particularly when such growths are necrotic.

(6) *Rest in Bed.* It is customary to keep the patient in bed for a week or more prior to operation, not only to accustom him to his surroundings, but to improve his general condition and to complete any chemical investigations which may be required.

(7) *Glucose.* For two or three days before the operation a 5 per cent solution of glucose is given per rectum or by mouth until some fifteen pints have been absorbed.

(8) *Artificial Sunlight.* Artificial sunlight baths are given, often with very marked beneficial effects.

(9) *Skin Preparation.* The skin of the abdomen is prepared the day before operation. The abdominal wall is thoroughly cleansed with ether soap, shaved, and then painted lavishly with tannin-alcohol or tinct. metaphen (Abbott) over an area extending from the nipples to the thighs. One hour before the patient is taken to the operating theatre the skin is again painted, after which a sterile sheet is fastened around him.

(10) *Diet.* Until the evening before the operation the patient is kept on a light, nutritious, and assimilable diet.

(11) *Enema.* A purge is given the night before the operation, followed by an enema the next morning.

In cases where *gastrostomy* is indicated the pre-operative treatment is mainly concerned with supplying the patient with as much fluid as possible, introducing it into the circulation by the rectal or intra-venous route.

*Operative Treatment.*

(1) *Palliative Operations.*

- (a) *Gastrostomy.*
  - (i) Stamm.
  - (ii) Witzel.
  - (iii) Marwedel.
  - (iv) Kader.
  - (v) Lepage-Janeway.
- (b) *Jejunostomy.*
  - (i) Witzel.
  - (ii) Mayo-Robson.
  - (iii) Coffey.
- (c) *Gastro-Jejunostomy.*
- (d) *Exclusion of Growth.*
- (e) *Excision of Growth.*

(2) *Radical Operations.*

- (a) *Partial Gastrectomy.*
  - (i) *Anterior Polya Methods.*
    - 1. Moynihan.
    - 2. Balfour.
  - (ii) *Posterior Polya Methods.*
    - 1. Finsterer.
    - 2. Lahey's method of dealing with the proximal jejunal loop.
- (b) *Total Gastrectomy.*
  - (i) Method I—Moynihan.
  - (ii) Method II.

*Palliative Operations*

*Gastrostomy.*

The main indications for gastrostomy are as follows :

- (1) Diseases of the pharynx and larynx in which swallowing becomes impossible.
- (2) Cancer of the œsophagus (some cases).
- (3) Stricture of the œsophagus, as may result from corrosive poisoning, syphilitic ulceration, or possibly peptic ulceration.
- (4) Cancer of the cardiac end of the stomach.

The clinical features and the X-ray appearances of a growth of the lower end of the gullet or of the cardia are as a rule quite characteristic. Having established the diagnosis that a cancerous growth is obstructing the cardiac portion of the stomach, it must be decided which is the most favourable moment for the performance of a gastrostomy. It is generally agreed that operation should not be undertaken as long as the patient is still capable of swallowing solid food. It should, however, not be deferred until he is *in extremis* or obviously dying of starvation.

It must be admitted that this operation is one of despair; it is performed upon patients whose condition is extremely poor through emaciation, and who are doomed to die a few weeks or months later. The immediate mortality is high, and the amount of relief afforded is but transient at best.

The object of all gastrostomy operations is the same, namely, to establish a fistulous communication between the stomach and the skin surface so that the patient may be fed. The various operations about to be described differ only in minor details. They may be divided into those in which the tract is mucous-lined and those in which the tract is serous-lined. The former type of operation is performed when a permanent fistula is required, as for an impassable simple stricture of the œsophagus, the latter type being undertaken when it appears that the patient has but a short time to live, e.g. for cancer of the cardiac end of the stomach, or as a palliative measure in those conditions in which the disease is capable of being corrected, e.g. peptic ulcer of the lower end of the œsophagus.

*Stamm's Operation.* This is the simplest and most satisfactory type of gastrostomy for cancer of the cardia, and is recommended in preference to the other methods about to be described. The operation can be performed quite easily under a local anæsthetic. Inhalation anæsthetics should as a rule be avoided owing to the likelihood of subsequent chest complications. The incision, which is made over the upper and outer third of the left rectus muscle, commencing at the costal margin and proceeding vertically downwards, should be as small as possible, as, owing to the patient's poor general condition, suppuration in the wound and burst abdomen are frequent. It also affords the easiest and most direct access to the contracted stomach found in these cases. The cut margins of the peritoneum and posterior sheath of the rectus are picked up and drawn apart with Littlewood forceps. As the stomach is small, tubular, and lies tucked away under

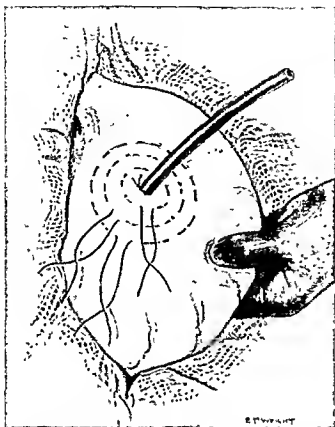


Fig. 210.—GASTROSTOMY. STAMM'S METHOD. A TUBE IS INTRODUCED INTO THE STOMACH AND FIXED BY A SINGLE STITCH. THREE PURSE-STRING SUTURES HAVE BEEN INSERTED IN THE MANNER SHOWN AND ARE READY TO BE TIED, STARTING WITH THE ONE NEAREST TO THE TUBE.

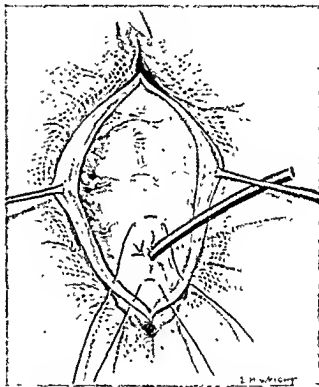


Fig. 211.—STAMM'S GASTROSTOMY. THE PURSE-STRING SUTURES ARE TIED. SECTION SHOWING THE RESULTING INVERTED COVE OF STOMACH WALL.



the liver, the first structure which comes to view is usually the great omentum or the transverse colon. By gently pulling the colon downwards, the upper portion of the stomach comes into view and can be delivered through the wound. The portion selected for the gastrostomy should be in the body of the stomach on the anterior wall, high up and as far removed as possible from the pylorus.

The wound is carefully packed off with gauze swabs and mackintosh squares soaked in saline to prevent any contamination of the wound



*Fig. 212.*—GASTROSTOMY. STAMM'S OPERATION COMPLETED BY ANCHORING THE STOMACH TO THE PERITONEUM AT THE LOWER END OF THE ABDOMINAL INCISION. THE UPPER SUTURE IS TIED ABOVE AND THE LOWER SUTURE BELOW THE TUBE.

or peritoneal cavity during the further steps of the operation. A point midway between the greater and lesser curvatures on the anterior wall of the body of the stomach is selected for the insertion of the tube, and the site is marked off by two pairs of Allis forceps which seize the stomach and lift it upwards.

A small opening, sufficiently large to admit a No. 12 or No. 14 Jaques catheter, or a length of rubber tubing of about the same diameter, is then made between the Allis forceps in the anterior wall of the stomach, either with a knife or with a Post electric cautery. The

rubber tube is inserted into the stomach cavity for 2-3 inches and is attached to the margins of the wound by a catgut suture which picks up all the coats of the stomach and a portion of the tube. A sero-muscular purse-string suture is introduced half an inch away from the tube, and as it is tightened the tube is pushed into the cavity of the stomach, the suture being tied firmly enough to grip without compressing the tube (fig. 210). A second and a third purse-string suture are similarly applied and the margins around the tube are further invaginated, with the result that a cone-shaped portion of the stomach projects into the cavity of the organ, embracing the tube (fig. 211).

When the last purse-string suture has been tied the stomach is anchored to the peritoneum and posterior sheath of the rectus muscle in the manner depicted in figure 212. The abdominal wound is then closed with interrupted sutures, the gastrostomy tube being made to project through the lower end of the incision. The tube is

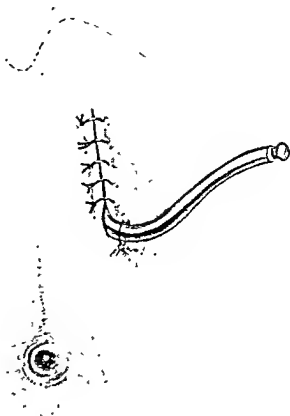


Fig. 213—GASTROSTOMY. THE TUBE IS DRAWN THROUGH THE LOWER PORTION OF THE INCISION AND ANCHORED TO THE SKIN BY A STITCH WHICH ENCIRCLES THE TUBE.

plugged with a spigot and fastened to the skin by an encircling stitch which prevents it from being accidentally withdrawn (fig. 213).

Before the patient leaves the theatre some warm citrated milk is introduced through the tube to test its patency and smooth working, and also to supply the patient with the nourishment he much needs.

*After-Treatment.* Fluid nourishment is introduced through the tube every two hours during the day and once or twice during the night. It is better to start with small frequent feeds and gradually increase the

amount until the patient is receiving sufficient at long intervals. At first only 2-3 oz. can be given at a time, but later the stomach will be found to tolerate as much as 5-10 oz. The fluids most frequently used for feeding the patient consist of citrated milk, concentrated glucose solution, beef tea, Allenbury's or Bengers' food, Ovaltine, egg, orange juice, etc.

The tube works loose in 10-14 days and should be removed, sterilised by boiling, and replaced. Apart from this it should be kept continuously in its original position, as otherwise the fistula tends to contract. If, on the other hand, the aperture becomes unduly stretched a larger tube may be inserted to prevent leakage, or the fistula may be permitted to contract by removing the tube for a few hours every day.

Before the patient is discharged he should be instructed how to remove and re-insert the tube, clean it, and feed himself through it.

**Witzel's Operation.** This operation is indicated where the stomach is so small and tubular that it is very difficult or even impossible to raise a cone from the anterior wall as in Stamm's operation.

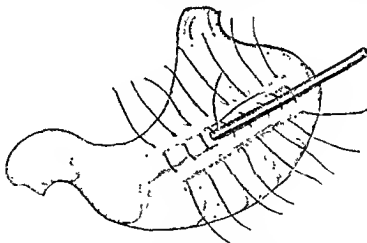
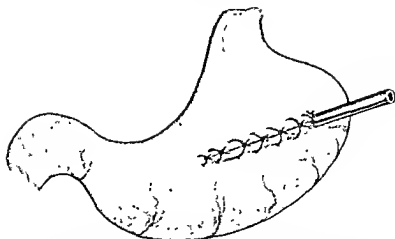


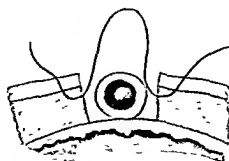
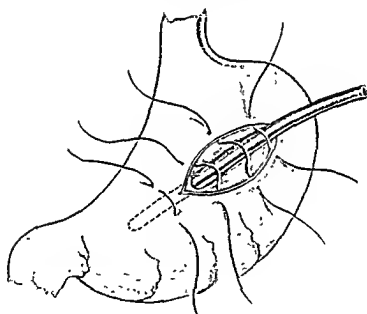
Fig 214.—GASTROSTOMY. WITZEL'S METHOD. THIS FIGURE SHOWS THE METHOD OF INSERTING THE SUTURES.

The abdominal incision, the exposure, and the method of delivering the stomach are the same as in the operation just described.

A No. 12 or No. 14 rubber catheter, or a piece of rubber tubing of similar diameter, is introduced for 2-3 inches into the cavity of the stomach through a small puncture made in the anterior wall, midway between the greater and lesser curvatures. The tube is fixed to the



*Fig. 215.—GASTROSTOMY. WITZEL'S METHOD. THE SUTURES HAVE BEEN TIED AND CUT SHORT. THE OPERATION IS COMPLETED BY ANCHORING THE STOMACH TO THE MARGIN OF THE PERITONEUM.*



*Fig. 216.—GASTROSTOMY. MARWEDEL'S METHOD. THE LOWER FIGURE SHOWS THE PARTS IN SECTION.  
(After Karachner, modified)*

incision by a single catgut stitch or purse-string suture which pierces the tube.

The catheter or rubber tube is then laid on the stomach for 2 inches and pressed firmly into its surface so that it lies in a gutter or groove of stomach wall. This groove is then converted into a tunnel by introducing a series of interrupted Lembert sutures in the manner shown in figure 214, two or three interrupted sutures being passed beyond the opening in the stomach to guard against the possibility of leakage. The tube, therefore, lies in a serous-lined tunnel and a valvular effect is produced (fig. 215).

A small stab wound is next made, 1 inch or so to the outer side of the original abdominal incision, and through this the gastrostomy tube is drawn. The surrounding stomach wall is carefully anchored to the peritoneum and posterior sheath of the rectus muscle, after which the abdominal incision is closed.

*Maricdel's Operation.* This is a modification of Witzel's method. An incision  $1\frac{1}{2}$ –2 inches long is made through the sero-muscular coat in the anterior wall of the body of the stomach about midway between the curvatures, down to but not through the mucous membrane. At the lower end of this incision the mucous membrane is perforated and a catheter is introduced into the cavity of the stomach for 2–3 inches. The catheter, being fixed to the margin of this little opening by a catgut stitch, is then made to lie in a tunnel by suturing the edges of the sero-muscular incision with a series of interrupted Lembert sutures (fig. 216). This method has no advantages over Witzel's more simple operation.

*Kader's Operation.* The stomach is delivered through the abdominal incision and a catheter or rubber tube is inserted into the stomach as in Stamm's gastrostomy. Two vertical and parallel sero-muscular folds of the anterior wall of the stomach are then drawn together, above and below the tube, by the introduction of a few Lembert sutures. The suture line is further invaginated by the introduction of another series of sutures which pick up the stomach wall on either side of the original line of sutures (fig. 217). The gastrostomy tube is thus buried by a two-fold pleat of stomach wall, and a cube, instead of a cone as in Stamm's operation, is made to project into the cavity of the viscus.

*Lepage-Jancway Operation.* This operation is indicated where a permanent gastro-cutaneous fistula is required for feeding purposes.

A left paramedian or a left transrectus or muscle-split incision, commencing at the costal margin and proceeding downwards for about 2 inches, is the incision of choice. A large cone of the anterior wall of the stomach, high up and as near to the fundus as possible, is drawn through the wound, which is carefully protected and packed off with mackintosh squares and gauze swabs. A flap of the anterior wall of the stomach, 2 inches by 1 inch, with its long axis stretching from the greater to the lesser curvature, and with its base on the greater curvature, is demarcated by picking up the surface of the stomach

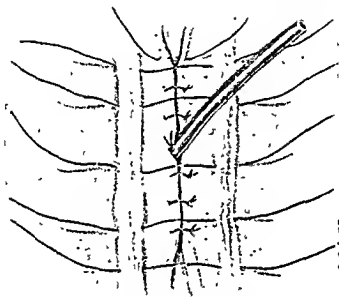


Fig 217.—GASTROSTOMY. KADER'S METHOD.  
(After Moynihan, modified.)

with four pairs of Allis forceps. Two pairs of these forceps mark the free upper end of the flap and should be placed about 1 inch apart, whereas the forceps which mark the base of the flap are about  $1\frac{1}{4}$  inches apart.

An incision about 1 inch long is then made parallel to the lesser curvature just above the two upper pairs of Allis forceps, and extending down to the mucous coat. All bleeding points are picked up and tied before the stomach is opened with scissors at this site, and a suction tube is introduced to aspirate the gastric contents. A rectangular flap of the anterior wall of the stomach is then cut through with scissors. The Allis forceps on the lesser curvature grasp the whole thickness of the free end of the flap which is turned back, its base acting as a hinge (fig. 218).

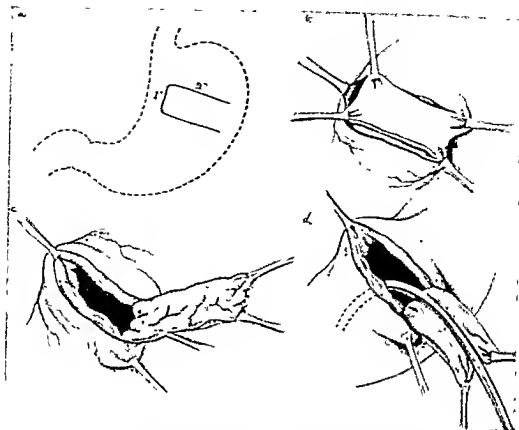


Fig. 219.—GASTROSTOMY. THE LEPAGE-JANEAU OPERATION.

- (a) THE POSITION AND EXTENT OF THE FLAP MADE IN THE ANTERIOR WALL OF THE STOMACH.  
 (b) THE POINTS AT WHICH THE ALLIS FORCEPS ARE APPLIED TO CONTROL THE FLAP AND FACILITATE SUBSEQUENT SUTURING.  
 (c) THE FLAP TURNED DOWNWARDS PRIOR TO INSERTING THE TUBE INTO THE STOMACH CAVITY.  
 (d) THE TUBE IN POSITION.

(Drawn from Horsley's "Stomach and Duodenum," Kimpton; with slight modifications. By kind permission.)

A No. 12 or No. 14 Jaques catheter, or a piece of rubber tubing of similar diameter, is introduced into the stomach for 2-3 inches and made to lie in the centre of the rectangular flap. Firm downward traction is made on the end of the flap and upward traction on the upper end of the wound in the stomach to facilitate the suturing of the mucous membrane. Starting at the lesser curvature, the edges of the mucous membrane are drawn together with a continuous through-and-through suture which picks up only the mucous membrane, continues over the flap, and buries the tube. When it reaches the extremity of the flap the suture is tied, one end being kept long. A second suture of No. 0 or No. 00 20-day chromic catgut, which may be a continuous Lambert or a right-angle suture, brings together the serous and muscular coats. When this suture reaches the end of the flap it is knotted and clamped to the end of the tube with a hæmostat. At the point where the tube

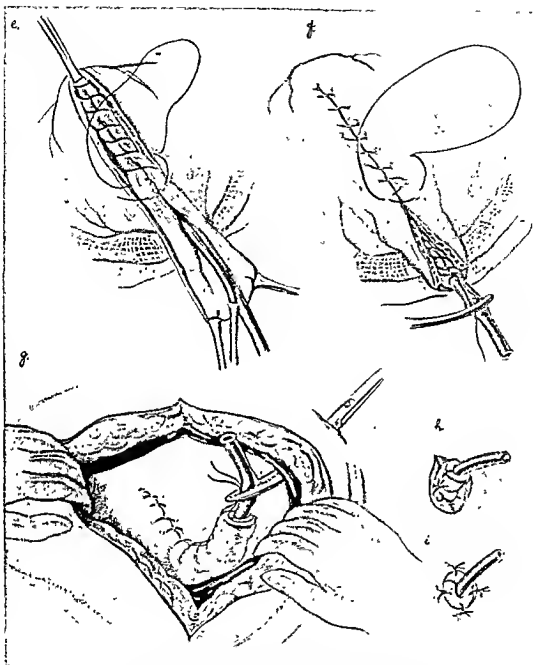


Fig. 210.—GASTROSTOMY. THE LEPAGE JANKWAY OPERATION.

- (e) THE MUCOUS MEMBRANE IS CLOSED AROUND THE TUBE BY A CONTINUOUS SUTURE. HORSLEY EMPLOYS A LOCKSTITCH FOR SUTURING THIS LAYER
- (f) THE SERO-MUSCULAR SUTURE IS ALMOST COMPLETE. HERE HORSLEY USES A CONTINUOUS CUSHING INVERTING STITCH.
- (g) THE RUBBER TUBE AND THE NEWLY CONSTRUCTED TUBE OF STOMACH WALL ARE DRAWN THROUGH A SEPARATE STAR INCISION.
- (h) and (i) THE TUBE OF STOMACH IS ANCHORED TO THE RECTUS SHEATH, AND THE MUCOUS MEMBRANE TO THE MARGINS OF THE SKIN INCISION. THE ABDOMINAL WOUND IS THEN CLOSED

(Drawn from Horsley's "Stomach and Duodenum," Kington; with slight modifications By kind permission.)



emerges from the gastric flap it is fixed by a single catgut stitch which pierces all the coats of the flap and a portion of the tube.

The gastric flap and the tube are made to project through the lower portion of the incision or are drawn through a small stab wound which is made to the left of the abdominal incision. The end of the continuous sero-muscular suture is threaded through a needle which picks up the anterior sheath of the rectus and stitches the flap to it. Sufficient traction is then made on the mucosal stitch to facilitate this layer being sutured to the skin margins of the stab wound or to the lower portion of the abdominal incision (fig. 219). The abdominal wound is then closed in the usual manner.

Quick and Martin (*Surg., Gynec., and Obst.*, xlv, 426, 1928) reported a series of 172 cases in which this operation was performed with a mortality of 18 per cent. Since 1928, however, by modifying their technique to guard against wound infection, Martin and Watson were able to report 52 cases with a mortality of 3, i.e. 5.8 per cent.

Three oz. of peptonised milk are given three-hourly through the tube, this being increased until the patient is receiving 8-10 oz. four-hourly.

During the first three or four post-operative days rectal or intravenous salines should be given to combat the dehydration which is such a marked feature of these cases.

The tube is removed on the eighth day and is inserted again only when it is required for feeding purposes. The gastric fistula proves most satisfactory and leakage is minimal.

### *Jejunostomy.*

In cases of cancer of the stomach jejunostomy may be indicated:

- (1) As an alternative procedure to gastrostomy.
- (2) Where the invasion of the stomach wall with growth is so extensive that it renders the performance of a gastro-jejunostomy or a gastrostomy impossible.
- (3) In cases of irremovable malignant hour-glass stomach.
- (4) Where there is obstruction of the duodeno-jejunal flexure or proximal jejunum, due either to direct spread of growth from the stomach or to malignant glands at the root of the mesentery (fig. 220).

In desperate cases, where the simpler operation of Witzel is adequate under the circumstances, Coffey's or Mayo-Robson's operation would appear unnecessarily complicated.

*Gastro-Jejunostomy.* Gastro-jejunostomy is only indicated for irremovable cancer of the pylorus associated with obstruction. Although cases have lived 2 years or more following this operation for growth of the pyloric portion of the stomach, the average expectancy of life is about 8 months, i.e. about 2 months more than where simple exploration has been performed.

The operative mortality of gastro-jejunostomy for cancer of the stomach is about 10-15 per cent.



Fig. 220.—OBSTRUCTION OF THE DUODENO-JEJUNAL FLEXURE DUE TO CARCINOMA OF THE STOMACH AND MALIGNANT GLANDS IN THE ROOT OF THE MESENTERY. WITTEL'S JEJUNOSTOMY WAS PERFORMED UPON THIS CASE, AFFORDING IMMEDIATE RELIEF AND COMPARATIVE COMFORT FOR THE REMAINING NINE MONTHS OF THE PATIENT'S LIFE. (Author's case.)

### *Exclusion of the Growth.*

This operation, devised by Devine, gives greater relief than gastro-jejunostomy and is associated with a mortality which is no higher. In the majority of cases, at least it prevents the patient from dying of obstruction, and the immediate effects following the operation are sometimes very marked. Where, therefore, a growth proves irremovable but there is a sufficiency of healthy tissue proximal to it, the stomach should be transected, the distal end closed, and an anastomosis made between the open end of the stomach and the proximal jejunum. Secondaries in the liver, which cause less pain than obstruction, are usually the cause of death in these cases.

Balfour considers that this operation has a wider application than

has hitherto been accorded to it in special types of inoperable gastric cancer. He has often been surprised at the length of comfortable life which followed the operation when it was clearly indicated.

### *Excision of the Growth.*

Wherever possible, even in cases in which there are secondaries in the liver or obvious metastatic implants in the regional lymph glands, if the tumour mass can be resected together with a healthy portion of the stomach, this should be undertaken, as by performing partial gastrectomy on these incurable cases the patient is rid of a foul necrotic sloughing mass, toxæmia is markedly diminished, obstruction is prevented, and a period of improved health and comparative freedom from pain is assured for at least a few months.

## PARTIAL GASTRECTOMY

### *General Considerations.*

*Suture and Ligature Material.* The blood-vessels and omenta are tied off with silk, linen thread, or catgut, according to the preference of the individual surgeon. I find No. 1 or No. 2 20-day chromic catgut strong enough and reliable enough for the ligature of the larger blood-vessels and for tying off portions of the omenta. Some surgeons use silk or thread sutures for anastomosis, whilst others prefer to use catgut only throughout the operation. I usually use a continuous suture of No. 0 20-day chromic catgut for invaginating the duodenal stump, but I insert a few reinforced sutures of fine silk or thread to strengthen the suture line even further. For sero-muscular sutures I use fine silk or thread, but for the through-and-through inner suture I prefer No. 0 or No. 00 20-day chromic catgut.

The ideal ligature and suture material has not yet been discovered. It is not uncommon to find a surgeon, who has for many years used only catgut for his ligatures and sutures in abdominal work, adopt the use of silk or thread, or vice versa. But most seem to show a final preference for catgut. Although catgut is in a general way very satisfactory, I myself prefer to use some silk sutures in gastrectomy cases for the sake of the additional security afforded, especially where the tissues are in poor condition and the powers of healing retarded.

*Clamps.* There are three types of clamp used in gastrectomy operations: (a) The Friedrich-Petz clamp (fig. 221); (b) Cruskies

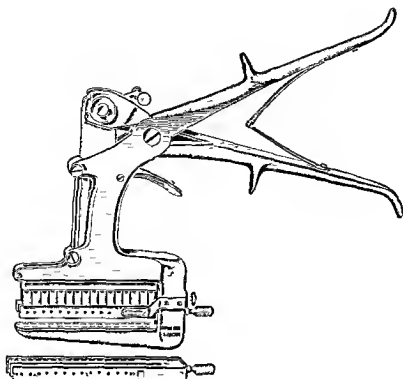


Fig. 221.—THE FRIEDRICH PETE CLAMP.

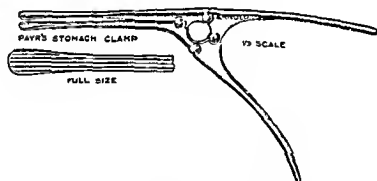


Fig. 222.—THE PAYE CLAMP.

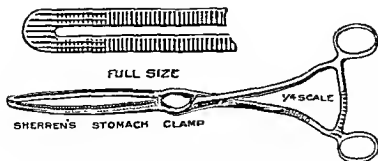


Fig. 223.—THE SHERREY CLAMP.

clamps or enterotribes, e.g. Payr's (fig. 222); and (c) Gastro-jejuno-stomy clamps, e.g. Sherren's (fig. 223), of which some are made with strong firm blades, others with soft flexible blades.

I frequently use the Petz clamp for resections. The small Payr clamps are used for crushing the duodenum at the site of resection. For anastomosis I generally use Sherren clamps, the blades of which are protected by rubber tubing, although clamps do not form an essential part of the operation and can be dispensed with in a number of cases. These rubber-covered clamps should always be applied very carefully and gently by the surgeon himself, never firmly enough to prevent bleeding, and never so firmly as to damage the friable mucosa. Clamps used in this manner do not injure the gut nor do they interfere with the subsequent nutrition of the parts concerned in the anastomosis. They also render the operation easier to perform, facilitate suturing, and prevent a great deal of contamination.

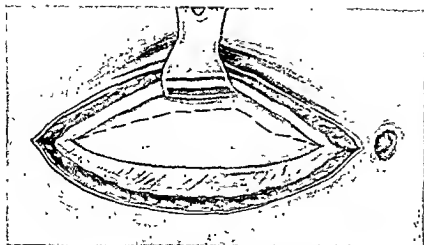
*Cautery.* I often use a Post electric cautery (fig. 224) for cutting across the duodenum between the Payr clamps, and occasionally for



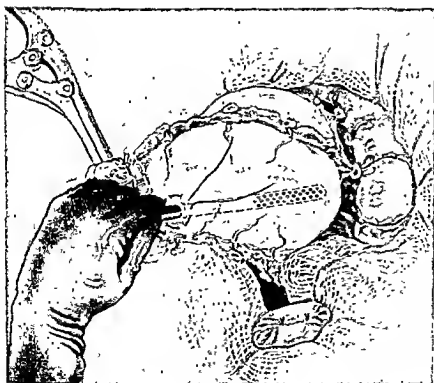
Fig. 224.—THE POST ELECTRIC CAUTERY.

making incisions into the gut. It is also a useful instrument for cauterising or destroying small chronic peptic ulcers, as in Balfour's operation.

*Suction Apparatus.* A suction apparatus should be kept handy during the performance of all operations upon the stomach, and is indispensable where anastomosis is carried out without clamps. If at operation the stomach is found to be distended with fluid, it is an easy matter to introduce the suction tube into the cavity of the stomach through a small incision, and to withdraw all the contents before proceeding with the operation. This greatly facilitates all subsequent manœuvres and makes the operation much more simple than if the stomach were tense and distended (fig. 225). It is certainly a more effective method of emptying the stomach than passing a stomach tube through the mouth or nostril.



*Fig. 226.*—Mid Line Epigastric Incision. The Skin Incision is made strictly in the Mid-line, between the Xiphisternum and the Umbilicus. The Recti are separated and the Peritoneum exposed. The Dotted Line shows the Line of Incision through the Peritoneum. This Incision in the Peritoneum is curved outwards to the Left to avoid the Ligamentum Teres.



*Fig. 225.*—The Introduction of a Section Tube during the Performance of a Partial Gastrectomy to Empty the Distended Stomach prior to Proceeding with the Operation  
(After Balfour, [modified].)

*Choice of Incision.* There is a choice of numerous incisions, but probably not more than five are employed in gastric surgery.

(a) *Mid-line Incision.* This incision, which is the one most frequently employed in gastric surgery, commences at the tip of the xiphisternum and proceeds vertically downwards to a point just above the umbilicus. The incision is carried through the linea alba, and the peritoneum is exposed. As the ligamentum teres lies more or less in a line with the incision, it is advisable to open the peritoneum to the right, or preferably to the left, of the middle line and to avoid it by retracting the muscle outwards (fig. 226).

(b) *Right Paramedian Incision.* This incision commences 1 inch to the right of the xiphisternum and extends vertically downwards to a point 1-2 inches below and to the right of the umbilicus. The anterior sheath of the rectus is opened and the underlying rectus muscle is dissected from its sheath as far as the middle line so that it can easily be retracted outwards. This exposes the posterior sheath of the rectus, which is then incised together with the underlying peritoneum for the whole length of the incision.

(c) *Right Transrectus or Muscle-split Incision.* This incision commences over the costal margin on the right side and proceeds vertically downwards at the junction of the inner and middle thirds of the upper portion of the right rectus muscle for about 5 inches. All the structures in the line of this incision are divided, including the muscle itself.

(d) *Left Paramedian Incision.* This incision is in every respect similar to the right paramedian, but is placed to the left instead of to the right of the middle line. It is employed in certain cases of cancer of the stomach where the growth is situated high up on the lesser curvature, in order to afford easier approach to the cardiac end of the stomach.

(e) *Transverse Incision.* A transverse epigastric incision is employed by some surgeons, but it is one which I personally seldom employ as I consider the exposure afforded by it to be somewhat cramped and inadequate.

In the making of all these incisions it is important that hæmostasis should be complete, even the most minute blood-vessels being clipped; there must also be no fraying, bruising, tearing, or other damage to the fibres of the rectus muscle.

Before the peritoneal cavity is opened tetra-cloths are fixed to the skin margins and the rectus muscle is covered with mackintosh squares or gauze swabs. A small red towel is put on a special tray at the foot of the operating table, and on this tray the soiled instruments are placed. All the instruments laid on this towel are considered as contaminated and should not be touched again by the surgeon or his assistants, but be removed by the nurse and re-sterilised.

When the anastomosis is complete the surgeon and his assistants should change their rubber gloves and put on fresh sterile ones.

*Exploration of the Abdomen to Determine Operability.* This is conducted in the following manner :

*Examination of the Stomach.* The edges of the wound are widely retracted and the stomach is inspected and carefully examined to determine its *size, shape, and position*. The large ptosed stomach usually lends itself easily to gastrectomy, whereas the small contracted tubular steer-horn stomach presents considerable difficulty in resection in that it lies high up and is tucked away under the liver.

The stomach is then methodically palpated to determine the *position and extent of the growth*. Growths situated in the *region of the cardiac orifice* are usually impossible to resect as they frequently involve the lower portion of the œsophagus. If in such cases the intra-abdominal portion of the œsophagus is long and unduly mobile, it may be feasible in exceptional cases to perform total gastrectomy.

In the majority of cases of cancer of the cardia no radical operation is possible, and gastrostomy or jejunostomy must be undertaken to prevent the patient from starving to death.

Malignant growths situated high up in the *fundus* or on the *greater curvature* are usually of the polypoid type and are slow-growing. Total excision of the stomach is often possible in such cases.

The outlook with growths springing from the *body of the stomach*, and particularly with those arising from the region of the greater curvature, is good as they lend themselves readily to partial gastrectomy. It is said that fully 50 per cent of cancers of the stomach originate in the *pyloric portion*, and that growths in this position, as they produce symptoms early in the course of the disease and are operated upon without delay, give the most favourable prognosis. This, however, is not generally the case, as infiltration of the underlying head of the pancreas and fixation of the growth to this structure,



together with metastatic and often widespread implants in the regional lymph nodes, soon takes place, thus reducing the chances of permanent cure.

With leathery scirrhus growths situated actually in the *pyloric canal*, the prognosis is good as, owing to the early advent of obstruction, operation is often performed before the glands have become involved. With growths in this region the supra-pyloric and infra-pyloric groups of glands must be very carefully examined, and the mobility of the pylorus determined before proceeding with the operation.

In practice, malignant growths situated about the *middle third of the lesser curvature* prove more easy to resect than those in the pyloric region, and the affected glands can be removed without difficulty. An important step in the operation is the palpation of the whole length of the lesser curvature to discover to what extent it is involved, as if it is apparent that the growth has spread upwards and invaded the region of the cardiac orifice no radical operation can be attempted. Nevertheless it should be remembered that undue thickening of the lesser curvature may be due to fibrosis, œdema, or even muscular hypertrophy rather than to growth.

Partial gastrectomy, or preferably total gastrectomy, should be performed for the *localised variety of leather-bottle stomach*. In the *generalised variety*, even if a small margin of healthy stomach wall remains between the growth and the cardiac orifice, total gastrectomy should be undertaken. If resection proves impossible, no palliative procedure is worth while and the abdomen should be closed.

It must be emphasised that *fixation of the stomach* may be due to inflammatory adhesions, and does not necessarily preclude resection. Fixation of the stomach to the anterior edge of the liver may similarly be due to inflammatory adhesions, and even where it is the result of direct spread of growth a small margin of the liver may be excised in order to facilitate the mobilisation of the stomach prior to resection. In certain cases, where the growth is anchored to the pancreas, it is possible to shave away a part of the gland with the attached portion of stomach. But *wide fixation and extensive involvement of the pancreas* render the case inoperable. In certain cases, where the transverse colon has become invaded with growth, either directly or through the median of the great omentum, it may be possible to resect a portion of the transverse colon together with the stomach and great omentum.

*Enlargement of the lymphatic glands* does not necessarily entail involvement by cancer. Although Balfour states that a patient may

be cured after partial gastrectomy even where all involved lymph nodes are not removed, it is nevertheless true that if there is a mass of metastatic glands in the upper part of the lesser curvature near the cardiac orifice, the chances of cure are doubtful even after resection of the stomach and removal of some of the glands. The lymph glands must therefore be examined systematically, particularly the group which surrounds the pyloric portion of the stomach and the first part of the duodenum. The glands along the lesser curvature, the coeliac axis group, those hidden in the portal fissure, and those at the root of the mesentery, should likewise be palpated in all cases.

Having completed the examination of the lymph glands the *omenta* are next scrutinised for areas of involvement or seedlings of growth. Local involvement of the omenta does not exclude the possibility of resection; widespread growth in the omenta, however, spells inoperability.

The *liver* should be inspected for secondaries and palpated to detect any hurried nodular growths or irregularities on its surface. The *peritoneum* should also be examined for minute seedlings of growth—carcinomatosis, and a hand should be passed into the recto-vesical pouch to discover whether metastatic growths are present in this region. In the female both *ovaries* are examined as a routine, in order to discover if tumour masses have formed here through cancer cells having alighted by gravitation upon their surfaces.

*Choice of Operation.* Most surgeons acquire a preference for those operations which have served them best. No two surgeons will perform the operation of partial gastrectomy in exactly the same manner. The Billroth I and II operations are to-day different in almost every respect except mechanics from those practised by the surgeon from whom they derive their name. All partial gastrectomies are modifications of the Billroth I and II. Although there are certain surgeons who perform the Billroth I for cancer of the stomach, I consider that in the majority of cases it is not such a satisfactory procedure as the Billroth II, or as the numerous modifications of the Polya operation. In the former, the surgeon is concerned more with the approximation of the gastric stump to the open end of the duodenum than with wide excision of the growth, and he may consequently sacrifice too little stomach in order to achieve an easy junction. Again, should there be a recurrence of the growth, the small stoma soon becomes blocked.

*Essentials of Partial Gastrectomy Operations :*

(1) Perfect technique. This includes :

- (a) Scrupulous cleanliness.
- (b) Absolute hæmostasis, the minutest blood-vessels being tied.
- (c) Carefully approximated suture lines which are sufficiently reinforced with interrupted sutures.
- (d) Correct mechanics.

(2) Excision of the growth with a wide margin of healthy tissue above and below the diseased area. This would also include :

- (a) Removal of the whole of the first part of the duodenum.
- (b) Removal of the whole of the lesser curvature of the stomach.
- (c) Removal of *at least* half of the greater curvature of the stomach.
- (d) Removal of the regional glands, such as the supra-pyloric, retro-pyloric, and infra-pyloric, the glands along the lesser curvature—the lower coronary and upper coronary, the right paracardial glands, and those along the greater curvature—the right gastro-epiploic.
- (e) Removal of the greater portion or the whole of both omenta.

The great omentum may be detached from the colon and removed, this being considered by some surgeons to be an essential step in the operation.

(1) *The Polya-Moynihan Operation.* This operation is commenced by picking up a portion of the gastro-colic omentum with forceps and making an incision into a bloodless area so as to expose the lesser sac. The margins of this opening in the gastro-colic omentum are drawn widely apart to permit of an easy exploration of the posterior wall of the stomach and pancreas. The stomach is lifted up and any adhesions which bind it to the pancreas are separated. The omentum is then stripped from the underlying mesocolon, the middle colic artery is identified, and a large abdominal swab is packed into the omental bursa. The right portion of the great omentum, as close to the upper border of the transverse colon as possible, is picked up.

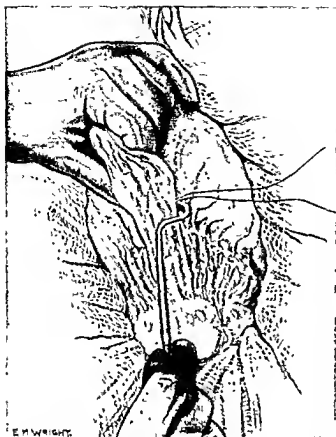


Fig. 227.—PARTIAL GASTRECTOMY. A METHOD OF LIGATING PORTIONS OF THE GASTRO-COLIC OMENTUM.

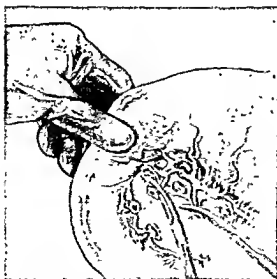


Fig. 228.—PARTIAL GASTRECTOMY. LIGATURE OF THE BLOOD VESSELS BELOW THE PYLORUS AND ON THE MEDIAL ASPECT OF THE FIRST PART OF THE DUODENUM, PREPARATORY TO MOBILISING THIS PORTION OF THE GUT.  
(After Balfour, modified)

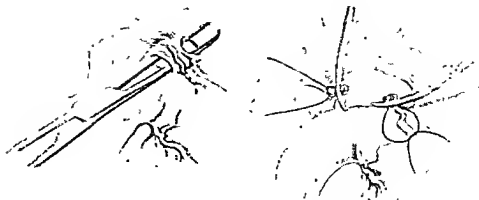


Fig. 222.—PARTIAL GASTRECTOMY. LIGATURE OF THE PYLORIC ARTERY.

piece by piece, and divided between stout ligatures (fig. 227). As the separation approaches the duodenum care must be taken not to injure the middle colic artery or any of its branches, as in this region the gastro-colic omentum is often adherent or congenitally fused to the mesocolon.

When the lower border of the first part of the duodenum is reached, the right gastro-epiploic artery is ligatured and divided (fig. 228). The stomach and duodenum are very gently separated from the underlying head of the pancreas by gauze dissection or division of adhesions. The hand is then passed under the stomach just to the right of the incision, and the finger tears through the gastro-hepatic omentum as near to the

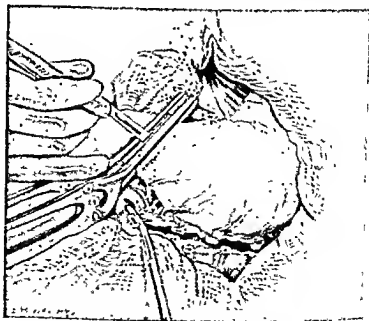


Fig. 236.—PARTIAL GASTRECTOMY. THE FIRST PORTION OF THE DUODENUM IS BEING DIVIDED BY A KNIFE, BETWEEN THE STAP CLAMPS.

liver as possible. Then, by drawing the stomach downwards and over to the left, the pyloric artery is rendered taut and clearly visible. When this artery has been identified, it is clamped in two places, divided between the forceps, and ligatured with strong catgut (fig. 229). The upper portion of the duodenum can now be further freed from the pancreas.

The duodenum is clamped by two small Payr clamps, which are applied side by side about  $\frac{3}{4}$ –1 inch away from the pylorus. A gauze pack soaked in saline or a mackintosh square is passed underneath the duodenum and pyloric portion of the stomach, and the gut is divided between the clamps with a knife which has been dipped in phenol, or with a Post electric cauter (fig. 230). The clamp on the gastric side of the duodenum is covered with a mackintosh square and drawn well over



Fig. 231.—PARTIAL GASTRECTOMY. CLOSURE OF THE DUODENUM. APPLICATION OF A SUTURE OVER THE PAYR CLAMP.

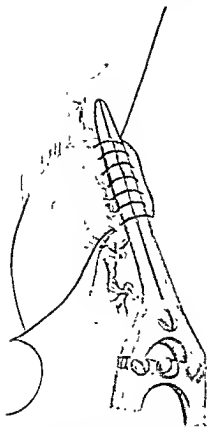


Fig. 232.—PARTIAL GASTRECTOMY. CLOSURE OF THE DUODENUM. A RUNNING SUTURE HAS BEEN APPLIED OVER THE PAYR CLAMP.

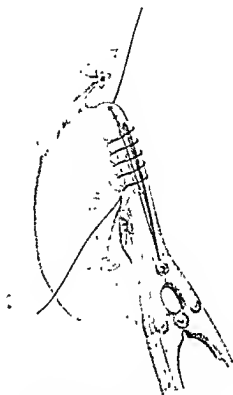


Fig. 233.—PARTIAL GASTRECTOMY. CLOSURE OF THE DUODENUM. THE SUTURE IS NOW TIGHTENED AS THE PAYR CLAMP IS WITHDRAWN.

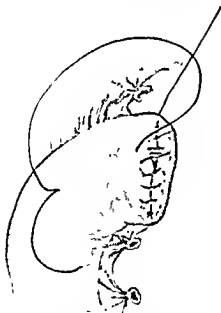
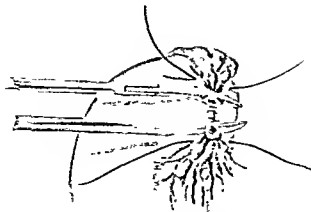


Fig. 234.—PARTIAL GASTRECTOMY. CLOSURE OF THE DUODENUM BY A RUNNING SUTURE. THE SUTURE IS SHOWN RETURNING ALONG THE GUT TO ITS STARTING POINT. WHEN THE ENDS ARE TIED A PURSE-STRING EFFECT IS PRODUCED.

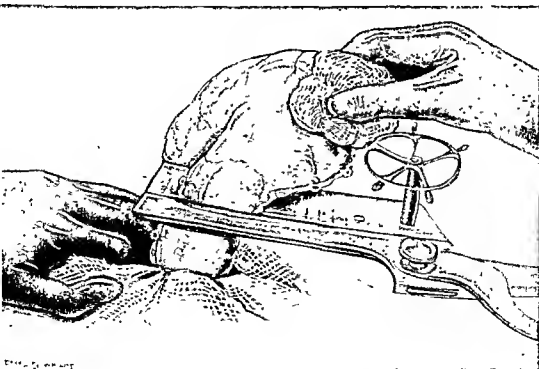


Fig. 235.—PARTIAL GASTRECTOMY. CLOSURE OF THE DUODENAL STUMP COMPLETED.



*Fig. 236.*—PARTIAL GASTRECTOMY. THE CLOSED END OF THE DUODENUM IS PROTECTED BY DRAWING ADJACENT TAGS OF OMENTUM OVER THE SUTURE LINE AND ANCHORING THEM IN THE MANNER SHOWN.

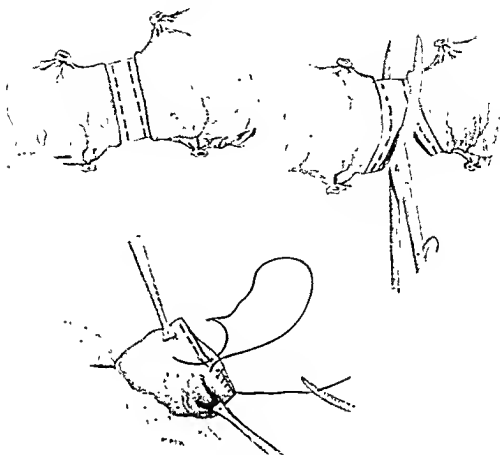
to the left, while the other clamp is rotated laterally to expose the under-surface of the duodenum, permitting of a little further mobilisation of the duodenal stump which is then sutured over with a right-angle continuous stitch, this being drawn tight as the clamp is removed (*fig. 231*). This stitch is carried back to the starting-point and tied firmly, producing a purse-string effect (*figs. 232, 233, 234 and 235*).



*Fig. 237.*—PARTIAL GASTRECTOMY. PETAL CLAMP APPLIED TO THE FIRST PART OF THE DUODENUM, READY FOR THE APPLICATION OF THE CLIPS.

(Adapted from Kirschner's "Operative Surgery," Lippincott, by kind permission of Julius Springer, Berlin.)





*Fig. 234*—PARTIAL GASTRECTOMY. THE APPEARANCE OF THE CRUSHED FIRST PORTION OF THE DUODENUM AFTER THE REMOVAL OF THE PETZ CLAMP.  
DIVISION OF THE CRUSHED GUT BETWEEN THE TWO ROWS OF CLIPS.  
METHOD OF INVAGINATING THE DUODENAL STUMP.

The suture line is further reinforced with a few interrupted mattress or Lembert sutures of fine silk or thread, and wisps of omentum are drawn across the sutured area and tied into position to afford additional protection (fig. 236).

Where the duodenal stump is very mobile its cut end, after being closed in the above manner, may be further invaginated by a purse-string suture.

As an alternative to dividing the duodenum between the Petz clamps, the gut may be crushed with a Petz instrument and cut across between the two rows of clip sutures (fig. 237). The duodenal stump, including its row of Petz clips, is sewn over with a continuous Lembert suture and buried, the suture line being further reinforced with interrupted stitches of fine silk (fig. 238). The greater part of the left half of the gastro-colic omentum is now ligatured off, piece by piece, just

above the transverse colon, and the left gastro-epiploic artery is secured and tied with No. 2 20-day chromic catgut, about the middle of the greater curvature or preferably a little higher up than this (fig. 239).

As ligature of the omenta and blood-vessels proceeds, pains must be taken to keep well outside the zone of the infected glands which are left attached to the portion of the stomach about to be resected. The

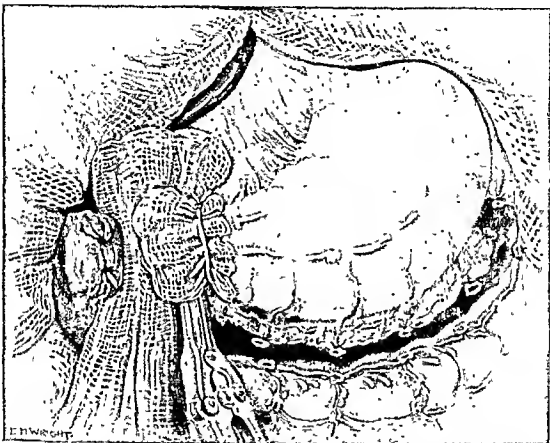


Fig. 239.—PARTIAL GASTRECTOMY. THIS FIGURE DEPICTS THE STAGE IN THE OPERATION IMMEDIATELY BEFORE THE DIVISION OF THE GASTRO-HEPATIC OMENTUM AND THE LIGATION OF THE CORONARY ARTERY. THE LEFT GASTRO-EPILOIC ARTERY HAS JUST BEEN TIED.

coronary artery must next be sought and ligatured near its origin. If the stomach is distended with fluid and gas it is best to aspirate its contents by introducing a suction tube through a small stab incision which is made in the posterior wall, close to the pyloric region. This incision is encircled by a purse-string suture, which is tied as soon as the suction tube is removed (fig. 240). The empty and flaccid stomach is easy to manipulate, and the subsequent steps of the operation are rendered very much easier.

There are many methods of securely ligaturing the coronary artery

near its origin, and when this vessel, which acts as an anchor, is divided the stomach can be lifted from its bed without difficulty. One method is for the assistant to grasp the free end of the stomach with the right hand, pull it firmly downwards, and rotate it over to the left. This manipulation brings the main branches of the coronary artery



*Fig. 240.—PARTIAL GASTRECTOMY. THE OPENING THROUGH WHICH THE RECTUS TUBE WAS INTRODUCED INTO THE STOMACH HAS BEEN CLOSED WITH A PURSE-STRING SUTURE, PRODUCING A PUCKERED EFFECT. THE STOMACH HAS BEEN DRAWN OVER THE LEFT COSTAL MARGIN, AND THE CORONARY ARTERY IS BEING DIVIDED BETWEEN TWO HEMOSTATS NEAR ITS ORIGIN FROM THE CELIAC AXIS. (After Balfour, modified.)*

readily to view, so that a ligature can be applied exactly where it is required; but this procedure does not invariably ensure that the artery is ligatured near its origin.

An aneurysm needle threaded with a length of No. 2 20-day chromic catgut is then insinuated between the border of the lesser curvature and the artery, the surgeon's left index finger being used to steady and guide the point through the lax tissues (fig. 241). The artery is trebly ligated and divided between the lower ligatures.

Another plan is to grasp the remaining portion of the gastro-hepatic

omentum, high up on the lesser curvature, between the index finger and the thumb of the left hand, and with the aid of the right thumb to work a passage through between the lesser curvature and the artery itself, until the index finger of the left hand projects through the tissues. Through this rent a hæmostat is passed to act as a guide for

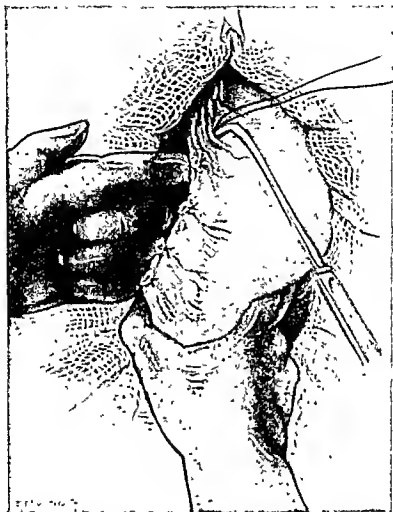


Fig. 241.—PARTIAL GASTRECTOMY. A METHOD COMMONLY EMPLOYED IN LIGATING THE CORONARY ARTERY.  
(After Kirchner, modified.)

an aneurysm needle which is threaded with a strong ligature. After the artery has been tied in three separate places it is divided between the middle and lower ligatures.

The method I usually employ in tying this artery is to draw the stomach firmly over the left costal margin so as to put the artery on the stretch. A strand of No. 2 chromic catgut, threaded on an aneurysm needle, is then passed around it, near its origin, and firmly tied in two places. The artery is then divided between the ligatures.

The distal end of the coronary artery is then seized with forceps and, together with the fatty tissues and glands in the remaining portion of the gastro-hepatic omentum, it is dissected downwards for a short distance (fig. 242). In doing this a raw surface will result

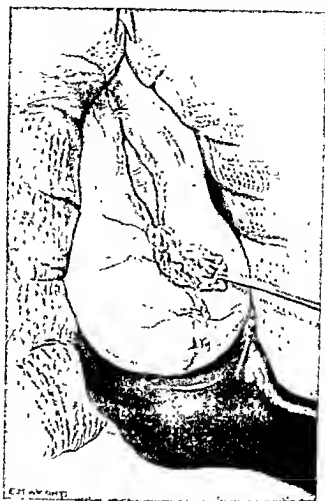


Fig. 242.—PARTIAL GASTRECTOMY. CORONARY ARTERY TIED NEAR ITS ORIGIN. THE GLANDS AND FATTY TISSUES BETWEEN THE TWO LAYERS OF THE GASTRO-HEPATIC OMENTUM ARE CAREFULLY DISSECTED DOWNWARDS ALONG THE LESSER CURVATURE.

on the lesser curvature from the stripping of the peritoneum from this region. The stripping downwards of this sero-fatty layer with its contained glands is an important step in the operation as infected tissues are often included in and secured with the stripped portion. The process also facilitates the introduction of sutures in the region of the lesser curvature after the stomach has been cut adrift. The raw surface is peritonised with a number of closely-applied interrupted sutures before proceeding with the further steps of the operation (fig. 243).

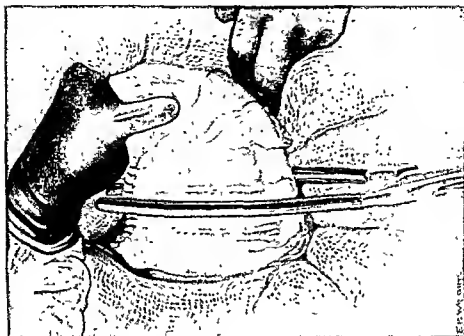


Fig. 234.—PARTIAL GASTRECTOMY. THE STOMACH IS DRAWN OVER TO THE LEFT AND THE FIRST STOMACH CLAMP IS ABOUT TO BE APPLIED.

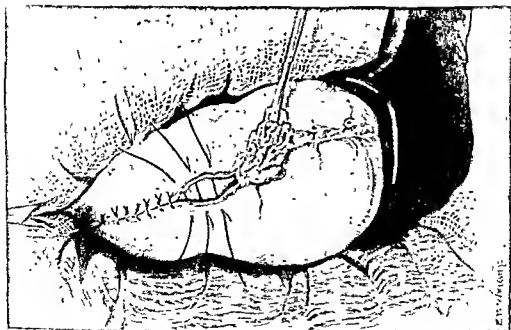


Fig. 243.—PARTIAL GASTRECTOMY. THE CLAVUS AND FATTY TISSUES BETWEEN THE TWO LAYERS OF THE GASTRO-HEPATIC OMENTUM ARE DISSECTED DOWNWARDS, AND THE RESULTING RAW SURFACE ON THE LESSER CURVATURE IS PROTECTED BY A SERIES OF INTERRUPTED SUTURES.

A Sherren clamp is now applied across the whole breadth of the stomach from the greater to the lesser curvature, as high up as possible (fig. 244). The transverse colon is lifted out of the wound and gently pulled upwards to make the mesocolon taut. The fingers of the right hand are passed along the mesocolon towards the left of the spine, and

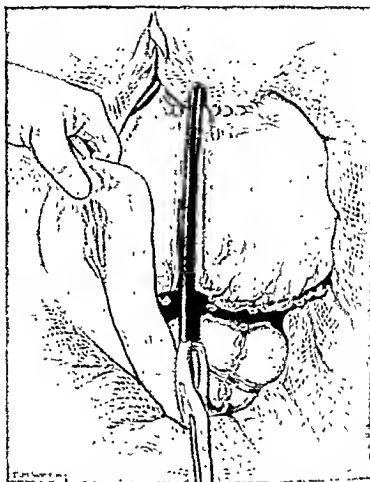


FIG. 245.—PARTIAL GASTRECTOMY. THE FIRST STOMACH CLAMP IS POSITIONED. THE PROXIMAL JEJUNAL LOOP IS BROUGHT FROM BENEATH THE TRANSVERSE COLON. THE PORTION OF JEJUNUM SELECTED FOR THE PURPOSE OF ANASTOMOSIS SHOULD BE ABOUT 4-6 INCHES FROM THE DUODENO-JEJUNAL FLEXURE.

the duodeno-jejunal flexure is identified. The first loop of the jejunum is brought out through the wound in front of the transverse colon, which is then replaced into the abdominal cavity (fig. 245). The proximal jejunum is laid from left to right against the stomach, and the portion selected for the anastomosis is picked up with two pairs of Allis forceps and put on the stretch, this portion of the jejunum being then embraced by a Sherren clamp (fig. 246). The distance between the duodeno-

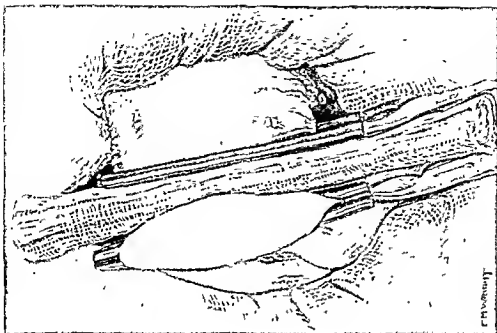


Fig. 247.—PARTIAL GASTRECTOMY. A ROLL OF GAUZE IS PLACED IN BETWEEN AND OFF TO THE TWO LAMINAE TO PREVENT CONTAMINATION OF THE PERITONEAL CAVITY.

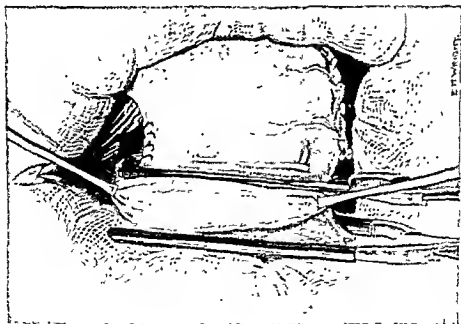


Fig. 248.—PARTIAL GASTRECTOMY. THE FIRST STOMACH CLAMP IN POSITION. A SECOND CLAMP IS BEING APPLIED TO THE JEJUNUM.



jejunal flexure and the proximal part of the anastomosis is only 4 inches, usually not more than 4 inches.

A long roll of gauze is then placed longitudinally in between and deep to the two clamps to prevent contamination of the peritoneal cavity by leakage of gastric or intestinal contents (fig. 247). The two clamps are laid side by side so that the greater curvature of the stomach lies against the proximal end of the clamped jejunum and the lesser curvature against the distal end (fig. 248).

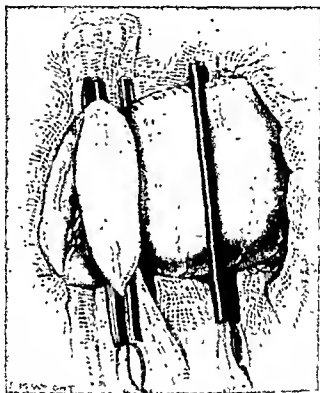


Fig. 248.—PARTIAL GASTRECTOMY. THE APPLICATION OF THE SECOND STOMACH CLAMP. THE PARTS ARE NOW READY FOR ANASTOMOSIS.

The first row of sutures is now introduced as a continuous sero-muscular suture of fine silk, the individual stitches being placed close to one another—not more than one-eighth of an inch apart. When the suture reaches the lesser curvature it is locked (fig. 249). The posterior wall of the stomach, about  $\frac{1}{2}$  inch above the line of suture, is incised from the greater to the lesser curvature, through the serous and muscular coats, down to the mucous membrane (fig. 250). The stomach is turned over to the right and the incision is continued on the anterior wall.

If there is no undue tension on the stomach it may now be cut

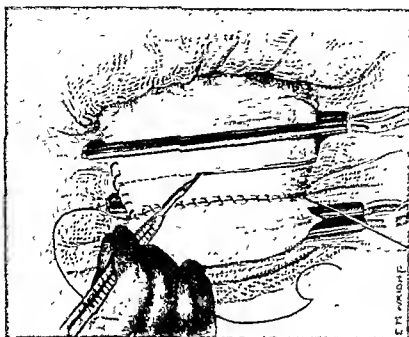


Fig. 250. — PARTIAL GASTRECTOMY. THE LINE OF INCISION INTO THE STOMACH. THE KNIFE ONLY CUTS THROUGH THE SERO-MUSCULAR LAYER. AT A LATER STAGE THE MUCOUS MEMBRANE IS PICKED UP AND DIVIDED WITH SCISSORS OR A CAUTERY.

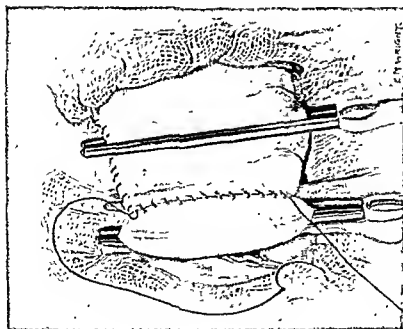


Fig. 249. — PARTIAL GASTRECTOMY. THE POSTERIOR SERO-MUSCULAR STITCH HAS BEEN INSERTED. WHEN THE SUTURE REACHES THE LOWER CURVATURE IT IS LOCKED.

through and removed (fig. 251), but if there is tension the organ is not removed until the posterior through-and-through suture has been inserted and is about to turn the corner at the lesser curvature. In the latter case a second gastric clamp should be applied to the stomach about  $\frac{1}{2}$  inch distal to the proposed line of transection.

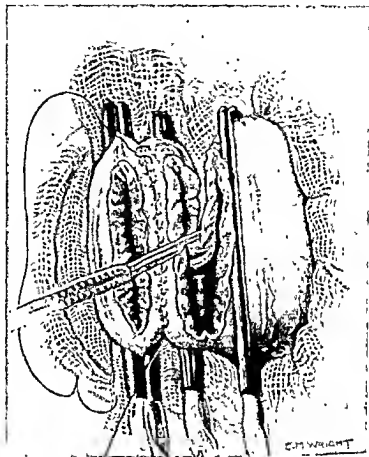


Fig 251 —PARTIAL GASTRECTOMY. THE POSTERIOR SERO-MUSCULAR STITCH HAS BEEN INTRODUCED, AND IS LOCKED WHEN IT REACHES THE LESSER CURVATURE. IF THERE IS NO TENSION ON THE STOMACH IT IS CUT AWAY IMMEDIATELY, AS SHOWN. THE OPERATION THEN PROCEEDS AS IN GASTRO-JEJUNOSTOMY.

The mucous membrane which bulges through the incision in the posterior wall of the stomach is picked up with a pair of dissecting forceps and incised, either with scissors or with a cautery, from the greater to the lesser curvature (fig. 252). The jejunum is opened along the entire length of the clamped loop, about  $\frac{1}{2}$  inch above the first row of sutures (fig. 253).

The contents of the gastric and jejunal pouches are then mopped up and the parts are cleansed with small gauze swabs soaked in saline.

The inner continuous through-and-through suture of No. 0 or No. 00

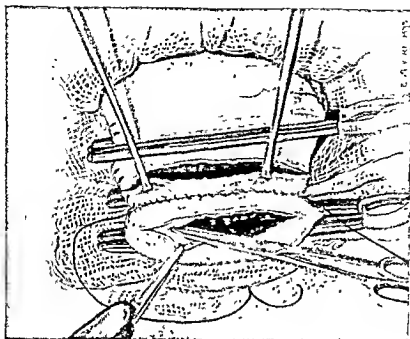


Fig. 273.—PARTIAL GASTRECTOMY. THE STOMACH HAS BEEN OPENED POSTERIORLY AND THE MUCOUS MEMBRANE OF THE JEJUNUM IS BEING DIVIDED WITH SCISSORS.

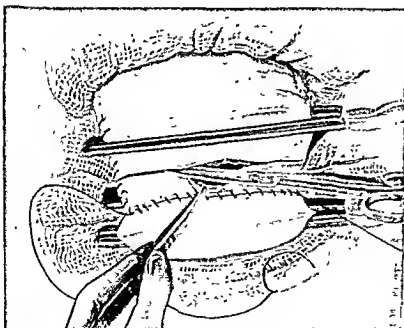


Fig. 265.—PARTIAL GASTRECTOMY. DIVISION OF THE STOMACH WALL POSTERIORLY. THE MUCOSA IS BEING DIVIDED WITH SCISSORS.

20-day chromic catgut is then introduced (figs. 254 and 255). It commences at the greater curvature, picking up all the coats of the stomach and jejunum, and is continued completely round the anastomotic opening, the individual stitches being placed not more than  $\frac{1}{8}$  inch apart. When it turns the corner of the lesser curvature it approximates and inverts the anterior margins of the stomach and jejunum. This inversion

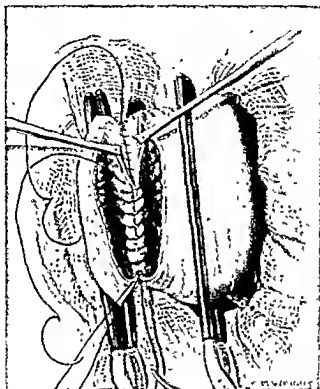


Fig. 254.—PARTIAL GASTRECTOMY. THE FIRST SUTURE IS COMPLETED, AND THE SECOND, A THROUGH AND THROUGH, IS BEING INTRODUCED AS IN THE OPERATION OF GASTRO-JEJUNOSTOMY. WHEN THIS LATTER SUTURE REACHES THE LESSER CURVATURE THE STOMACH IS CUT THROUGH AND REMOVED.

is accomplished by drawing the suture tight as it emerges from the jejunal mucosa, slight pressure being applied with the thumb and finger on the wound just behind the stitch.

When the suture reaches the point where it started it is knotted to that portion which was left long. The first sero-muscular suture is then picked up and continued anteriorly as a continuous Lembert suture which invaginates the first suture line. A few interrupted mattress sutures are placed here and there, wherever the suture line appears to require extra support (fig. 256).

When the through-and-through suture reaches the lesser curvature

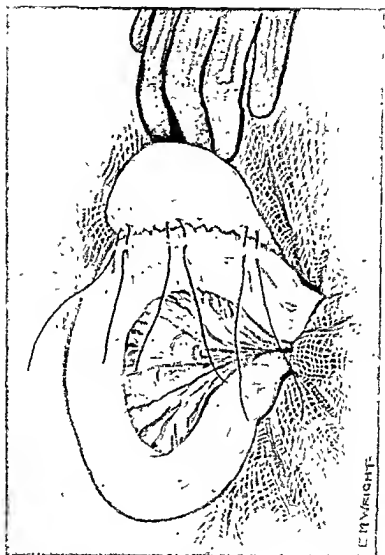


Fig. 254.—PARTIAL GASTRECTOMY. THE ANASTOMOSIS IS COMPLETED. A FEW HALSTED SUTURES ARE INSERTED TO RELIEVE TENSION AND TO REINFORCE THE SUTURE LINE.

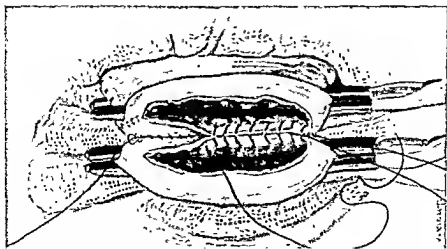


Fig. 255.—PARTIAL GASTRECTOMY. THE CONTINUOUS SUTURE LINE IS BEING INSERTED.

it may be made to proceed as a running stitch, uniting *only* the anterior margins of the mucous membrane of the stomach and jejunum (fig. 257). The anterior sero-muscular margins are approximated with a continuous stitch which commences at the greater

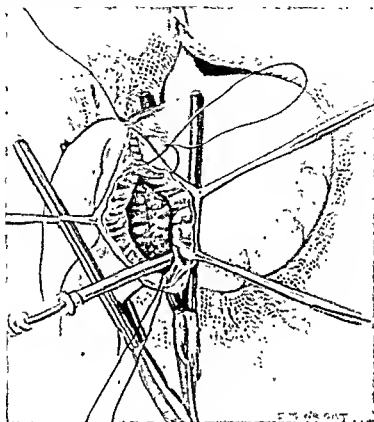


Fig. 257.—PARTIAL GASTRECTOMY. WHEN THE POSTERIOR THROUGH-AND-THROUGH SUTURE REACHES THE LESSER CURVATURE, IT IS USED FOR APPROXIMATING THE EDGES OF THE GROSS MEMBRANES OF THE STOMACH AND JEJUNUM UNTIL IT ARRIVES AT THE LOWEST POINT OF THE GREATER CURVATURE WHERE ITS END IS TIED TO THE LONG END OF THE ORIGINAL THROUGH-AND-THROUGH SUTURE. THE ILLUSTRATION SHOWS THE STEP IN THE OPERATION WHERE THE CLAMPS ARE TEMPORARILY LOOSENED TO INSPECT THE SUTURE LINE FOR POSSIBLE BLEEDING. AT THE SAME TIME A SUCTION TUBE IS INTRODUCED INTO THE REMAINING PORTION OF THE STOMACH TO REMOVE ANY ACCUMULATED SECRETIONS.

(After Balfour, modified.)

curvature and finishes at the lesser curvature. The anterior suture line is further buried by a right-angle Cushing stitch as shown in figure 258.

Finally, before the abdomen is closed, the anastomosis is inspected and a general survey is made of the operation field. If there is no bleeding and all appears well, the great omentum is lifted out of the abdomen, turned upside down, and folded in such a way that it covers the suture line and prevents it from adhering to the anterior abdominal

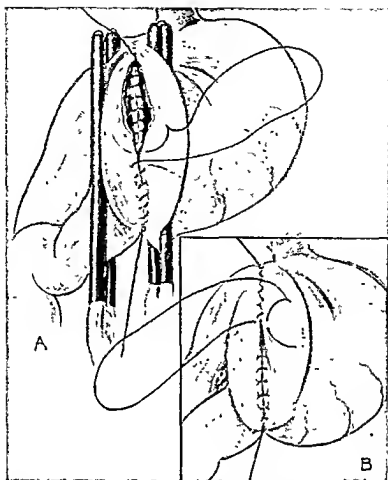


Fig. 258.—PARTIAL GASTRECTOMY. A. THE ANTERIOR THROUGH-AND-THROUGH SERO-MUSCULAR SUTURE IS BEING INTRODUCED. B. THE ANASTOMOSIS IS COMPLETED BY INVAGINATING THE SUTURE LINE BY A CUSHING STITCH.

wall, and also smoothes over any rough places in the bed of the stomach.

The abdominal wound is closed in layers. A continuous stitch of doubled No. 1 or No. 2 chromic catgut is used for the peritoneum, interrupted catgut stitches and a single continuous catgut suture for



1/2 SCALE. KIBA CLIP INSTRUMENTS



Fig. 259.—KIBA CLIP INSTRUMENTS.



the anterior sheath of the rectus muscle, while the skin margins are approximated with fine silkworm-gut sutures and Michel or Kifa clips (fig. 259). A few tension sutures of stout silkworm-gut are also occasionally used.

When there is a growth in the body of the stomach or in the region of the middle third of the greater curvature, the technique is sometimes varied, the operation being commenced by mobilising the first part of the duodenum, dividing the gut, and invaginating the duodenal stump before proceeding with any of the further steps of the operation. By this plan a piece of the gastro-hepatic omentum above the first part of the duodenum is selected and divided between ligatures. The index



Fig. 261.—PARTIAL GASTRECTOMY. THE METHOD OF MOBILISING THE FIRST PART OF THE DUODENUM PRIOR TO THE APPLICATION OF THE CLIPPING CLAMPS.  
(After Balfour, modified.)

finger of the left hand is introduced through this opening, passes behind the duodenum, and pushes forward the great omentum. Here an avascular portion close to the gut is opened with a hæmostat which is passed through it, behind the duodenum, and brought out through the opening in the lesser omentum (fig. 260). This hæmostat acts as a guide for the Payr clamps, one blade of each clamp being passed behind the duodenum. Both blades then clamp a portion of the first part of the duodenum, about 1 inch away from the pyloric outlet. During the introduction and application of the blades of these clamps the index finger of the left hand is passed through the top opening, and used to protect the underlying pancreas from damage and to guide the passage of the blades. The operation then proceeds in the manner already described under the Polya-Moynihan method (fig. 261).

(2) *The Polya-Balfour Operation.* (*Surg., Gynec., Obstet.*, xlv, 659-667, 1927.) The steps of this operation are in many ways similar to those of the method just described. There are, however, the following points of difference: The operation is commenced by mobilising the pylorus and the first portion of the duodenum. After the duodenum has been divided and its distal end is securely closed and invaginated, the operation proceeds as by the Polya-Moynihan method until a stage is reached when the jejunum is ready to be anastomosed to the stomach.

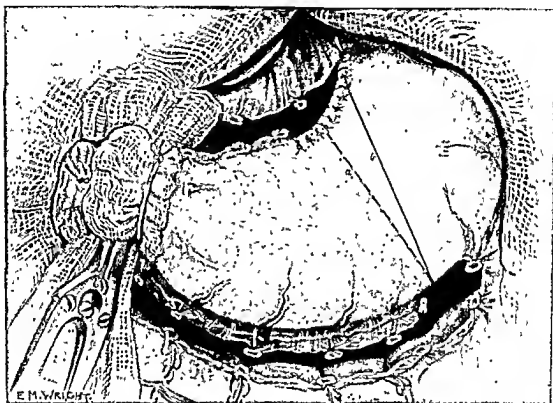


Fig. 261.—PARTIAL GASTRECTOMY. THE APPEARANCE OF THE STOMACH IMMEDIATELY BEFORE RESECTION.  
*a*—THE USUAL LINE OF SECTION THROUGH THE STOMACH IN CASES OF CHRONIC GASTRIC ULCER.  
*b*—THE LINE OF SECTION IN CASES OF CANCER OF THE STOMACH.

Here a *long* loop of jejunum is used instead of a short one. Here, too, the loop is taken from *right to left* across the stomach, so that the proximal part of the clamped jejunum lies against the lesser curvature and the distal part against the greater curvature. The distance between the duodeno-jejunal flexure and the uppermost point of the anastomosis may be as much as 12 inches or more.

The anastomosis between the jejunum and the stomach proceeds as in gastro-jejunostomy, but the operation is completed by making a small entero-anastomosis, with a stoma not exceeding 1 inch, between the proximal and the distal loops of the jejunum in order to prevent

retention of pancreatic juice and bile in the long proximal limb. Balfour considers that this is an important step in the operation and one which diminishes the likelihood of any untoward after-symptoms. When undertaken in cases of cancer of the stomach subsequent jejunal ulceration has never been known to occur.

(3) *Finsterer's Operation.* The abdomen is opened through a mid-line incision, and after conducting the abdominal exploration and deciding that resection of the stomach is feasible, the first step in the operation

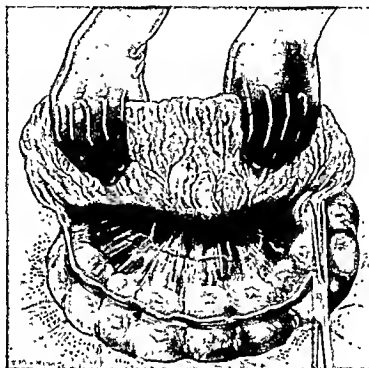


Fig 262.—FINSTERER'S OPERATION. STRIPPING THE OMENTUM FROM THE COLON.

consists in freeing the great omentum from the transverse colon. This is carried out by holding the omentum and colon apart and running a knife along the bloodless plane between them close to the wall of the colon. Alternatively, scissors may be used to snip the tissues close to the gut to effect this separation (fig. 262).

"The old plane of physiological adhesion being opened up, it is a simple matter to separate it down to the posterior abdominal wall with a gauze swab, when the faecal condition is re-established—above the posterior mesogastrium with the omentum, below the transverse mesocolon and colon. If the correct watershed is opened . . . there are no bleeding points. The only ligatures required are at the two

ends of the gastro-epiploic arch, one at the origin of the right vessel from the gastro-duodenal, and the other near the spleen." (Ogilvie, *B.M.J.*, p. 457, March 9, 1935.)

After the left and right gastro-epiploic arteries have been tied near their origins, the leash of blood-vessels which constitutes the pyloric artery is isolated, ligatured, and divided close to the upper border of the pylorus and duodenum. The pylorus and the first portion of the duodenum are carefully mobilised and the numerous anomalous blood-vessels springing from the head of the pancreas are individually picked up and ligatured. The duodenum is then divided and its distal end closed at a point  $\frac{3}{4}$  inch beyond the pylorus. This may be effected by one of three methods :

(a) By means of a Friedrich-Petz clamp, whereby the gut is severed through the crushed groove between the two rows of clips and the distal end invaginated with a series of closely-applied Lembert sutures.

(b) By crushing the gut with an enterotribe, tying a stout ligature around the groove, and, after dividing the gut, invaginating the distal closed end of the duodenum with two purse-string sutures.

(c) By dividing the duodenum between two Payr clamps and effecting the duodenal occlusion by suturing over the clamp with a right-angle continuous suture which is drawn tight as the clamp is removed, as depicted in figures 232, 233, 234 and 235.

The fan-shaped gastro-hepatic omentum is then divided as close to the liver as possible, and the coronary or left gastric artery is tied. Ogilvie lays particular stress upon the necessity of tying this artery as close as possible to its origin from the coeliac axis. He writes :

"The left gastric is not only the main source of supply of the lesser curve, but it anchors the upper part of the stomach to the posterior abdominal wall, and it is surrounded by the lymphatic glands which drain this part. Ligature of this vessel near its origin is essential in cancer surgery, because only after such ligature is it possible to clear the lymphatics right up to the oesophageal opening in one piece with the stomach. High ligature is necessary on grounds of function as well, because after a Polya gastrectomy the remainder of the stomach must be free to lie in a new plane and move in a fresh axis. The normal stomach is slung like a hammock between its two openings ; the left gastric artery springs from the centre of this arc, and is never pulled upon. The resected stomach hangs like a pendulum, vertically from the oesophagus and the peritoneal ligaments round the oesophageal opening. If the left gastric artery retains any attachments

with the lesser curve it becomes an anchor, limiting movements. A good many cases of post-operative discomfort are, I believe, referable to such fixation. . . .

"The stomach is thrown upwards towards the left shoulder of the patient, the main artery is put on the stretch: a ligature can be passed round it, slid down, and tied close to the cœliac axis. After such proximal ligature the glands and fatty tissue can be cut away, in operations for cancer, right up to the cardiac orifice, and stripped downwards, leaving the lesser curve bare to the point of intended section." (Fig. 263.)

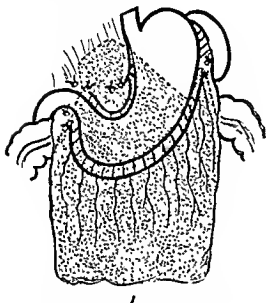


Fig. 263.—FINSTERLIN'S OPERATION. THE SHADED AREA DEPICTS THE PARTS TO BE REMOVED IN PARTIAL GASTRECTOMY FOR CARCINOMA.  
(After Opitz, B.M.J.)

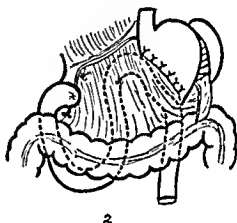


Fig. 264.—FINSTERLIN'S OPERATION OF PARTIAL GASTRECTOMY FOR CANCER OF THE STOMACH COMPLETED. NOTE THAT THE ANASTOMOSIS LIES IN THE INFRA-COLIC COMPARTMENT.  
(After Opitz, B.M.J.)

The stomach is now free and mobile, attached only by the œsophagus and the veil-like gastro-splenic omentum, and is ready for the application of the Petz clamp. This should be applied obliquely from a point on the lesser curvature just below the œsophagus, across the body of the stomach, to a point on the greater curvature some 2 inches or so below where the left gastro-epiploic artery was tied. The Petz clamp is forced home and the clips are introduced.

The stomach is then divided by a Post electric cautery between the rows of clips, and the resected portion, together with its attached omenta and glands, is removed in one piece.

The right or upper half of the cut surface of the stomach is then closed by two or more rows of interrupted Lembert sutures, the infolded

line reaching almost to the œsophagus. The anastomosis of the lower half of the stomach to the proximal jejunum is carried out as follows: An opening is made in the mesocolon, well to the left of the middle colic artery, and through this a fairly long loop of proximal jejunum is drawn into the supra-colic compartment. The left-hand side opening in the mesocolon is sewn to the posterior surface of the stomach before the anastomosis is started; the right-hand leaf is sutured to the anterior wall of the stomach after the anastomosis is completed, thereby leaving the whole of the anastomosis in the infra-colic compartment (fig. 264).

The portion of jejunum selected for the anastomosis should be some 6-8 inches from the duodeno-jejunal flexure. This loop is clamped and after the anastomosis is completed (as in the operation of gastro-jejunostomy) the proximal jejunum is then stitched to the closed (upper) part of the stomach, reinforcing the suture line and interposing a thick valve of gastric and jejunal wall between the outlet of the stomach and the proximal loop (fig. 265).

The jejunum now lies almost vertically and its efferent limb runs downwards in direct continuation with the mouth of the funnel-shaped stomach.

(4) *Lahey's Operation.* When undertaking a posterior Polya anastomosis I have found Lahey's method of dealing with the proximal jejunal loop to be a most satisfactory procedure, and I am grateful to him for his kind permission to quote his description of the operation as published in *Surgery, Gynecology, and Obstetrics*, August, 1933, Vol. lvii, 227-230.

"The following plan of management in posterior Polya anastomosis of the proximal loop of the jejunum to the gastric stump after partial gastrectomy has proved useful and satisfactory in our hands. Others very probably have employed the same plan, although I have not seen it used or described. While I am not interested in claiming priority concerning this plan, I do wish to describe it because I trust that it may prove as useful to other surgeons as it has in our clinic.

Following partial gastrectomy, connection between the small intestine and the stomach may be re-established by a variety of methods, one of the most popular of which is the end-to-side anastomosis of the jejunum to the open end of the resected stomach, an operation which, in this country, is called the Polya operation.

Various plans and modifications of this procedure have been practised. Donald C. Balfour has suggested that a loop of jejunum be brought up over the transverse colon and anastomosed to the cut end of the stomach—the ante-colic Polya anastomosis. This plan has

proved very useful, but it has two possible drawbacks. Occasionally, in attempting to carry out this step, I have found the length of the jejunal mesentery so short that when it is brought up at a reasonable level over the transverse colon to reach the cut end of the stomach, it produces such pressure upon the transverse colon that, were distension to occur in that structure, there would be danger of obstruction. It

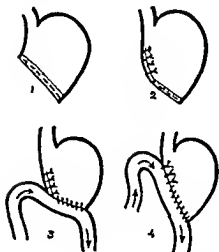


Fig 263.—FINSTERLIN'S OPERATION. CONNECTION OF THE STOMA AND VALVE. 1=SMALL GASTRIC POUCH, CLOSED BY ONE ROW OF CLIPS. 2=THE UPPER HALF OF THE STOMACH INVAGINATED WITH INTERRUPTED LEMBERT SUTURES. 3=AFTER SECURING THE LOWER HALF OF THE OPENED GROOVE TOGETHER WITH ITS CLIPS, THE ANASTOMOSIS BETWEEN THE STOMACH AND JEJUNUM IS PERFORMED AS IN THE OPERATION OF GASTRO-JEJUNOSTOMY. 4=PROXIMAL JEJUNUM ATTACHED TO THE CLOSED (UPPER) PART OF THE STOMACH, REINFORCING THE SUTURE LINE AND INTERPOSING A THICK VALVE OF GASTRIC AND JEJUNAL WALL BETWEEN THE OUTLET OF THE STOMACH AND THE PROXIMAL LOOP.

(After Oplive, modified, R.M.J.)

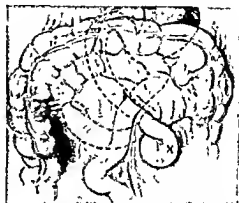


Fig 264.—DRAWING SHOWING THE ANGULATION OF THE JEJUNUM IF THE PROXIMAL LOOP IS CARRIED UP THROUGH THE MESOCOLON AND THE LIGAMENT OF TREITZ IS NOT SEVERED; ALSO THE THICKENED BARRELLED EFFECT OF THE PROXIMAL AND DISTAL LOOPS OF JEJUNUM AS THEY PASS THROUGH THE RENT IN THE MESOCOLON. EVEN IF THE MESOCOLON IS SUTURED TO THE GASTRIC STUMP, AS IS FREQUENTLY DONE, CONSIDERABLE ANGULATION OF THE PROXIMAL JEJUNUM RESULTS. NOTE THE LIGAMENT OF TREITZ, WHICH IN THE PLAN HERE DESCRIBED IS TO BE INCISED, THUS PERMITTING THE DISPLACEMENT OF THIS PROXIMAL LOOP OF JEJUNUM THROUGH THE MESOCOLON SO THAT IT RAYS ABOVE IT. THEN CUT A SINGLE LOOP OF JEJUNUM, THE DISTAL ONE, EMERGES THROUGH THE RENT IN THE MESOCOLON. NOTE THE LEVEL OF THE PROXIMAL JEJUNUM MARKED X IN ORDER THAT IT MAY BE COMPARED WITH THIS POINT WHEN TRANSPLANTATION HAS BEEN DONE ABOVE THE TRANSVERSE MESOCOLON BY THE PLAN DESCRIBED IN THE TEXT.

(Drawn from "Surg., Gynec., and Obstet.," by courtesy of Dr. Frank H. Lahey.)

is true, however, that in a large percentage of the cases, the jejunal loop can readily be approximated to the stomach without difficulty, so that this plan has been accepted as very useful. The second possible drawback is, in order that the jejunal loop will reach over the transverse colon, a long jejunal loop must be used, thus resulting in the dumping of gastric contents into a relatively low segment of the

jejunum. In all probability this criticism is not serious, but, other things being equal, an endeavour should be made to approximate the stomach to the small bowel as near to the duodenum as possible. It must be assumed that the farther from the duodenum the point of anastomosis between the stomach and jejunum be made, the less adapted that segment of bowel will be to receive gastric contents and the greater will be the likelihood of recurrent gastro-jejunal ulcer at the suture line.

I have always been inclined to use the posterior Polya type of anastomosis, passing the loop of jejunum through the transverse mesocolon, thus permitting the anastomosis of the jejunum to the cut end of the stomach at a level somewhat closer to the duodenum. In this manner the occasional difficulty with a short mesentery to the jejunum is avoided and approximation to the cut end of the stomach is made easy. I do not describe the plan here submitted as an argument against the ante-colic Polya plan of anastomosis but rather that it may be available for those who are interested and wish to employ the posterior Polya type of procedure.

In the posterior Polya type of anastomosis in partial gastrectomy, one of two plans may be employed in dealing with the proximal and distal loops of jejunum. After the end-to-side anastomosis between the jejunum and stomach, one plan is to suture the slit in the transverse mesocolon anteriorly and posteriorly to the stump of the stomach itself, thus placing the proximal and distal loops of jejunum entirely below the transverse mesocolon in the general peritoneal cavity. A disadvantage of this method is that if the gastric resection be high and the gastric stump short, it will be difficult to bring the transverse mesocolon up high enough so that it can be sutured to the gastric stump without distortion of the transverse colon. In some very high gastric resections, in our hands, it has proved impossible.

The other plan customarily employed is to bring both loops of jejunum up through the transverse mesocolon, to permit the stomach to retract to its natural height, and then to suture the rent in the transverse mesocolon about the double-harrelled loop of proximal and distal jejunum (fig. 266). The disadvantages of this procedure are that the jejunum must emerge from its retro-peritoneal position in the jejunal fossa, the afferent loop must ascend through the slit in the transverse mesocolon, and the efferent loop must also emerge through the transverse mesocolon, thus producing a double-harrelled effect with considerable angulation of the loop of proximal jejunum. Another undesirable factor in the plan is that it is not possible satisfactorily and



accurately to close the opening in the transverse mesocolon when two loops of jejunum pass through it.

By the plan we have employed and here suggest, an opening is made in the transverse mesocolon usually just to the left of the root of the ligament of Treitz. In some measure the site of the opening in the mesocolon must depend upon the location of the middle colic artery. Through this aperture there is passed a loop of jejunum which is

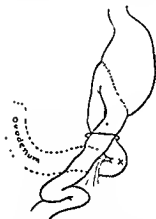


Fig. 207.—THIS DIAGRAMMATIC FIGURE DEMONSTRATES THAT WHEN AN INSUFFICIENT AMOUNT OF PROXIMAL JEJUNUM IS UTILIZED AND THE STOMACH IS PERMITTED TO DROP BACK INTO THE LEFT UPPER QUADRANT, TRACTION OCCURS ON THE FUTURE LINE DUE TO THE SHORT PROXIMAL JEJUNUM. THE DISADVANTAGE OF TOO SHORT A DISTAL LOOP OF JEJUNUM IS LIKEWISE ILLUSTRATED. NOTE THE LIGAMENT OF TREITZ AND THAT, BY INCISION OF THIS LIGAMENT, THE PROXIMAL LOOP OF JEJUNUM MAY BE TRANSFERRED ABOVE THE

MESOCOLON

(Lahay)

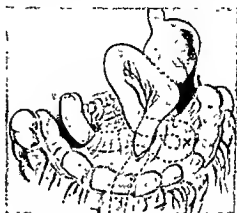


Fig. 208.—THIS FIGURE IS THE SAME AS FIG. 207, EXCEPT THAT THE RELATIONS ABOVE THE MESOCOLON WITH THE TRANSVERSE COLON TURNED DOWN ARE SEEN. THE ANGULATED PROXIMAL LOOP OF JEJUNUM IS SHOWN BELOW THE MESOCOLON IN DOTTED LINES. NOTE AGAIN THE POINT MARKED X IN THE PROXIMAL JEJUNUM BENEATH THE TRANSVERSE MESOCOLON.

(Drawn from "Surg., Gynec., and Obstet.," by courtesy of Dr. Frank H. Lahay)

sufficiently long so that when the stump of the stomach retracts upward into the left hypochondrium, there will be no undue traction and tension upon the point where the proximal loop of jejunum is attached to the cut end of the stomach at the point which marks the lesser curvature. I wish particularly to warn operators who are not familiar with this step that, in order to avoid tension as here described, the proximal loop of jejunum must always be a little longer than at first seemed necessary. This is extremely important, since, if the end-to-side anastomosis has already been made and following release of traction upon the stomach and the ascent of that organ, there is tension on the upper angle of

the suture line due to too short a proximal jejunal loop, a dangerous and distressing situation arises (figs. 267 and 268).

Having satisfactorily completed the posterior Polya anastomosis, the plan which I have employed and wish to describe is as follows :

The ligament of Treitz is cut from its lowest insertion into the jejunum, up to its origin in the mesenteric root. This permits of mobilisation of the uppermost part of the jejunum, so that the proximal

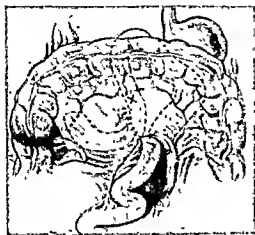


Fig. 260.—AFTER INCISION OF THE LIGAMENT OF TREITZ AND TRANSPLANTATION OF THE PROXIMAL LOOP OF JEJUNUM, THE SINGLE LOOP OF DISTAL JEJUNUM EMERGES FROM THE OPENING IN THE TRANSVERSE MESOCOLON. THE MESOCOLON HAS BEEN SUTURED ABOVE THE SINGLE LOOP OF JEJUNUM. THE TRANSVERSE COLON IS TURNED UP SO THAT THE PROXIMAL JEJUNUM IS SHOWN IN DOTTED LINES, INDICATING ITS POSITION ABOVE THE TRANSVERSE MESOCOLON. NOTE NOW THE POSITION OF X WELL ABOVE THE MESOCOLON AND ITS RELATION TO THE LEVEL OF THE JEJUNUM AT WHICH THE ANASTOMOSIS HAS BEEN MADE.

(Drawn from "Surg., Gynec., and Obstet.," by courtesy of Dr. Frank H. Lahey.)



Fig. 270.—THIS FIGURE IS THE SAME AS FIG. 260, EXCEPT THAT THE TRANSVERSE COLON HAS BEEN TURNED DOWN IN ORDER THAT THE PROXIMAL AND DISTAL LOOPS OF JEJUNUM MAY BE VIEWED FROM ABOVE THE MESOCOLON. NOTE THE RENT IN THE MESOCOLON SUTURED. ONE SIDE OF THIS RENT REPRESENTS THE INCISED LIGAMENT OF TREITZ. NOTE THE POINT X ON THE BOWEL NOW WELL ABOVE THE TRANSVERSE MESOCOLON. THE LACK OF AVULSION, AND THAT MORE THAN ENOUGH PROXIMAL JEJUNUM HAS BEEN UTILISED IN ORDER TO PREVENT TRACTION AND AVULSION WHEN THE STOMACH RETRACTS INTO THE LEFT HYPOCHONDRUM. NOTE ALSO THAT BUT A SINGLE LOOP OF JEJUNUM, THE DISTAL LOOP, PASSES THROUGH THE TRANSVERSE MESOCOLON.

(Drawn from "Surg., Gynec., and Obstet.," by courtesy of Dr. Frank H. Lahey.)

loop of jejunum now anastomosed to the stomach can be passed up through the slit made in the transverse mesocolon, and in this way the entire proximal loop of jejunum is brought above the mesocolon and is excluded from the greater general peritoneal cavity. While the true vascular root of the transverse colon is still above the junction of the jejunum with the duodenum, nevertheless there is less angulation of the proximal jejunum than when the proximal jejunal loop enters the greater peritoneal cavity at the jejunal fossa and is again passed upward out of the greater peritoneal cavity through a slit in the transverse

mesocolon. It has the additional advantage that now but a single segment of bowel, the distal jejunal loop, emerges through the transverse mesocolon (figs. 269 and 270). Snug suture of the slit in the transverse mesocolon about the single loop of bowel is thus possible and the danger of hernia through this slit is lessened.

The plan of the placing of the entire proximal loop of jejunum above the transverse mesocolon in a posterior Polya anastomosis has, if anything, not added to, but diminished, certain of the technical difficulties of this type of anastomosis after partial gastrectomy. In all of the cases in which it has been employed, the anastomosis has functioned well and any question of the need of entero-enterostomy between the proximal and distal loops of jejunum has been eliminated.

The plan submitted has proved valuable by actual repeated employment. It is not suggested that it be employed in preference to other methods but it is hoped that it may prove useful to those who like and employ the posterior Polya type of anastomosis after partial gastrectomy."

#### TOTAL GASTRECTOMY

This operation, in which the whole of the stomach is excised and the cut end of the œsophagus is anastomosed either to the cut end of the duodenum or to a loop of proximal jejunum, must be distinguished from that of sub-total gastrectomy in which a portion of the fundus and of the greater curvature is left behind. Although total gastrectomy was first performed by Conner in 1884, it was not until 1897 that Schlatter performed the first successful operation, after which the patient lived for 14 months. Paterson, in 1909, collected 27 cases of total gastrectomy for cancer of the stomach; of these 10 died as a result of the operation, and 17 recovered. Finney and Ricahoff, in an article which appeared in *Archives of Surgery* (p. 156, January, 1929), tabulated most of the cases which had been recorded up to that time. They were able to quote 62 cases from literature and added 5 of their own. Of these 67 cases there was a 53.8 per cent mortality, but there was a higher recovery with œsophago-jejunosomy than with œsophago-duodenostomy. Roeder, in a comprehensive article which appeared in *Annals of Surgery* (p. 221, August, 1933), reported 88 cases, including 3 of his own, with an operative mortality of 44. i.e. 50 per cent.

Such are the facts from literature, but what of the unrecorded cases?

It is not unreasonable to suppose that the unrecorded cases number

hundreds, and that the true mortality for this heroic operation is in the neighbourhood of 60 per cent.

Roeder considers that this operation is being performed more frequently owing to an increasing number of operators, safer anæsthesia, improvements in operative technique, a wiser choice of the patient to be operated upon, and more thorough pre-operative and post-operative treatment.

A knowledge that in most instances recurrences following partial gastrectomy are found in the region of the remaining portion of the stomach or in the regional lymph glands compels the surgeon to consider whether the performance of total gastrectomy instead of partial gastrectomy would have prevented a certain number of these recurrences.

Verbrugghen's interesting work on the intra-mural spread of carcinomatous cells shows quite clearly that if any attempt at radical cure is to be made, at least 4 cm. of apparently healthy stomach wall should be removed together with the primary growth, and in those instances where the growth is situated high up in the body of the stomach this can really only be achieved satisfactorily by total gastrectomy.

#### *Indications for Total Gastrectomy :*

- (1) In cases of localised leather-bottle stomach.
- (2) In cases of generalised leather-bottle stomach where the cardia appears to be uninvolved in growth.
- (3) In malignant growths of the fundus.
- (4) In carcinoma involving the upper reaches of the body of the stomach, and particularly the upper third of the lesser curvature, always provided that a healthy area of stomach between the uppermost limits of the growth and the œsophagus render the resection feasible, and that other factors are favourable for the performance of this extensive operation.

*Pre-Operative Treatment.* Much can be done to render some of these cases of cancer of the stomach on whom a total gastrectomy is contemplated, and many of whom are grave operative risks, fit for the ordeal of a severe and protracted operation. The following is a brief outline of the pre-operative measures usually advised :

- (1) Rest in bed for a few days.
- (2) Eradication of oral sepsis.

- (3) Sunlight treatment.
- (4) Gastric lavage.
- (5) A nutritious diet which includes milk, pre-digested foods, large quantities of glucose and other beverages rich in pepsin, hydrochloric acid, and vitamins.
- (6) The administration of intravenous and rectal salines.
- (7) Blood-transfusion.
- (8) Stimulation of hepatic and renal function by diathermy and other measures.

The main difficulties of the operation may be summarised as follows :

- (1) The great depth of the œsophagus, and its limited exposure even after reflection of the left lower costal cartilages.
- (2) The shortness of the sub-diaphragmatic section of the œsophagus.
- (3) The absence of a serous coat on the posterior surface of the œsophagus.
- (4) The friability of the walls of the œsophagus.
- (5) The tendency for the œsophagus to retract into the posterior mediastinum.

#### *Causes of Death following Total Gastrectomy.*

- (1) *Immediate.*
  - (a) Shock.
  - (b) Hæmorrhage.
  - (c) Peritonitis from leakage at the anastomotic line.
  - (d) Pulmonary complications.
- (2) *Delayed.*
  - (a) Anæmia.
  - (b) Recurrence of growth.

It will be seen from the above that at least three causes of post-operative death are due primarily to faulty operative technique, e.g. shock, hæmorrhage, and peritonitis, and that anæmia following operation should be prevented by the administration of pre-digested foods, hydrochloric acid, pepsin, liver and stomach extracts, and blood-supporting treatment. With improvements in technique the mortality will necessarily be decreased to a figure very much lower than it is at the present moment, i.e. about 50 per cent.

(1) *Moynihan's Operation of Total Gastrectomy—Method 1.*

I am indebted to Lord Moynihan for his kind permission to quote verbatim his classic description of the operation of total gastrectomy as it appears in his well-known work *Abdominal Operations* (pages 409–416, Vol. i, 4th edition, Saunders, 1926). He describes in detail the operation he performed upon a patient aged 43 who was suffering from malignant leather-bottle stomach.

He writes :

“The abdomen was opened in the middle line by an incision which was about 3 inches in length, sufficient to allow of exploration, but which was increased subsequently to a length of 8 inches. At the outset there was a very serious difficulty in exposing the stomach. The patient was a man who had been stout, but who had lost weight rapidly; the anterior abdominal wall therefore shelved downwards from the elevated costal margin in such manner as to make the upper part of the stomach appear to be at great depth from the surface. The patient, moreover, was not at all comfortable under the anæsthetic, and I had to wait a long time after opening the abdomen before I could proceed with the operation.

When the stomach was exposed it was seen to be small in size, with walls of great thickness and solidity. The whole organ, indeed, felt solid, resembling a very large uterus, having thick walls and an insignificant cavity within it. . . . The surface was smooth, white, and opaque; there were no adhesions and but few obviously enlarged glands along the curvatures. Towards the cardiac end the stomach was larger than elsewhere, so that the organ had something of the shape of a Florence flask; the larger part, however, was still very much smaller than the normal. This being the condition of the stomach it was at once evident that the performance of gastro-enterostomy was impossible, for there was not a sufficient cavity in the stomach to admit of any anastomosis being made. The alternative procedures were complete gastrectomy, and either jejunostomy or duodenostomy; after some deliberation I decided in favour of the former, and I proceeded at once to remove the whole stomach. It was at this point that the abdominal incision was enlarged. Hot moist swabs in two layers were then packed into the abdomen in the usual manner to isolate the field of operation. The stomach was now depressed as far as possible by forcible traction by an assistant, and two long clips were applied to the coronary artery at its origin from the celiac axis. The artery was divided between the clips and its proximal end was ligatured. The upper and lower coronary

groups of glands were detached downwards towards the stomach by gauze stripping, and the cardiac end of the stomach was denuded by the same means. The gastro-hepatic omentum was divided after ligature as close up to the liver as possible until the upper border of the pylorus was reached. Here, by gauze stripping, the pyloric artery and the gastro-duodenal artery were exposed as they separately arose from the main hepatic trunk.

The pyloric artery was ligatured and divided, and the finger was then passed downwards behind the pylorus and made to present at the lower border of the duodenum, where an opening was made in the great omentum. Through this opening the blade of a clamp was passed upwards behind the duodenum to present above the pylorus. When this clamp was closed it lay about 1 inch beyond the pylorus, and on the stomach side of it there lay the subpyloric group of glands. A second clamp with rubber-covered blades was now applied distal to it and the duodenum was cut between them. A single strong catgut suture was then passed through the proximal part of the duodenum and round the clamp to prevent the clamp from slipping away. The distal end of the duodenum was then closed by a continuous catgut suture, taking all the coats, and by a double layer of Pagenstecher thread sutures above this. The clamp holding the proximal part of the duodenum was now covered with a gauze swab and was lifted well towards the left, exposing the gastro-duodenal artery more conspicuously. The artery was ligatured and divided. Along the whole length of the greater curvature the gastro-hepatic omentum was divided at a distance from the stomach of from 1 to 2 inches, so that all glands, including one or two dropped glands, were left attached to the stomach. The whole stomach was now free, for the gastro-hepatic omentum had been entirely divided, the duodenum was severed, and the gastro-colic omentum ligatured and cut free. The whole stomach hung pendulous from the œsophagus. At this point the anesthetist was asked to flex the patient's neck as much as possible, in the hope that this might enable the œsophagus to be pulled downwards a little more readily, and it seemed that this hope was fulfilled. The œsophagus was dragged upon with a fair degree of force until at least  $\frac{3}{4}$  of an inch of it was visible below the diaphragm.

The next step, and the most important and difficult of all, was the anastomosis of the œsophagus to the jejunum. The transverse mesocolon was already exposed on its upper surface in the wound; it was divided in an avascular area and the upper loop of the jejunum pulled through it. A point on this about 8 inches from the duodeno-jejunal

flexure was selected for the anastomosis. A piece of it about  $2\frac{1}{2}$  inches in length was laid transversely along a line immediately behind the œsophagus. As it lay there transversely, the right leaf posterior, its



Fig. 271.—TOTAL GASTRECTOMY. MOYNIHAN'S OPERATION. THE STOMACH IS DRAWN FORCIBLY UPWARDS OVER THE LEFT COSTAL MARGIN. THE HIGHEST JEJUNAL LOOP HAS BEEN BROUGHT UP THROUGH AN OPENING IN THE TRANSVERSE MESOCOLON AND IS BEING SUTURED BY INTERRUPTED SUTURES TO THE POSTERIOR WALL OF THE (ESOPHAGUS WHICH HAS BEEN FREED FROM THE DIAPHRAGM AND DRAWN FORWARDS.

A = Esophagus.

B = Transverse mesocolon.

C = Closed end of the duodenum.

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upper end was to the left, its lower to the right. The anastomosis was now begun by introducing eight light interrupted sutures between this portion of the jejunum and the œsophagus (fig. 271). The part of the circumference of the jejunum used was that on the surface which was



now posterior, and on this surface about  $\frac{3}{4}$  of an inch from the mesenteric attachment. As the sutures were introduced into the œsophagus this was made to present and was well exposed by a forcible and continuous downward traction upon the stomach. The stomach, wrapped in a hot gauze swab, was used, and most efficiently used, as a retractor, or rather as an instrument of traction, upon the slightly dilated

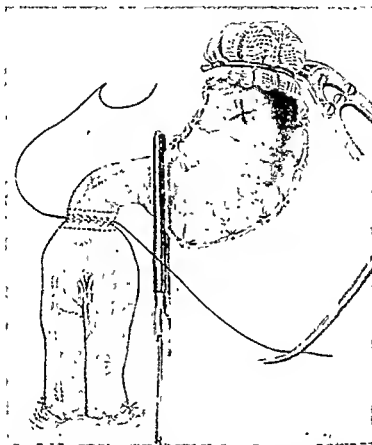
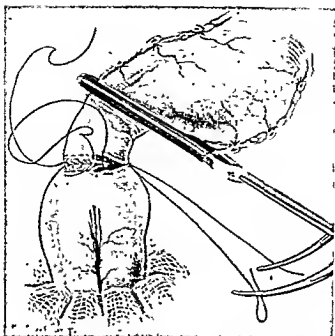
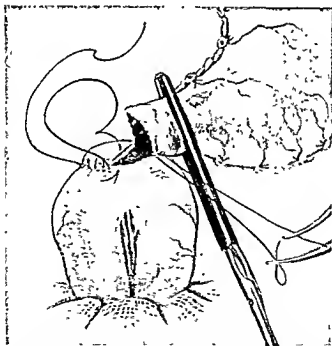


Fig 272.—TOTAL GASTRECTOMY. MOYNIHAN'S OPERATION. LINES OF INCISION INTO THE ŒSOPHAGUS AND JEJUNUM. THE POSTERIOR CONTINUOUS SERO-MUSCULAR SUTURE HAS BEEN INTRODUCED, BRINGING THE INTERRUPTED SUTURES

œsophagus. The help derived from this manœuvre was far greater than could be believed from a mere description. It converted what would have been an excessively difficult feat into one of comparatively easy accomplishment. Eight interrupted sutures then were introduced until the whole of the posterior half of the œsophagus was securely attached to the jejunum. In front of these a continuous suture was now introduced, exactly as in the operation of gastro-enterostomy, from left to right; the needle carrying this suture was then laid aside to be presently resumed (fig. 272). The attachment of the œsophagus to



*Fig. 273.*—TOTAL GASTRECTOMY. MOYNIHAN'S OPERATION. SUTURE OF THE MARGINS OF THE OPENINGS IN THE ESOPHAGUS AND JEJUNUM



*Fig. 274.*—TOTAL GASTRECTOMY. MOYNIHAN'S OPERATION. THE SUTURING ALMOST COMPLETE, THE STOMACH STILL BEING USED AS A TRACTOR.

the jejunum seemed now quite secure on this posterior aspect. In front of this continuous suture a small opening was made into the œsophagus and into the jejunum at the extreme left end of this attachment. A continuous through-and-through Pagenstecher thread suture was now begun and a few turns of the needle taken until the whole length of the small openings made had been united. These openings were then enlarged little by little from left to right, and as they were enlarged their cut edges were sutured by the same continuous stitch (fig. 273). This sequence of a small incision, a few stitches, slight enlargement of the incision, a few more stitches, was continued until the whole of the posterior part of the œsophagus was divided and sutured to the incision in the jejunum. Around the anterior wall of the œsophagus the same sequence was continued, the stitch being now changed to the 'loop on the mucosa' form. The result was that the stomach was retained as a tractor, drawing down the œsophagus until the last piece was severed, and at that moment the line of anastomosis was almost complete (fig. 274). Finally the outer continuous suture previously laid aside was resumed and continued round the anterior surface of the œsophagus and jejunum to its starting-point, where it was tied and cut short. The suture line was now complete. There were, it will be seen, eight interrupted posterior sutures, intended as anchor sutures, and the two continuous sutures, as in the usual operation of gastro-enterostomy.

A few anterior anchor sutures fixing the jejunum and the œsophagus to the diaphragm were taken and the main part of the operation was now complete. The great omentum was turned upwards over the operation area and the abdomen closed."

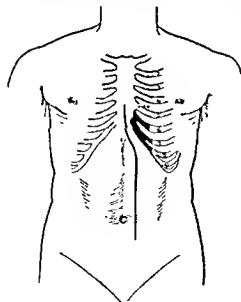
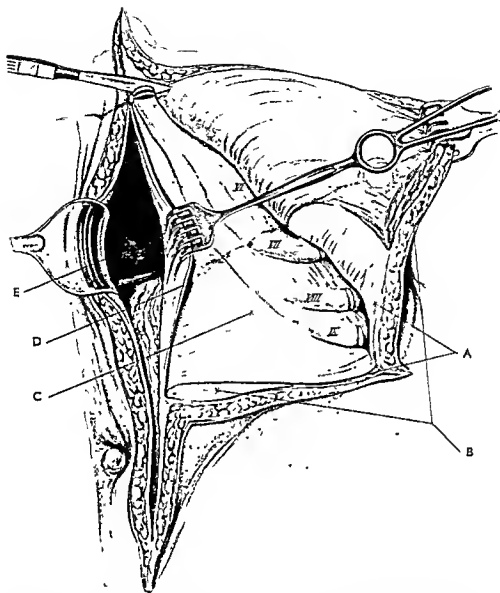


FIG. 275.—TOTAL GASTRECTOMY. INCISION. THE SHADED AREA INDICATES THE AMOUNT OF CARTILAGE MOBILISED. THIS MAY BE NECESSARY IN CERTAIN CASES WHERE EXPOSURE OF THE CARDIAC END OF THE STOMACH IS INADEQUATE.

## (2) Total Gastrectomy.—Method 2.

The abdomen is explored through a long left paramedian incision which commences over the lower portion of the sternum and proceeds downwards to the left of the xiphisternum, then slightly outwards to the junction of the inner and middle thirds



*Fig. 276.*—TOTAL GASTRECTOMY MARWEDEL'S METHOD OF EXPOSING THE SUB DIAPHRAGMATIC PORTION OF THE ESOPHAGUS BY REFLECTION OF THE LEFT LOWER COSTAL CARTILAGES. A MEDIAN LAPAROTOMY INCISION HAS BEEN ENLARGED ABOVE THE UMBILICUS BY A TRANSVERSE INCISION DIVIDING THE RECTUS AND EXTERNAL OBLIQUE MUSCLES. THE UPPER RIGHT ANGLED FLAP OF SOFT TISSUE HAS BEEN DISSECTED FREE, SO THAT THE ANTERIOR SURFACES OF THE LOWER RIBS LIE EXPOSED. THE INDIVIDUAL RIBS ARE DIVIDED IN THE REGION OF THEIR CARTILAGES.

A = External oblique.

B = Rectus muscle.

C = Internal oblique.

D = Peritoneum.

E = Liver.

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of the left rectus muscle, and downwards in this plane until it reaches a point about 2 inches below and slightly to the left of the umbilicus (fig. 275). The inner border of the left rectus muscle is dissected free from its sheath and retracted widely outwards, the posterior sheath of this muscle and the peritoneum being incised from the costal margin for the whole length of the wound.

If a better exposure of the œsophagus is desired the xiphisternum should be removed, or the left costal margin reflected as recommended



Fig. 277.—TOTAL GASTRECTOMY EXPOSURE OF THE STOMACH AND OESOPHAGUS.

by Marwedel. By this latter method the 6th, 7th, 8th and 9th costal cartilages may be mobilised to allow a wide retraction to the left and ample exposure of the œsophagus and cardia, but I am not wholly in favour of this procedure for all cases, as it prolongs the operation and increases its risks (fig. 276). Tetra-cloths are applied to the margins of the incision and the wound is protected with mackintosh squares and abdominal swabs in the usual manner, after which the wound is widely retracted by means of a large self-retaining retractor such as Bonney's (fig. 277).

After the stomach has been carefully examined and it is decided that

total extirpation of the organ is possible, the operation is started by separating the great omentum from the transverse colon, as in Finsterer's operation (see fig. 262). After the right and left gastro-epiploic arteries have been tied near their origins, the pyloric artery is secured and



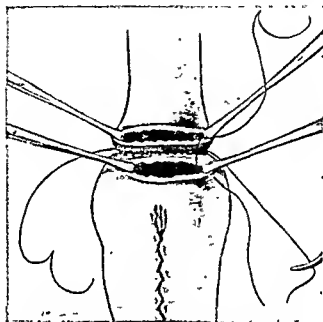
Fig. 278.—TOTAL GASTRECTOMY. THE STOMACH, ADEQUATELY FREED, IS BEING DRAWN FORWARD BY MEANS OF A GAUZE ROLL PASSED ROUND IT. THE PERITONEAL COVERING OF THE DIAPHRAGM WHICH HAS BEEN SPLIT IS PICKED UP BY TRACTION SUTURES, AND DISSECTED FREE SO THAT THE MUSCULATURE OF THE OESOPHAGUS AND THE LEFT VAGUS NERVE COME WELL INTO VIEW.

A—Left vagus nerve.

(Reproduced from "Kirschner's Operative Surgery," Lippincott. By kind permission of Julius Springer, Berlin.)

divided between strong ligatures and the fan-shaped gastro-hepatic omentum severed as close to the liver as possible, until the upper third of the lesser curvature is reached. The pylorus and the first portion of the duodenum are mobilised, the duodenum is crushed  $\frac{3}{4}$  inch away from the pylorus, divided, and its distal end securely closed and invaginated. The gastro-splenic omentum is separated from the spleen,

after which the stomach is drawn forcibly over the left costal margin so that the coronary artery can be rendered taut and a ligature applied close to the point where it springs from the cœliac axis. After the coronary artery has been divided, the loose fatty tissues and glands in the region of the right border of the œsophagus and the upper third of the lesser curvature are swept downwards in one mass. Extreme care must be taken in ligaturing the coronary artery not to damage the right border of the œsophagus or include it in a mass ligature.



*Fig 270—TOTAL GASTRECTOMY. END-TO-SIDE ANASTOMOSIS BETWEEN THE OESOPHAGUS AND PROXIMAL JEJUNUM. THE ANASTOMOSIS IS BEING PERFORMED WITHOUT THE AID OF CLAMPS.*

This is liable to happen as the gastro-hepatic omentum becomes thicker and thicker towards the cardia, and the right border of the œsophagus is difficult to distinguish.

After the anchor of the stomach, i.e. the coronary artery, has been divided between ligatures, the numerous filamentous bands on the inferior aspect of the œsophagus should be carefully picked up and cut to permit of further mobilisation.

The next step consists of freeing the œsophagus from its diaphragmatic foramen and from the last inch or two of the posterior mediastinum. An incision in the form of an inverted T is made through the peritoneum covering the anterior surface of the œsophagus, and the two peritoneal flaps are picked up with hæmostats, retracted, and dissected away with gauze pressure. When the œsophageal opening

is reached, a little blunt dissection is made with the finger around the opening, allowing the œsophagus to be pulled a little farther downwards into the abdominal cavity, this being further facilitated by the anæsthetist flexing the patient's spine and forcing the chin on to the chest.

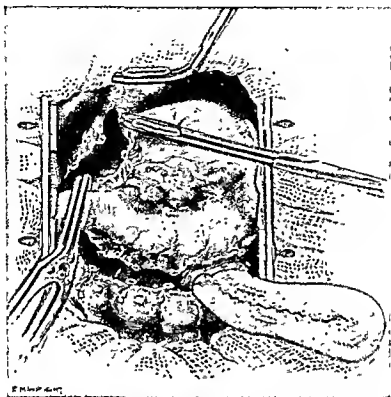


Fig. 280.—TOTAL GASTRECTOMY. THIS ILLUSTRATION DRAWN FROM A PHOTOGRAPH TAKEN AT OPERATION, SHOWS THE LONG SUB DIAPHRAGMATIC PORTION OF THE (ESOPHAGUS DIVIDED BETWEEN CLAMPS AFTER THE OMENTA AND THE BLOOD VESSELS OF THE STOMACH HAVE BEEN DIVIDED. THE DUODENUM IS ABOUT TO BE CUT THROUGH BETWEEN TWO PAYR CLAMPS. THE PROXIMAL LOOP OF JEJUNUM HAS BEEN DRAWN THROUGH A RENT IN THE MESOCOLON AND STITCHED TO THE OPENING AND A LARGE ENTERO-ANASTOMOSIS HAS BEEN MADE BETWEEN THE AFFERENT AND EFFERENT LIMBS OF THE JEJUNUM, AS RECOMMENDED BY ROEDER. THE PATIENT, A MAN OF 44, HAD A LARGE CARCINOMA INVOLVING A WIDE AREA OF THE ANTERIOR WALL OF THE STOMACH AND THE UPPER THIRD OF THE LESSEE CURVATURE. HE BORE THE OPERATION WELL, AND ENJOYED COMPARATIVE COMFORT AND A GOOD APPETITE UNTIL HIS DEATH TEN MONTHS LATER FROM SECONDARY DEPOSITS IN THE LIVER AND PERITONEUM (*Author's case.*)

As a further inch or two of the œsophagus comes into view, the left vagus nerve can be clearly seen on its anterior surface as a slender white cord or as a plexus of fine filamentous fibres (fig. 278). The right vagus nerve is situated on the posterior surface of the œsophagus. Both these nerves are now infiltrated with 1 per cent novocaine and divided, after which the œsophagus can be drawn down even farther.



When the œsophagus has been sufficiently mobilised, the proximal jejunum is anastomosed to the œsophagus (without the aid of clamps) as in Moynihan's operation (fig. 279).

At the completion of the operation a Ryle or Einhorn tube is passed through the nostril and led through the efferent limb of the jejunum and used for feeding purposes during the first few days after the operation.

In certain cases of cancer of the stomach, and particularly in diffuse leather-bottle stomach, when *the intra-abdominal portion of*

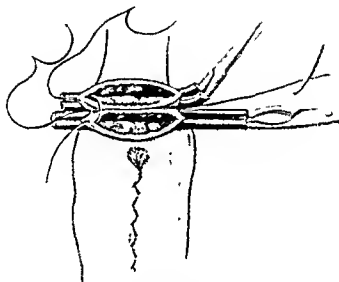


Fig 281.—TOTAL GASTRECTOMY. END TO SIDE ANASTOMOSIS BETWEEN THE ŒSOPHAGUS AND JEJUNUM. THE CONTINUOUS HÆMOSTATIC SUTURE HAS BEEN STARTED.

*the œsophagus may be unusually long*, Moynihan's method of using the mobilised stomach as a tractor is not essential. Instead, the œsophagus may be doubly clamped and cut away before proceeding with the anastomosis. This is performed as follows:

A small curved clamp with rubber-covered blades is made to grasp the œsophagus just proximal to the proposed line of section, whilst another clamp grasps it on the distal (stomach) side, the gut being divided between these two clamps. The middle portion of the transverse colon is then picked up and drawn through the abdominal incision to put the mesocolon on the stretch. A wound is then made in an avascular area of the mesocolon, and a long loop of proximal

jejunum brought through this rent. The length of this loop will vary with individual cases, but it should always be long enough to ensure that there is no traction on the suture line after the anastomosis is completed. An entero-anastomosis, 3-4 inches long, is made between the afferent and efferent limbs of the jejunal loop with the object of forming a greater food pouch, the apex of the loop being subsequently anastomosed to the cut end of the œsophagus.

Hoffman and Roeder suggest that the stoma of the entero-anastomosis should be at least 6 inches long (fig. 280). The jejunal loops are stitched to the rent in the mesocolon to prevent traction and herniation. The apex of the jejunal loop is then brought to the under-surface of the œsophagus, and anastomosed to its cut end in the following manner:

The curved clamp on the œsophageal stump is rotated so as to bring the posterior surface well into view. Six to eight interrupted sutures of the finest silk or thread anchor the jejunum to the posterior surface of the œsophagus in a line with, close to, and on the distal side of the clamp. *The greatest care must be taken in introducing these sutures, as the œsophagus is very friable and the sutures readily cut out.* A posterior continuous Lembert suture is then introduced (fig. 281). A small incision is made in the jejunum through all its walls, its length

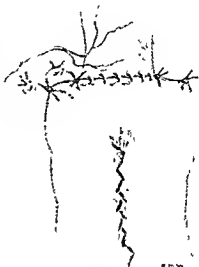


Fig. 282.—TOTAL GASTRECTOMY. THE END-TO-SIDE ANASTOMOSIS BETWEEN THE ŒSOPHAGUS AND JEJUNUM IS COMPLETED. TO PREVENT ACUTE ANGOULATED THE JEJUNUM ON EITHER SIDE OF THE ANASTOMOTIC LINE IS SUTURED TO THE DIAPHRAGM. THE SMALL ARTERY TO THE LOWER END OF THE ŒSOPHAGUS SHOULD BE PRESERVED TO PREVENT THE POSSIBILITY OF SUBSEQUENT SLOUGHING.

corresponding to the cut end of the œsophagus. The posterior margin of the œsophagus is then united to the posterior margin of the incised jejunum by a continuous through-and-through all-coats suture which extends completely around the anastomotic opening without change, the clamps being removed as the suture turns the corner. The sero-muscular stitch is then picked up again and is continued as a Lembert suture which invaginates and gives additional security to the suture line. When this is completed, a few interrupted sutures are introduced along the line of anastomosis, and a portion of the jejunum on each side of the union is stitched to the diaphragm or remnants of omentum

to prevent acute angulation, to relieve tension on the anastomosis, and to keep it immobile (fig. 282). The two peritoneal flaps which were raised from the anterior wall of the œsophagus are sutured to the jejunum in front of the anastomosis to form a barrier between the peritoneal cavity and the lower portion of the posterior mediastinum, affording also an excellent means of reinforcing the suture line.

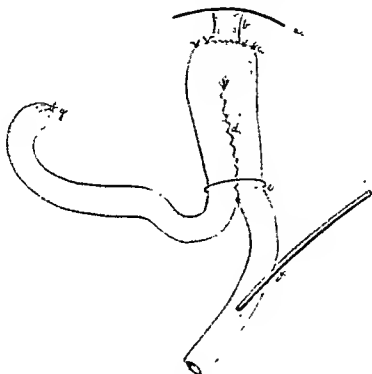


Fig. 283.—TOTAL GASTRECTOMY. DIAGRAM OF THE PARTS AFTER TOTAL GASTRECTOMY.

- a = Diaphragm.
- b = Œsophagus.
- c = Anastomosis between the œsophagus and jejunum.
- d = Entero anastomosis.
- e = Opening in the mesocolon.
- f = Jejunostomy.
- g = Closed end of the duodenum.

THE JEJUNOSTOMY IS NOT AS A RULE REQUIRED, AS A TUBE CAN BE PASSED THROUGH THE NOSE AND LED INTO THE EFFICIENT JEJUNAL LOOP TO BE USED FOR FEEDING PURPOSES AND TO RELIEVE STRAIN ON THE ANASTOMOTIC LINE.

If, when the operation is completed, it appears that the transverse colon is dragging or is likely to drag unduly on the line of anastomosis, it is a wise precaution to anchor the middle third of this portion of the gut to the peritoneum of the anterior abdominal wall (*transverse colopexy*). A jejunostomy for feeding purposes may be performed after Witzel's method, the tube being drawn out through a separate stab wound (fig. 283); but this does not form an essential or desirable

step in the operation as an Einhorn tube can easily be passed through the nose or mouth, through the anastomosis, and be guided into the efferent jejunal loop, where it can be kept *in situ* for feeding purposes for over a week and is well tolerated by the patient. The use of a tube in this way puts the anastomotic line at rest by relieving tension, thus promoting its rapid healing.

The abdominal wound is closed in the usual manner, and tension or supporting sutures are introduced to guard against the possibility of a ruptured abdomen.

### (B) SARCOMA OF THE STOMACH

*Incidence.* Ewing considers that 1 per cent of all gastric tumours are sarcomatous. Haggard found 244 cases of sarcoma of the stomach reported in literature, of which 107 were operated upon, the remainder being undiagnosed until post-mortem examination. D'Aunoy and Zoeller (*Amer. Jl. Surg.*, Vol. ix, 1930) collected 355 cases, and agree with Ewing that the incidence is 1 per cent of all stomach tumours. Anschutz and Konjetzny estimate the incidence to be somewhat higher—2 per cent.

Waltman Walters states that at the Mayo Clinic sarcoma of the stomach occurred in the proportion of 1 case of sarcoma to 159 of carcinoma between the years 1908 and 1920. Other authors give the ratio as 1 to 200.

*Age Incidence.* Sarcoma of the stomach occurs at an earlier average time of life than carcinoma of the stomach, the commonest age being 40 years or thereabouts.

*Pathology.* Three varieties are described :

- (1) Intra-gastric.
- (2) Extra-gastric.
- (3) Gross sarcomatous infiltration of the stomach, resembling linitis plastica.

Of these types extra-gastric tumours are more common than intra-gastric, whilst the infiltrating variety, which is of much greater malignancy, is commoner than either. Bertrand, in 70 cases of this disease collected from literature, found 4 of the intra-gastric, 31 of the extra-gastric, and 35 of the diffuse infiltrating types.

Metastases are found in about 30 per cent of the cases examined at

autopsy, and occur in the abdominal lymph glands, liver, and distant organs. It should be noted, therefore, that in about 70 per cent of cases of gastric sarcoma the growth is confined *solely* to the stomach, and consequently the results following wide removal of the growth should be very satisfactory.

The *intra-gastric* types arise in the submucous coat, and in addition to having a limited lateral spread their main direction of extension is towards the lumen of the stomach. There they may form large circular submucosal tumours which sometimes become pedunculated. The mucous membrane is tightly stretched over the growth and may ulcerate and bleed profusely. When the growth is situated near the pylorus and has a long pedicle it may cause a "ball-valve" pyloric obstruction. The growth is histologically a fibro- or spindle-celled sarcoma and is of low malignancy.

The *extra-gastric* types originate in the subserosa and grow away from the stomach towards the general peritoneal cavity. They involve only a small portion of the stomach wall and often become pedunculated. They frequently attain a large size and form hard, circular, freely-movable abdominal tumours. The pedicle by which the growth is attached to the stomach may become very attenuated, and it is even possible for such tumours to be cut adrift from their origin and to be caught up in coils of intestine or to migrate into the pelvis where they may be mistaken for uterine fibroids. When situated on the lesser curvature they grow in an upward direction towards the liver between the layers of the gastro-hepatic omentum, whilst when springing from the greater curvature they may lie between the layers of the gastro-colic omentum, fill the lesser sac, and possess some of the clinical features of a pseudo-pancreatic cyst.

Growths of this type often undergo cystic degeneration, and hæmorrhage into the tumour mass frequently occurs. They may cause confusion in diagnosis and have been mistaken for the liver in Banti's disease, for pancreatic cysts, mesenteric cysts, hydatid cysts, cystic swellings of the kidney, ovarian cysts, or uterine fibroids.

On exploratory operation they often appear at first sight to be inoperable owing to the numerous vascular adhesions by which they are surrounded; but they are, in fact, easily dissected out after these adhesions have been separated.

These tumours are only locally malignant, and the prognosis following their removal is very good.

The *infiltrating* types of sarcomata are round-celled growths, which rapidly extend in the submucous coat in a manner similar to that which

occurs in leather-bottle stomach, and eventually involve a large portion of the stomach. It may be very difficult, both from macroscopic and from microscopic examinations, to differentiate between a diffuse sarcoma of the stomach and carcinomatous linitis plastica.

*Diagnosis.* A correct pre-operative diagnosis is rarely made. On X-ray examination the intra-gastric type is often diagnosed as carcinoma of the stomach or simple tumour. As previously stated, the extra-gastric types may be mistaken for cysts or tumours of the pancreas, liver, kidney, ovary, or uterus; whilst the diffuse infiltrating sarcomata of the stomach resemble linitis plastica in its clinical, radiological, and pathological features.

*Treatment.* Nearly 70 per cent of these tumours prove to be resectable. With the intra- and extra-gastric forms large resections are, as a rule, unnecessary. After carefully ligaturing off the surrounding adhesions, extra-gastric growths are shelled out from their vascular bed. The area of attachment of the pedicle to the stomach is examined to determine as far as possible the amount of stomach wall involved in growth, and the extent of the implication will decide the amount of stomach to be removed by wedge-excision. The resulting gap is closed with a series of continuous sutures in such a manner that no appreciable narrowing results and there is no tension on the suture line.

This operation of removal of the tumour with wedge-excision of the stomach wall is also applicable to intra-gastric growths which have a narrow pedicle. Where the pedicle is broad, the tumour sessile, or where a considerable portion of the stomach wall is infiltrated, partial gastrectomy is to be preferred.

The late results are favourable. This is shown by the result of 38 cases operated upon at the Mayo Clinic of which 12 remained cured over a period of one to nine years or more. The operative mortality in this series was 13 per cent.

### (C) INNOCENT NEW GROWTHS OF THE STOMACH

Innocent new growths of the stomach are rare. Figures from the Mayo Clinic show that less than 0.5 per cent of gastric tumours are benign. When developing in the body of the stomach they do not cause symptoms until they have attained a large size. On the other hand, benign growths situated in the pyloric segment may at quite an

early stage produce pyloric obstruction, or even intussusception or volvulus of the stomach when the growth is forced into the duodenum. In such cases a portion of the stomach wall may become strangulated, and may even perforate, giving rise to general peritonitis.

All innocent growths of the stomach are apt to produce attacks of colicky pain, occasional sharp bouts of vomiting, and hæmorrhage which may at times be very profuse. These tumours originate in any of the layers of the stomach, and either remain restricted to one layer or spread beyond it into some other portion of the stomach. They are generally submucous, sessile or pedunculated, and freely movable. They frequently form rounded or flat tumours which project into the stomach cavity or into the peritoneal cavity. The majority of benign tumours develop in the pyloric region, and arise from the anterior or posterior wall, showing a predilection for the zone of the greater curvature. They vary in size from  $\frac{1}{2}$  to 18 inches in diameter, and are all prone to undergo malignant degeneration. Many *pathological varieties* are described, such as :

- (1) Myomata.
- (2) Fibromata.
- (3) Adenomata.
- (4) Lipomata.
- (5) Angiomata.
- (6) Gastric polyposis.
- (7) Cysts.

*Myomata.* These are the commonest innocent tumours and rank third in importance among the growths of the stomach. They are usually single, variable in size, occur chiefly in the pyloric region, may be sessile or pedunculated, and intra- or extra-gastric. When the fibrous stroma is very marked they are termed fibromyomata. They may undergo myxomatous or sarcomatous degeneration. They do not cause hæmorrhage until their mucous-covered surface becomes ulcerated, when hæmatemesis and mæna may be severe. When situated in the pyloric segment of the stomach they may cause a ball-valve obstruction, or even produce intussusception when the tumour is dragged into the duodenum and pulls the stomach wall with it.

Those of the extra-gastric type originate in the subserosa and spring from the greater curvature. They may form large oval or spheroidal lobulated abdominal tumours, causing great difficulty in diagnosis.

*Fibromata.* These are exceedingly rare. They usually originate in the pyloric region, and may be sessile or pedunculated.

*Adenomata.* These are gastric polypi which may be single or multiple. They form rounded sessile or pedunculated tumours (fig. 284). Stewart found that 28 per cent of polypi of the stomach were associated with carcinoma, but only 4.9 per cent of carcinomata were associated with polypi. Benedict and Allen, in a series of 17 cases of gastric polypi which were giving fairly severe symptoms, found that there was microscopical evidence of potential malignancy in 7 cases, i.e. an incidence of 41.2 per cent. They also state that Miller, and Eliason and Wright, after searching extensively in many microscopical sections taken from adenomatous polypi, found carcinomatous change in 8 of 23 cases, an incidence of 35 per cent.

When a portion or the whole of the gastric mucous membrane is studded with closely packed adenomata the condition is termed polyposis. The massive cauliflower (or polypoid) carcinomata probably originate in polypi which are situated in the region of the greater curvature,

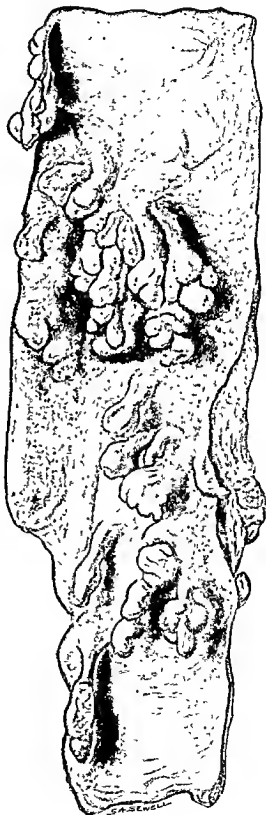


Fig 284.—ADENOMATA OF THE STOMACH. PORTION OF STOMACH, FROM THE MUCOSAL SURFACE OF WHICH THERE SPRING A NUMBER OF LOBULATED PEDUNCULATED POLYPOID GROWTHS.

(Museum, Royal College of Surgeons)



the slowness of their growth, their unusual position, and the frequent absence of secondaries in the glands until the disease is far advanced, suggesting this possibility.

*Lipomata.* These form lobulated yellow submucous or subperitoneal tumours and give rise to but few symptoms.

*Angiomata.* These form round, soft, mobile, sessile, submucous tumours. They are dark red or black in colour, often undergo sarcomatous degeneration, and bleed freely when the surface of the tumour becomes ulcerated.

*Gastric Polyposis.* In this disease a localised portion or the whole of the mucous surface of the stomach becomes studded with numerous polypi. The condition is exceedingly rare, and Sinclair (*Br. Jl. Surg.*, Vol. xx, p. 645, 1933) estimates that less than 100 cases have been reported to date. In 1919 Balfour considered it to be the rarest of all benign tumours, and stated that in 8000 operations performed at the Mayo Clinic for gastric lesions, gastric polyposis had been found in only one instance. Brunn and Pearl (*Surg., Gynec., and Obst.*, 559, Nov., 1926), collected 84 cases and included 5 of their own.

I operated upon a case in 1926, a man of 43, who was referred to me as suffering from cancer of the stomach. At operation, the stomach, which felt doughy, was opened and explored through an incision made in the anterior wall, but owing to the diffuse and almost universal implication of the mucous membrane with closely packed polypi I deemed it unwise to attempt total gastrectomy, and nothing further was done apart from removal of a small segment of the mucous membrane for microscopical examination. The pathological report confirmed the operative diagnosis of polyposis.

This patient is alive to day, but complains of epigastric pain, anorexia, and bouts of vomiting.

Gastric polyposis is not a pathological entity, as polypoid masses may be either neoplastic or inflammatory in origin; in fact, many regard the adenomatous masses as being secondary to chronic hypertrophic gastritis. In one variety there are a large number of discrete, pedunculated or lobulated tumours, whilst in the other the masses appear to be almost confluent and form a well-demarcated plaque.

The disease may be localised or generalised; in the former condition the polypoid masses are segregated in the pyloric segment and along the greater curvature, whereas in the latter type the whole mucous surface is implicated.

At operation doughy, worm-like masses may be felt in the stomach,

and when the viscus is opened the "warts" may be seen closely packed together, forming either a velvety red plaque or a group of discrete polypi which vary in size from that of a pea to that of a cherry. The mucous membrane is drawn into large folds which superficially resemble the convolutions of the brain. The polypi are papillary adenomata which are composed of columnar epithelial cells arranged in well-formed acini which are supported by lax connective tissue stroma. As stated above, they may cause profuse bleeding or undergo malignant degeneration.

The disease may give rise to no characteristic symptoms, but as a rule there is a long history of chronic dyspepsia, anorexia, epigastric pain or discomfort, vomiting of large quantities of blood-stained mucus, and anæmia. Free hydrochloric acid is absent in about 95 per cent of these cases. X-ray examination will show the characteristic mottled appearance or numerous interrupted filling defects. These irregular defects in the contour of the stomach are due to the indentation of the polypi.

With regard to *treatment*, partial gastrectomy is advised for the localised variety when the tumours are confined to the pyloric region of the stomach. When, however, the disease is generalised the treatment should be the same as that recommended for chronic gastritis, including frequent gastric lavage, the administration of hydrochloric acid, and a bland nutritious diet.

*Cysts of the Stomach.* These, which are of no surgical importance, may be enumerated as follows :

- (a) Retention cysts.
- (b) Hæmorrhagic or traumatic cysts.
- (c) Degeneration cysts.
- (d) Lymphangiomatous cysts.
- (e) Dermoid cysts.
- (f) Hydatid cysts.

*Symptoms of Innocent New Growths of the Stomach.* It is rare for the symptoms produced by benign tumours to be sufficiently characteristic to allow of a positive pre-operative diagnosis. Symptoms, when present, usually suggest that the patient is suffering from chronic gastritis, chronic peptic ulceration, malignant growth of the stomach, or

some grave form of anæmia. The symptoms of all innocent tumours of the stomach may be grouped conveniently together as follows :

(1) *Epigastric Discomfort and Pain.* This is variable. With the large extra-gastric tumours there will be a sensation of dragging, weight, or discomfort in the region around the umbilicus. The symptoms of pyloric growths often mimic those of chronic duodenal ulcer, whilst pedunculated growths in this region may give rise to colicky pain somewhat similar to that produced by gall-stones.

(2) *Vomiting.* As chronic gastritis is frequently associated with benign gastric tumours, there is often vomiting of large quantities of mucus or blood-stained mucus, this being followed by a certain degree of relief from the epigastric discomfort.

With pedunculated growths in the pyloric segment obstruction may be intermittent. When the growth is impacted in the pyloric canal vomiting will occur ; when the tumour falls back into the cavity of the stomach vomiting ceases and there is immediate relief from pain, the long pedicle which attaches the growth to the stomach being responsible for this ball-valve action.

(3) *Hamatemesis and Melæna.* Bleeding which at times may be very severe does not occur until the mucous membrane over the growth has become ulcerated. A grave anæmia may result from a slight but continuous loss of blood. Occult blood is frequently found in the stools of patients suffering from innocent growths of the stomach.

(4) *Loss of Appetite.* Owing to the accompanying chronic gastritis and frequent absence of hydrochloric acid in the gastric juice, there may be loss of appetite.

On *physical examination* nothing will as a rule be discovered on abdominal palpation. Occasionally, however, a freely movable tumour may be felt in the epigastrium which should always raise suspicion of benign growth of the stomach. Where an extra-gastric tumour becomes fixed by adhesions, the overlying area will be tender on palpation.

As previously stated, occult blood tests are often positive, and a test meal will frequently show that there is absence of hydrochloric acid. On X-ray examination of the stomach after the administration of a barium meal, filling defects which simulate those found in cases of carcinoma of the stomach will often be noticed. The large rounded

pedunculated tumours present no difficulty in diagnosis as they produce filling defects which have smooth, clearly defined outlines. While on screening there is delay in emptying with large tumours of the stomach, with small tumours which produce irritability of the stomach the peristalsis is vigorous and emptying is rapid.

*Treatment of Innocent New Growths of the Stomach.*

(1) *Of the large single pedunculated tumours with a narrow base of attachment.*

(a) Intra-gastric. An incision should be made through the anterior wall of the stomach to expose the tumour. Its base should be very carefully palpated to determine whether there is any induration, as this, when present, will denote, or at least suggest, that the tumour is undergoing malignant degeneration. If the base of attachment is small and there is no induration, the tumour can be removed by wide excision of its base together with a margin of healthy mucosal tissue, the resulting wound in the mucous membrane being closed with a series of interrupted sutures. If, on the other hand, there is definite induration at the base of the tumour, a still wider segment which includes all the layers of the stomach wall is excised, together with the tumour.

(b) Extra-gastric. As these tumours often undergo sarcomatous degeneration, a V-shaped portion of the greater curvature should be removed around the point of attachment and the aperture closed with three tiers of sutures.

(2) *Of diffuse sessile tumours.* When these occur in the pyloric segment of the stomach, partial gastrectomy should be performed. When they occur in the body of the stomach, however, local excision is usually sufficient. Where any doubt exists as to the benignity of the growth, a wide resection, often amounting to partial gastrectomy, is always preferable.

(3) *Of multiple tumours.* When these are grouped in the pyloric portion of the stomach partial gastrectomy should be undertaken. When, however, the whole lining of the stomach is involved total gastrectomy is the only possible method of dealing radically with the condition.

## CHAPTER VIII

### THE AUTHOR'S TECHNIQUE OF THE PÉAN-BILLROTH I OPERATION

by

ENRIQUE FINOCHIETTO

THERE are many methods of performing a partial gastro-duodenal resection, and the operation which I am about to describe can be undertaken in 70-80 per cent of gastric and duodenal ulcer cases. During the last ten years I have practised it on nearly four-fifths of my cases, and I have been entirely satisfied with the immediate and late results.

This operation was performed for the first time by Péan, in Paris, and Billroth, in Vienna, within a few weeks of one another. Latin authors call it Péan's operation, but it is more generally known throughout the world as the Billroth I. I give credit where credit is due, and consider that it is more correctly termed the Péan-Billroth I operation.

Péan and Billroth performed an economic resection, limiting themselves to what was, in fact, nothing more than a pylorectomy. Nowadays, however, the excisions are very extensive and include at least 3-4 cms. of the first part of the duodenum, the pylorus, the vestibule, the greater part of the lesser curvature of the stomach—even all of it, and approximately two-thirds of the greater curvature. But the extent of the resection is partly governed by the position and nature of the pathological lesions encountered at operation.

In modern times von Haberer has been the champion of this type of operation. It now has many advocates, and appears to be increasing in popularity. I have made one or two modifications in the technique which I consider to be important features, and these I will describe in detail.

The *dangers* of the operation are those usually encountered in any gastric operation, but the two most important are hæmorrhage and infection.

Infection is due to contamination of the wound or peritoneal cavity during the process of operation, or afterwards through a leak from an ineffectively approximated suture line. I attach great importance to

careful and scrupulous hæmostasis during every step of the operation, and consider that quite a number of lives are lost through the oozing which may result from severed adhesions and raw surfaces which are imperfectly ligatured. This blood-letting from many small points is a great cause of shock or plastic peritonitis.

The *preparation* of the patient and the *post-operative treatment* are as important as in any other abdominal operation. In practically all my cases I use local anaesthesia. Formerly, besides the infiltration of the abdominal wall, I used splanchnic anaesthesia after the method



Fig. 285.

described by Braun. But for the past four years I have abandoned this latter procedure in favour of a more simple "creeping" local analgesia of the omenta, which I have found to be even more satisfactory.

*Fig. 285.* This is an illustration of the syringe I now use for local anaesthesia, and which is known as Schimmelbusch's syringe. It is made for me by Collin, of Paris. It is a very convenient syringe and can be used with one hand only.

Every surgeon has his pet instruments; in fact, some of us find it impossible to operate without them. I have had a number of instruments specially adapted for my own use in abdominal work, and those made for me by Collin are particularly good.

I explore the abdomen through a mid-line incision which starts from the tip of the xiphisternum and ends just above the umbilicus. At the completion of the operation this incision is closed with a strong

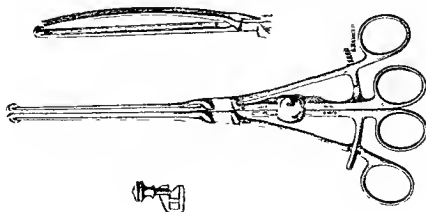
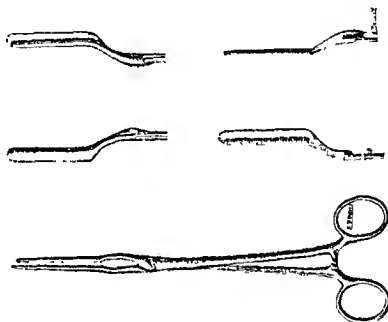


Fig. 286.

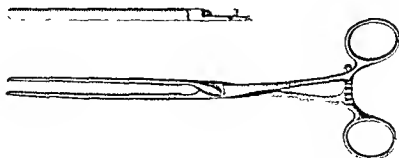
catgut suture which embraces all the layers of the abdominal wall in this region, except the skin, which I approximate with interrupted linen thread sutures and Michel clips.

*Fig. 286* is my twin clamp, which has a simple device for adjustment by means of a screw fixed to one of its blades. This screw, which works on an inclined plane, permits of the clamps being easily adjusted so that they lie close together in any desired position.



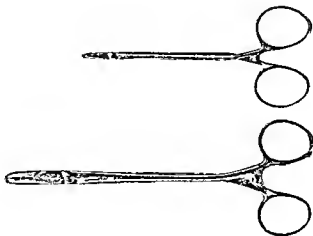
*Fig. 286.*

*Fig. 287.* This is the author's duodenal clamp which has one of its blades in the shape of an oar. When this is accurately applied to the duodenum and rotated, the posterior surface of the gut is thrown into a prominent fold which facilitates the introduction of the sutures (see figs. 308, 310, 313 and 314).



*Fig. 287.*

*Fig. 288.* This I call a non-slipping clamp. One is applied to the stomach and one to the duodenum, prior to resection. They have on one edge of each blade numerous serrations which prevent the tissues from slipping from their grip. The one applied to the stomach is placed just distal to the proposed line of section. The position of these clamps is shown in figures 309, 311, 312 and 315.



*Fig. 289.*

*Fig. 289.* A needle-holder, 18 cms. long, which can also be used for deep ligatures. This is a modification of Schoemacher's needle-holder. At the end of one blade it carries a spur, and on the inner side of one of the finger-rings there is a small spring which holds both ends of the ligature. The smaller needle-holder is used for holding very delicate needles threaded with fine sutures (figs. 313 and 314).

*Fig. 290* shows the surgeon's table with all the instruments in their correct positions for use. On the right are swabs and two pairs of gloves. The surgeon changes his gloves on completion of the anastomosis and again just before closing the abdominal wall. All the needles are threaded before the operation is begun. There are four straight intestinal needles, two curved ones holding fine catgut, and two others threaded with silk or cotton thread.

The instruments comprise scalpels, dissecting forceps, scissors, needle-holders, two ring forceps, and the triangular forceps of Duval. There is one pair of forceps, the blades of which are covered with a small swab. This is dipped in iodine to touch the mucous membrane after resection.

There are also on the surgeon's table curved mosquito forceps, two curved Kocher forceps for ligatures, three Kocher forceps, a local anæsthetic outfit, the author's duodenal clamp, two non-slipping



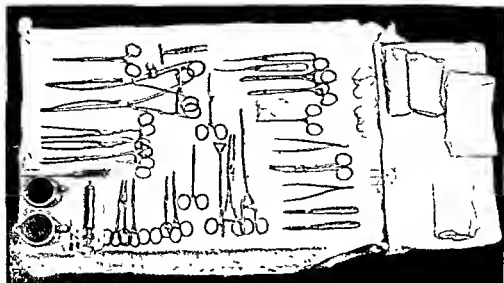


Fig. 290.

clamps, and the two portions of the twin clamp. The T-forceps are used for intestinal suture, but not in the operation I am about to describe.

*Fig. 291.* The assistant's table with instruments placed in their correct positions. On the left of the picture will be seen several pairs of cotton gloves. To the right, commencing below, four Kocher forceps, a pair of Farabœuf retractors, strong scissors, three vaginal

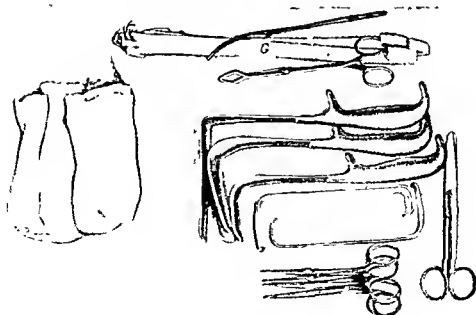


Fig. 291.

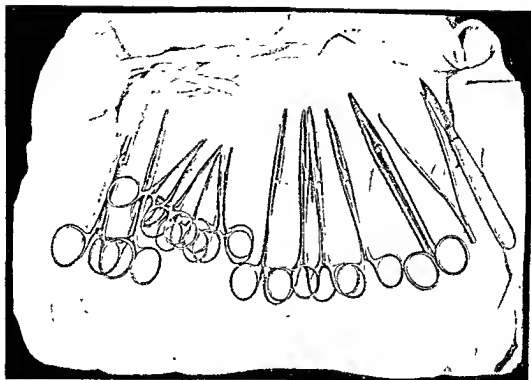


Fig. 292.

retractors which are used for abdominal work, a pair of lozenge-shaped forceps, and one pair of large curved Kocher forceps for passing ligature threads. G=long narrow paper envelopes containing the ligatures—silk or cotton thread.

*Fig. 292.* A tray for soiled instruments. This tray lies on a special towel on the patient's thighs.

*Fig. 293* shows the position of the surgeon, his assistants, and the instrument tables. The surgeon can be identified by means of his head-lamp.

#### OPERATION

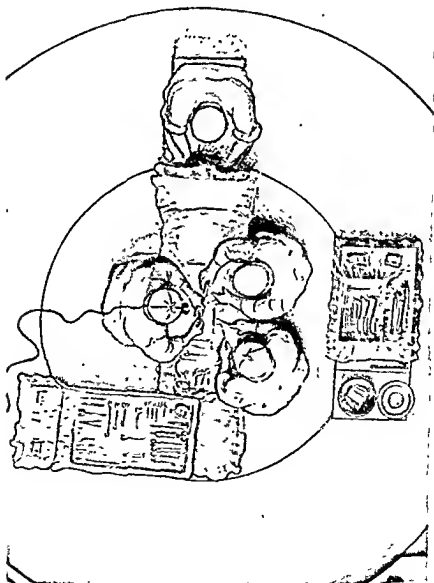
After opening the abdomen and completing the anaesthesia, a general exploration of the abdominal viscera is carried out, and much time is devoted to a most scrupulous examination of the lesions that are found.

The whole stomach and duodenum are very carefully inspected and palpated. If there is an organic lesion, such as a chronic gastric ulcer situated on the lesser curvature of the stomach, the degree of fixation and other changes that have occurred are very carefully noted. If a chronic ulcer is present one must determine whether it is adherent

upwards and towards the left, and whether or not it has formed binding adhesions to the left lobe of the liver, or has even penetrated this organ.

If there is a chronic duodenal ulcer anteriorly placed, the extent of the cicatrization, of the deformity of the bulb, and of the surrounding adhesions is easily discerned. The attachments of a duodenal ulcer situated on the posterior wall are, however, sometimes impossible to gauge until a dissection of the parts around is well advanced.

When cancer is present in the pyloric region or in the body of the



stomach its extent, mobility, and lymphatic involvement are determined before deciding upon the type and extent of resection most suitable under the circumstances.

The first step in the operation consists of examining the posterior wall of the stomach through an opening in an avascular area in the middle of the gastro-colic omentum. All adhesions which are present between the posterior wall of the stomach and the stomach bed are divided between two ligatures. By dividing all the adhesions the entire posterior wall of the stomach and the pancreas can be explored, and if a penetrating ulcer is present its extent can be ascertained.

In cases of chronic duodenal ulcer, when the duodenal bulb is distorted and scarred it may be partially covered by a mass of sero-vascular membranes. Some of these adhesions are membranous, or even ligamentous. Being very vascular they require most careful division and ligature.

Some duodenal ulcers may form large inflammatory tumours and produce gross distortion, deformities, and tight strictures of the duodenum. The adhesions in such cases often anchor the duodenum upwards towards the liver or backwards towards the posterior abdominal wall. They often involve the head of the pancreas which becomes very much indurated.

The duodenum may be so inextricably welded to the pancreas that separation of the two viscera becomes a physical impossibility, and the radical operation has to be abandoned in favour of some simple drainage operation such as posterior gastro-jejunostomy, or an exclusion operation after the method of Hans Finsterer, of Vienna.

There are a number of cases in which conditions at first sight would seem to preclude the performance of any resection operation, but where patient dissection and freeing of the adhesions, and mobilisation of the duodenum will render the Péan-Billroth I operation possible. The difficult cases are those in which inflamed lymphatic glands are found along the lesser curvature and along the inner border of the duodenum, and in which the inflammatory tumour distorts the gut and drags the thickened omenta with it.

Through the opening in the gastro-colic omentum which has been used for exploring the posterior aspect of the stomach and pancreas, the pancreatico-duodenal angle is inspected. Adhesions here are often very thin and permit of easy separation. If a posterior duodenal ulcer is present its attachments can easily be identified when this angle has been well cleared.

The exploration of the duodenum is not complete until we have

freed its outer border, which is so often fixed by means of thick sero-vascular bands. The Péan-Billroth I operation can only be performed when the duodenum has been completely freed and mobilised. I consider this mobilisation of the duodenum to be the most important step in the operation, and in order to overcome this displacement of the organs to the right I have often been obliged to alter the classical technique described many years ago by Kocher.



Fig. 294.

*Fig. 294.* Instead of dissecting the peritoneum and the outer border of the duodenum, I commence by severing the filamentous bands and adhesions over the very middle of the gut by means of a blunt instrument, and by enlarging the opening thus made both upwards and downwards. Along this outer border of the duodenum the mobilisation proceeds until the colon is displaced downwards. The duodenum, when freed, can be turned over and carried towards the middle line. Behind it can be seen the inferior vena cava in all its width and about 7-8 cms. of its length. The adhesions

and bands are firmer and more vascular at the upper part, where most of them require two ligatures. The freed duodenum permits of a more thorough examination of any duodenal ulcers which may be present, of the head of the pancreas, and of the structures contained in the right border of the gastro-hepatic omentum. It is very dangerous to remove any portion of the pancreas whilst resecting a peptic ulcer; if, therefore, an ulcer has penetrated the pancreas it is best to sacrifice a portion of gut and to leave the ulcer attached to the pancreas, rather than to attempt local excision of a portion of the gland.



Fig. 295.

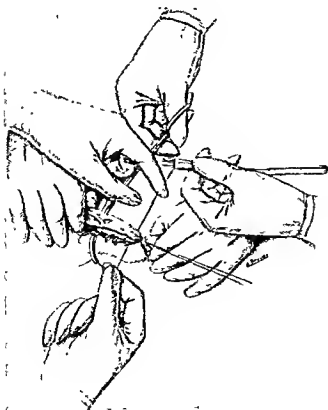


Fig. 296.

In actual practice, if I cannot free the duodenum well away from the gland, I often desist from this type of operation.

Before proceeding further with the operation and after ensuring a complete hæmostasis, I place a gauze swab to cover the raw surface which has resulted from the mobilisation of the duodenum, and this swab I leave in position, not removing it until the operation is nearly complete.

Next comes the division of the right part of both omenta. Ligatures are placed on each vessel before it is cut. The vessels are tied in continuity. The vessels attached to that portion of the stomach or duodenum about to be resected are ligatured and tied with two knots, and a little further away two ligatures are placed on the same vessel, each tied with three knots.

*Fig. 297* shows these three ligatures applied and about to be divided with scissors. No forceps are used in picking up blood-vessels as they are a hindrance.

At the completion of the operation I make quite sure that there is not a single bleeding point anywhere, and this method of tying blood-vessels in continuity ensures a bloodless field.

I use cotton gloves over the rubber ones to enable me to tighten the knots more securely, and also to prevent the ligatures from slipping.

In order to avoid excessive handling and exposure of the ligatures, Dr. Ricardo Finochietto and I have devised the plan of having the ligatures cut into uniform lengths of 32 cms., and placed in paper sheaths which have been sterilised in an autoclave (*figs. 295, 296 and 291, c.*).

*Figs. 295 and 296.* When ligaturing the omenta I make an opening in a non-vascular region, through which my assistant passes the end of the thread with the tip of the long Kocher forceps. This thread I pick up and tie.

*Fig. 297.* The portion of omentum containing the ligatured blood-vessels is divided with scissors, leaving enough tissue on either side to prevent any slipping of the ligatures.

*Fig. 298.* (See also *fig. 305.*) For the ligature of the smaller vessels the surgeon passes two forceps under the vessel, one being used to pick up the end of the threads which are handed to him, and the other being kept as a guide.

*Fig. 302.* For the very small vessels encountered during the separation of the duodenum, curved mosquito forceps are used. They pick up the points of the blood-vessels which are immediately ligatured.

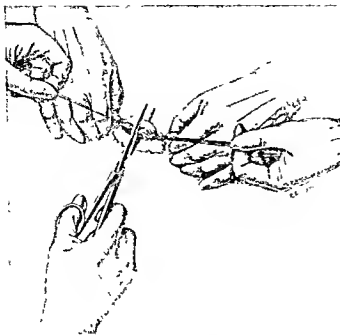


Fig. 297.

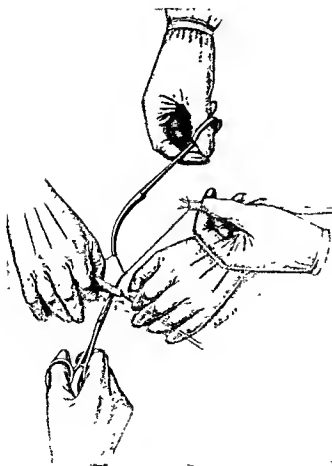
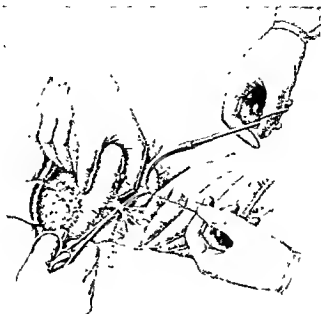


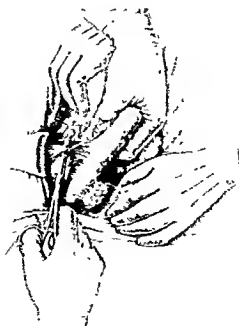
Fig. 298.



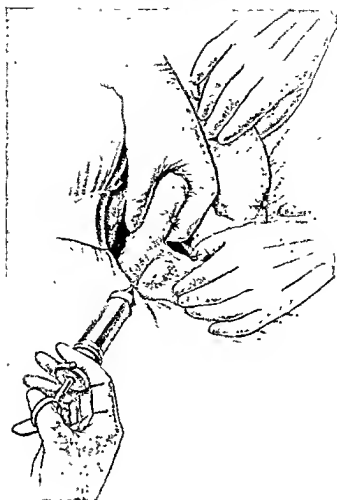
*Figs. 299, 300 and 301.* The ligature of the omenta commences at the original opening in the gastro-colic omentum, and, working towards the right, the blood-vessels are picked up as they are encountered, one by



*Fig. 299.*



*Fig. 300.*

*Fig. 301.*

one, and ligatured. As the ligaturing proceeds towards the duodenum the blood-vessels are tied very close to the gut. The vessels on the lesser curvature and those situated upon the upper and outer border of the duodenum are sometimes difficult to secure owing to the inflammatory reaction which may be present. Again, they are deeply placed. The mobilisation of the duodenum, however, greatly facilitates the application of the ligatures. Before proceeding with the division of the left half of the omenta, the whole of the posterior surface of the duodenum to be resected and used in the anastomosis must be freed so that the anastomotic sutures may be inserted without the slightest degree of tension.

In cases of gastric ulcer or where the duodenum is mobile, no difficulty will be experienced in isolating and ligaturing the vessels in the region of the pylorus and duodenum. When a duodenal ulcer is present the duodenum must be separated from its posterior attachments for 18-20 mm. beyond the ulcer.

*Fig. 302.* As the space between the head of the pancreas and the duodenum is narrow, every care must be taken not to injure the pancreas when separating the two viscera. The dissection of the duodenum from the pancreas is carried out by means of a sharp scalpel. When a penetrating ulcer is found it may be deemed advisable to excise the ulcer, leaving the ulcer crater adherent to the pancreas. In very difficult cases I have found it easier to commence by dissecting the affected zone in a concentric manner, that is, by gradually separating the upper and inner sides and by burrowing with the point of the scissors or with a dissector both from below and on the outer side of the gut, and then in between healthy pancreas and duodenum.

This concentric dissection may be the only way of obtaining a clear view of the ulcer and permitting of an easy separation of the ulcer from the pancreas.

A series of membranes, like thick veils, bridge the space between the head of the pancreas and the duodenum. Some of these membranes start in front of the duodenum and continue downwards over the transverse mesocolon. If these are cut the inner border of the duodenum suddenly becomes visible, and by using the points of the Mayo scissors as a dissector, opening them and closing them, a cleavage is made



*Fig. 302.*

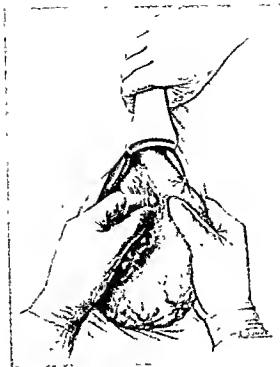


Fig. 303.

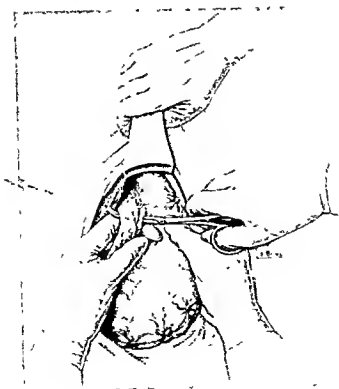


Fig. 304.

between the very small blood-vessels crossing between the duodenum and the pancreas. These vessels are picked up with mosquito forceps and ligatured with fine silk.

*Fig. 301.* To make this step easier I used to infiltrate the space with saline or anæsthetic solution ; but lately I have found this unnecessary. I now make certain at this stage that I possess the requisite 18-20 mm. of healthy tissue beyond the ulcer ; that is, sufficient space to permit of resection and easy suture.

The posterior surface of the duodenum is raw, and the muscular layer is visible. But if the mobilisation of the duodenum has been correctly performed, the surface will be smooth enough to hold the sutures. If the gut has been unavoidably opened, it is temporarily clamped with ring forceps.

As previously mentioned, before proceeding with the operation a gauze swab is placed in the region just dissected. The separation of the left half of the gastro-colic omentum is continued until the point of the greater curvature, just below the gastro-splenic omentum, is reached, where an artery is found coursing transversely. This part of the greater curvature of the stomach below the gastro-splenic omentum must be well cleared of all vascular or fatty tissues which would, at a later stage, hinder correct and accurate application of the sutures.

Having completed the ligature of the blood-vessels on the greater curvature, I now proceed to tie the coronary artery (the left gastric artery).

*Figs. 303, 304 and 305.* The forefinger of the left hand is passed under the gastro-hepatic omentum, high up in the region of the lesser curvature, as shown in figure 303. The forefinger of this hand is applied to the lesser curvature and pushes forward the blood-vessels contained in the omentum. An opening is made between the vessels and the stomach wall by means of the left forefinger and the thumbs working together. Through this opening the forceps and ligatures are passed. The threads of the two proximal ligatures are held by the forceps, so that this stump can be used at the completion of the operation to cover the anastomotic line in the stomach (see fig. 330).

*Fig. 306.* The stomach is now pulled downwards and to the left, exposing on its posterior surface several large veins which course under the serous layer. These veins run in the direction of the lesser curvature and may be perforated and give rise to troublesome hæmatomata at a later stage when the anastomotic sutures are being applied. At this stage these should therefore be carefully isolated and ligatured.

We have now three-quarters or four-fifths of the stomach completely

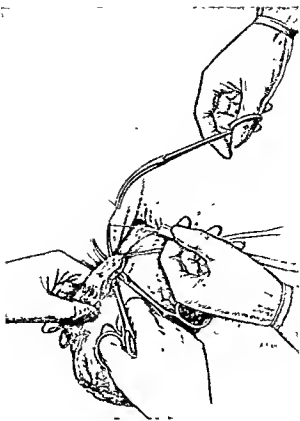


Fig. 305.

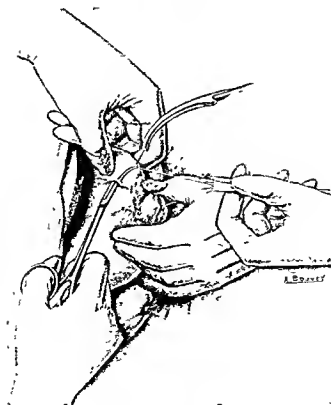


Fig. 306.

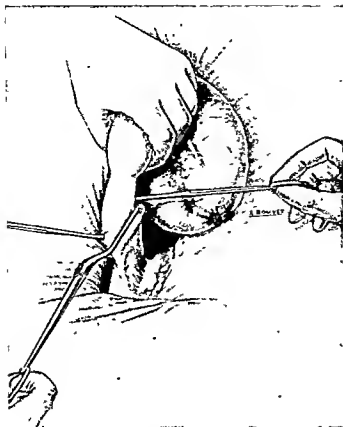
mobilised, so that a wide resection can be carried out without much difficulty.

In duodenal ulcer cases we have now to decide whether or not a Péan-Billroth I operation is the most suitable undertaking under the circumstances. There are a few contra-indications to its performance, and we would cite those cases in which the first portion of the duodenum is extensively scarred, narrowed, and cedematous. Again, there are cases where the pancreas and duodenum have become so moulded together that an easy approximation of the divided end of the duodenum and stomach is not feasible. There would, in fact, be too great a strain imposed upon the suture line if an anastomosis were performed under such conditions.

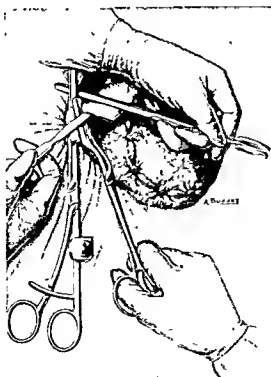
*Fig. 307.* The duodenal clamp is now applied, the gut first being steadied by Allis forceps, one pair being placed at the upper and another pair at the lower border of the gut. The blades of the duodenal clamp are made of standard size. They are applied to the duodenum either transversely or obliquely, depending upon the width of the duodenum. When the duodenum is narrow the clamp is applied obliquely so that the whole length of the blades is grasping the gut, thus ensuring an opening of adequate size when the gut is cut across.



*Fig. 307.*



*Fig. 309.*



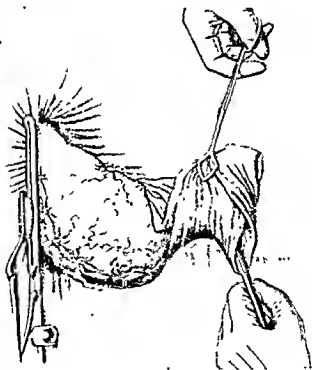
*Fig. 303.*



*Figs. 307, 308 and 309.* The duodenum is cut across between the non-slipping clamp and the duodenal clamp. These two clamps are placed a few mm. apart. The gut is then shaved off at the upper border of the duodenal clamp (fig. 309).

Should the lumen of the duodenum be narrow, the duodenal clamp should be placed obliquely, as suggested by Mayer, to increase the calibre of the gut (fig. 307). Even if by this method it is found that the duodenal lumen is still too narrow for anastomotic purposes, it can be further increased by making a small incision in the lower border of the duodenum (see fig. 319).

*Figs. 308 and 309.* Between the duodenal clamp and the pancreas there should be sufficient room to place the duodenal half of the twin clamp. As soon as the gut is severed the iodine-soaked swab is gently passed over the edges of the mucous membrane.



*Fig. 310.*

*Fig. 310.* The pyloric end of the stomach and the small duodenal stump are now covered over with a gauze swab, and the stomach is drawn over to the left.

*Fig. 311.* The stomach half of the twin clamp is then placed high up on the stomach, whilst a pair of Allis forceps supports the gastric pouch. The two halves of the twin clamp are drawn together and locked

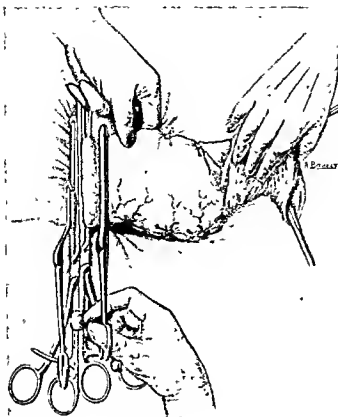


Fig. 311.

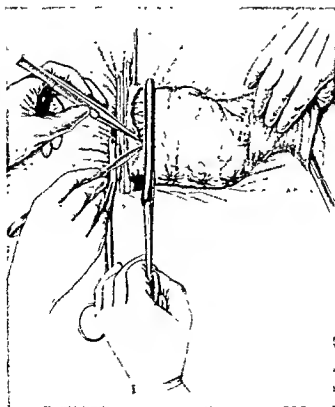


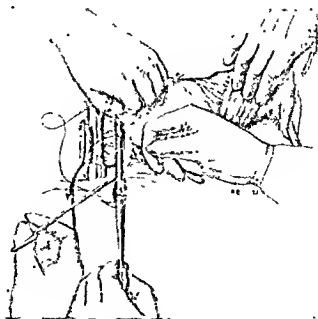
Fig. 312.

by tightening the screw. A non-slipping clamp is then placed on the stomach at the exact site where section is to be performed later. It is usually applied about 18-20 mm. from the gastric portion of the twin clamp.

The field of operation is now carefully isolated with gauze swabs and large packs. Packs are also placed over the handles of the clamps to prevent the sutures from becoming entangled. The twin clamps, besides being a great help in bringing together the portions of gut to be anastomosed, are hæmostatic, and thus care must be taken to loosen the blades temporarily just before the completion of the operation. If any bleeding points are seen these are underrun and ligatured.

*Fig. 312.* The stomach is turned over to the patient's left, exposing its posterior surface. An incision is now made close to the non-slipping clamp, through the serous and muscular coats of the stomach, starting at the lesser curvature and ending at the greater curvature. A sero-muscular cuff is dissected free for about 8 mm., care being taken not to injure any of the submucous vessels or puncture the mucous membrane itself.

*Figs. 313 and 314.* The continuous suture is inserted with a small Lane needle (cleft-palate needle), threaded with fine silk and held in my small needle-holder. The continuous suture picks up the sero-muscular layer of the stomach which has been separated, and the posterior surface of the duodenum which in this region possesses no



*Fig. 312.*

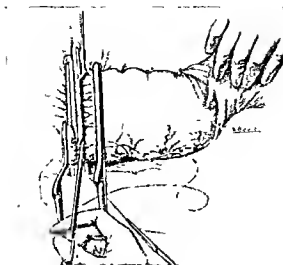


Fig. 314.

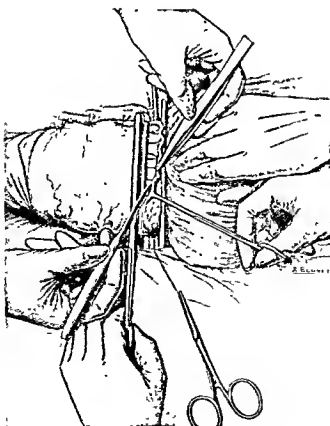


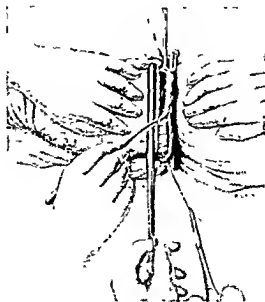
Fig. 315.

peritoneal covering. The suturing is carried out with the utmost care, the individual stitches being placed very close to one another. Pains must be taken to avoid perforating the lumen of the duodenum. If a little crystalline mucus is seen it often indicates that some of the Brunner glands have been pierced, but this is of no consequence. On the other hand, if the mucous membrane has been perforated there will be a bubble of air accompanied by a hissing sound. Such a puncture will have to be very carefully sutured before the operation is continued. The first stitch is carried to the top end of the duodenum, and after being tied it is cut short (see fig. 319).

*Fig. 315.* The stomach is now turned over to the right so as to expose its anterior wall. In a similar manner the anterior sero-muscular coat is separated from the underlying submucous coat for 8 mm. The incision here through these layers is placed as close to the non-slipping forceps as possible, and every precaution is taken not to injure the underlying submucous vessels or perforate the mucous membrane.

*Figs. 316 and 317.* The stomach is now opened by cutting through the entire anterior portion of the mucous membrane, as close as possible to the non-slipping forceps. This layer of the mucous membrane is carefully cleansed, and a large swab is placed into the space at the cut end of the stomach.

*Fig. 318.* The stomach is again turned over, this time to the left. This permits of the posterior portion of the mucous and submucous layers of the stomach being divided against the protecting swab. The



*Fig. 316.*

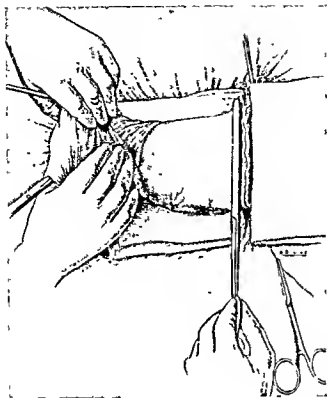


Fig. 317.

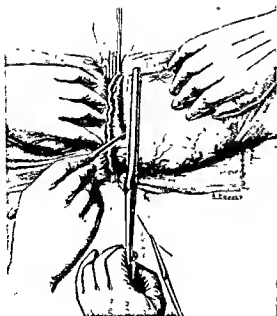


Fig. 318.

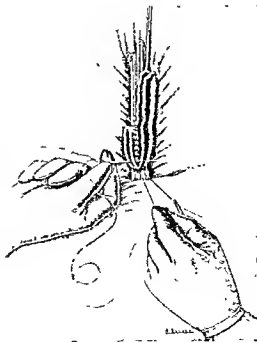


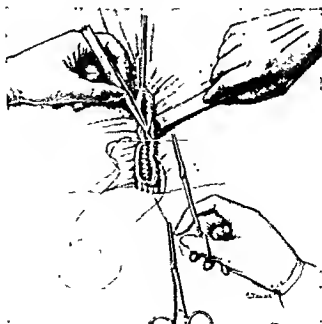
Fig. 319.

excised portion of the stomach is then covered with a large swab and removed.

*Fig. 319.* The size of the duodenal stump is now again gauged, as it is still possible to enlarge it even further if necessary by making a small slit, 4-6 mm. long, in its lower border. The next suture is again a continuous one. The needle is introduced at the lowest portion of the posterior wall of the duodenum, piercing all the coats, and then picks up, at the opposite side, the mucous and submucous coats of the stomach which lie in apposition to it. The suture is continued upwards until the top end of the duodenum is reached. Here the needle is made to transfix the mucous and submucous layers of the stomach, both of the anterior and of the posterior walls, as is clearly shown in figure 320.

*Fig. 320.* It will be noted that the stitch here is a continuous one and is introduced with a straight needle threaded with fine catgut. As stated above, it commences at the greater curvature, picking up on one side the whole thickness of the duodenum, and on the other side the mucous and submucous stomach layers.

When the upper end of the duodenal stump is reached, the suture picks up *both* mucous coats of the stomach, dividing this organ into two portions. The suture of these layers is a delicate procedure. The thread must be introduced firmly but not too tightly, so that it is hæmodynamic. When introducing the needle, pains must be taken not to perforate the submucous vessels of the stomach.



*Fig. 320.*

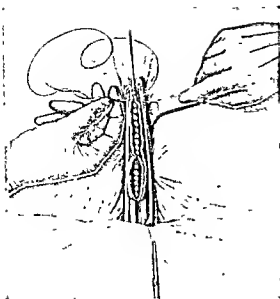


Fig. 321.

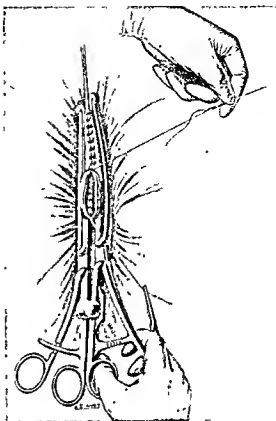


Fig. 322.

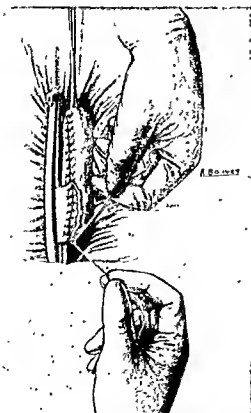


Fig. 323.

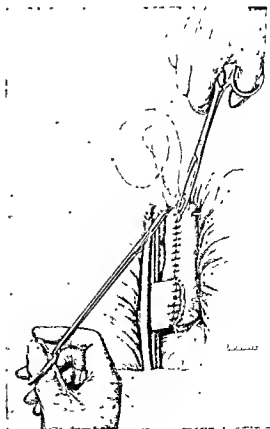


Fig. 324.



*Fig. 321.* The suture is now carried up to the lesser curvature, approximating both layers of the mucous and submucous coats of the stomach. Having arrived at the extreme end of the lesser curvature, the same continuous suture is carried downwards, picking up the sero-muscular coats of the stomach.

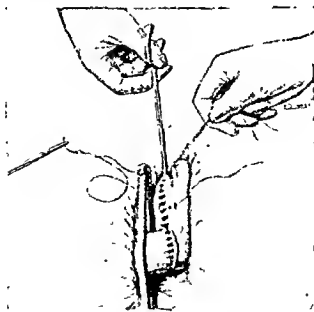
*Fig. 322.* It is customary at this stage for the assistant to loosen the gastric clamp, and to insert here and there a few interrupted sutures where any bleeding points are observed.

*Fig. 323.* When the duodenum is again reached, the whole thickness of the anterior lip of the duodenum is sewn to the anterior mucous and submucous layers of the stomach, still using the same continuous suture. At this stage the surgeon and his assistants change their cotton gloves.

*Figs. 327 and 329.* The anastomotic line in that portion of the stomach not attached to the duodenum is now invaginated by interrupted cross stitches. For the introduction of these sutures I use a curved needle, threaded with fine silk or cotton.

*Figs. 324, 325, 326, 327, 329 and 330.* These pictures show how these interrupted cross sutures are applied.

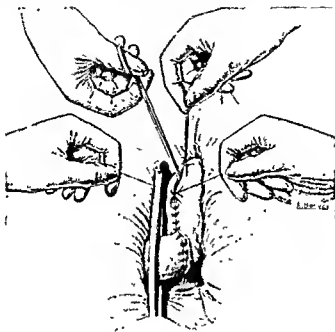
*Fig. 324.* The first cross stitch is placed on the lesser curvature, picking up the serous layer, a little distance away from the anastomotic line. The needle is then made to pick up a portion of the serous coat of the gut at a suitable point just posterior to the suture line. A double



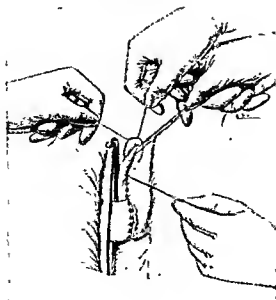
*Fig. 324.*

stitch is made with the thread, which is then tied off, the ends being left long.

*Figs. 325 and 326.* The *second* cross stitch is placed 3 cms. lower than the first. As this suture is tied, the assistant invaginates the gastric cone with Allis forceps, thus partially re-forming the graceful curve of the lesser curvature.



*Fig. 326.*



*Fig. 327.*

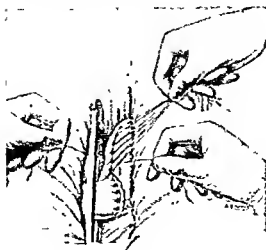


Fig. 328.

*Figs. 327 and 329.* A few intermediate sutures are similarly introduced until the sero-serous layers are neatly approximated. This is a delicate step in the operation, as the upper portion of the posterior wall of the stomach is thin and easily perforated with the needle. Such an accident should be repaired immediately by securely invaginating the punctured area.

*Fig. 328.* At the upper end of the anastomosis a "purse-string" suture, which picks up the serous layers of the stomach and duodenum, is applied, and when tightened this dangerous angle is neatly closed.

*Fig. 329.* This shows the interrupted cross stitches being applied to the anterior aspect of the anastomosis.

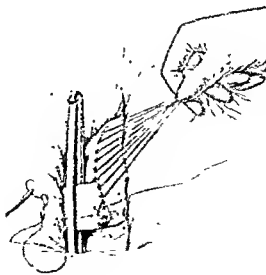


Fig. 329.

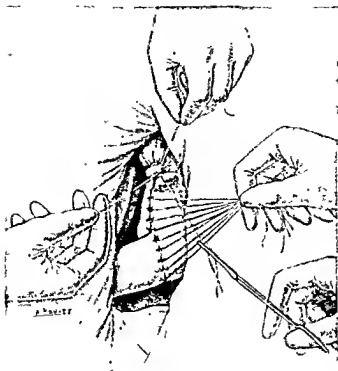


Fig. 330.

*Fig. 330.* As an additional precaution, the stump of the coronary artery, where the ligature threads have purposely been left long, is drawn downwards and tied by the first two cross stitches, so as to anchor it over the upper portion of the anastomotic line. All the long ends of the cross stitches which have been used as retractors are now cut short.

Before closing the peritoneum the suture line is carefully inspected to see if an additional stitch is required here and there. Having made quite sure that hæmostasis is complete, and that there is no oozing point to be seen anywhere, the abdomen is finally closed.

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I am deeply indebted to my friend, Dr. Enrique Finochietto, of Buenos Aires, a craftsman of the first order and a surgeon of wide repute, who, while on a short holiday to England visiting the surgical centres, at my request kindly consented to describe the foregoing operation for this book. He wrote this article in his native tongue—Spanish. It was then translated into English by Dr. Jorge L. A. Mulcahy, a personal friend of Dr. Finochietto, himself also an accomplished surgeon who holds several important surgical posts in Buenos Aires, including those of Senior Assistant Surgeon to Professor Dr. Pedro Chutro, and Surgeon to the British Hospital, and to the Ramos Mejia Hospital.—(EDITOR.)

SECTION 3

ULCER-CANCER OF THE STOMACH

by

F. A. KNOTT

### SECTION 3

## ULCER-CANCER OF THE STOMACH

BEFORE considering the clinically important points which fall under this heading it is essential to realise exactly what the term ulcer-cancer has come to mean. Nowadays ulcer-cancer denotes a carcinoma which arises from and in relation to a previously existing simple peptic ulcer, a condition clearly to be distinguished from a primary carcinoma which happens to undergo secondary ulceration—peptic or necrotic. Not that this distinction is always easy to make with certainty, either during examination of the patient or in the histological laboratory, but its existence must be emphasised in order to make quite clear that in the paragraphs which follow we are concerned only with that somewhat rare event, the development of carcinoma in a previously formed simple peptic ulcer.

### FREQUENCY

To physician and surgeon alike, the frequency with which carcinoma may be expected to develop in a chronic ulcer is of the utmost importance. Firstly, it concerns the length of time for which the physician may justifiably persevere with purely medical treatment. Secondly, it must influence the surgeon in deciding upon how extensive the operative measures should be.

In recent years there has been an increasing tendency among those with the best opportunity closely to study the histology of the condition, to conclude that true ulcer-cancer is a relatively rare condition.

It must be noted, however, that M. J. Stewart (1929-1931), in his admirably analysed series of post-mortem and operation specimens (by far the largest and most complete ever published), finds that ulcer-cancer occurs in just over 6 per cent of gastric ulcers, but remarks that this figure must be taken as the absolute minimum for the series concerned. Evidently the percentage is low, as would be expected from the known facts that the most common site of gastric carcinoma is at the pyloric end of the stomach, and that simple peptic ulcer is most commonly found on the lesser curvature. But clearly it is not so low that the clinician can justifiably neglect the possibility that an old-standing gastric ulcer may ultimately give rise to a malignant neoplasm.

Considerable importance, therefore, attaches to the means available for detecting the earliest signs of such change.

#### ACCESSORY DIAGNOSTIC METHODS

The possible signs and symptoms which may accompany ulcer-cancer, and the valuable assistance in its early detection which radiological examinations can give, are dealt with elsewhere (see page 1496). Here only laboratory methods will be discussed.

*Gastric Analysis.* The majority of patients in whom definite ulcer-cancer can be proved, give a relatively long history of digestive

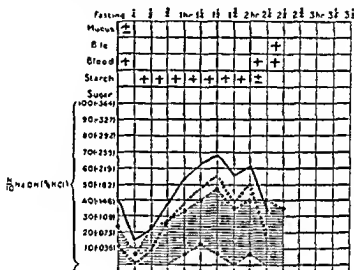


Fig. 351.—FRACTIONAL TEST MEAL IN ULCER CANCER.

Blood present in resting juice but no foulness and no lactic acid. Little fall in acidity since earliest tests.

disturbance corresponding to the existence of the initial simple ulcer. The usual findings of a gastric analysis in this condition have already been described in a separate section (see page 125). Whatever the level of gastric acidity shown in test meals performed during this earlier phase, the onset of carcinomatous changes in the ulcer does not greatly alter it.

The original ulcer and, therefore, the ulcer-cancer do not usually involve any great part of the acid-producing area of the gastric mucosa, and accordingly there is little or no tendency for the acid in the gastric juice to fall.

This is in marked contrast with the result usually obtained in gastric carcinoma undergoing secondary ulceration. With a much shorter history of digestive disturbance and a tendency to pyloric involvement,

this type of case usually shows, as noted elsewhere, definite changes in the test meal results as the condition advances. The gastric acidity gradually falls, often as far as complete achlorhydria. Coincident with the disappearance of free HCl, particularly if there is retention of food in the stomach, aciduric bacilli tend to develop freely with the production of lactic acid. It is in this type of case that we may expect to find the large volume of foul resting-juice which contains altered blood and, as Izod Bennett has emphasised, is so characteristic of gastric carcinoma. In true ulcer-cancer, although the resting-juice may contain altered blood, a foul specimen of very low or absent acidity is rarely found, unless the secondary carcinomatous change has

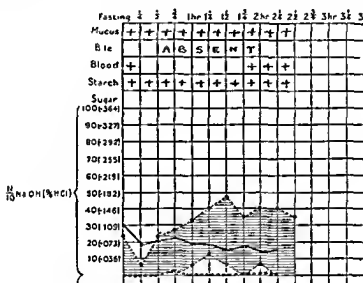


Fig. 332.—FRACTIONAL TEST MEAL IN PRIMARY CANCER.  
The resting juice foul and containing altered blood and lactic acid. Free acid absent.

become far advanced before the patient is examined: an uncommon event in view of the relatively long history.

There is, however, in this connection one small but important observation which may give rise to the suspicion that a known chronic ulcer has begun to undergo malignant degeneration. As will be mentioned later with reference to faecal occult blood, persistent altered blood in the resting-juice of an ulcer patient whose earlier test meal results have indicated only intermittent hleeding, i.e. only during relapses, should always suggest the possibility that malignant changes have commenced.

The more recently introduced methods of gastric analysis, namely histamine and alcohol test meals, have little or no advantage over the Rehfuß method as far as ulcer-cancer is concerned, except that very



slight persistent bleeding may be rather more obvious in the samples, particularly the earlier ones, owing to the absence of the solid matter of the gruel meal.

*Fæcal Occult Blood.* In the diagnosis of ulcer-cancer this test can be helpful only if special precautions are taken in its performance. As Hurst has insisted, the correct and only reliable way of making full use of the very delicate chemical and spectroscopical tests now available for detection of occult blood in the fæces is for repeated examinations to be made at fairly short intervals, the patient being placed before each test specimen is taken upon a hæmoglobin- and chlorophyll-free diet for at least three days. In this way changes in the amount of bleeding or its complete cessation can be detected with great accuracy. As a simple ulcer heals, the fæcal occult blood steadily diminishes and finally disappears. But Hurst has clearly proved from the records of his own cases that in the earliest stages of carcinomatous change in a gastric ulcer, though the radiological appearances may still be those of a chronic simple ulcer and the size of the crater become apparently greatly reduced as a result of medical treatment, yet however long this treatment is continued and however great the clinical improvement appears to be, the occult blood in the stools will persist. He very rightly holds, therefore, that in cases of chronic gastric ulcer, if at the end of one month's strict medical treatment fæcal occult blood is still present in undiminished amount, operation should be advised without further delay.

But when so heavy a responsibility is placed upon the results of a biochemical test, it is clearly necessary to be aware of the significance of any technical fallacies. They may be summarised briefly. The chemical tests (the guaiac and benzidine tests) are definitely more delicate than the spectroscopic tests (detection of acid hæmatin and acid hæmatoporphyrin). Mild hæmorrhage from the stomach typically results in a stool giving positive results with the chemical tests and one in extracts of which the spectrum of hæmatoporphyrin can be detected. The spectrum of acid hæmatin, a much less altered product of hæmoglobin, is found only when bleeding is profuse or comes from the colon and lower alimentary tract. Bell has shown that in normal people taking no hæmoglobin or chlorophyll in their diet for three days the spectroscopic tests always give negative results, whereas in a few the chemical tests remain weakly positive. These, however, also become negative on the fourth or fifth day of dieting. It is clear, therefore, that if the dieting has not been absolutely strict, it is best to rely, when

investigating the possibility of continued slight gastric bleeding, upon detection of the hæmatoporphyrin spectrum and to have an opinion on the chemical tests only if the patient is known to have taken no trace of hæmoglobin or chlorophyll by mouth for at least four days. Care must be taken to exclude possible bleeding from the gums.

On very rare occasions other possible sources of error might have to be considered. The hæmolytic types of anæmia and acholuric jaundice may produce traces of hæmatoporphyrin in the stools, but also give a positive result with the indirect van den Bergh test which, together with other blood examinations, can be used to exclude them. Patients showing the hæmorrhagic diathesis with thrombocytopenia and a tendency to bruising may also show persistent faecal occult blood, but here also examination of the blood will readily detect the condition.

*Macroscopical Appearances.* Turning now to the pathology of ulcer-cancer, but viewing it mainly in its clinical bearing, we must first consider the possibility of detecting the carcinomatous change by simple direct examination. In many of the recorded cases there are but slight changes visible to the naked eye. When examining a gastric ulcer, the main points to remember are that any secondary malignant change has usually an entirely local origin somewhere at the mucosal edge of the ulcer and that, as it progresses, this mucosa becomes thicker and more firm and nodular than that bordering the rest of the ulcer. Later, the thickening tends to extend, still in the mucosa, outwards from the ulcer and round the edge of the crater. At first there is strikingly little tendency for the ulcer floor to be invaded, and not until the mucosal thickening has extended completely round the ulcer, becoming meanwhile more and more obvious to the eye and to the touch, does general deep invasion of the stomach wall occur.



Fig. 333.—SECTION THROUGH ULCER-CANCER.  
There is a dense fibrous base to the old simple ulcer which had broken completely through the muscular layers. Secondary carcinoma has developed at the margins in the heavily shaded zones.

Even before the stomach is opened this special distribution of thickening may, subject to any limitations imposed by adhesions, be easily felt. Also inspection of the serosal surface and the finding of old chronic inflammatory reaction with fibrosis and scarring gives, in ulcer-cancer, a clear indication of the nature of the original lesion.

But it is when the ulcer is cut through transversely that the lines of development of ulcer-cancer, so admirably described by M. J. Stewart, are most clearly seen. If the carcinomatous change be still in a relatively



Fig. 334.—SECTION OF ULCERATED PRIMARY CARCINOMA. The cancer cells (black areas) deeply invade the submucous and muscular layers, but there is no gross break through and replacement by old scar tissue.

early stage, the typical structure of the original chronic ulcer remains clearly evident. It will have broken completely through the muscular wall which, from its distinctive tint, can be seen ending abruptly on either side of the bridge of granulation

and white fibrous tissue forming the base of the ulcer (fig. 333). This broken muscular wall seen in true ulcer-cancer stands in marked contrast with the condition usually met with in primary gastric carcinoma. Here, though it may be invaded by the neoplastic cells, the muscularis remains continuous and intact, never having been broken through and destroyed by an earlier ulcerative process (fig. 334).

The macroscopic section will also show more definitely the marginal mucosal thickening described above and in it the distribution of the carcinomatous tissue which, whiter and more firm than the rest, can be seen extending in the directions named.

*Microscopical Appearances.* The final proof that any thickening is truly carcinomatous must, however, come from microscopical examination, and here, in the detection of ulcer-cancer, certain particularly important fallacies must be noted.

In stomachs which have long been the site of chronic peptic ulceration it has been clearly shown by both Dible and Taylor as well as by Stewart that groups of misplaced epithelial cells may be found deep to the muscularis mucosæ. In a certain number of apparently normal stomachs this may occur, but when the deeper layers have been subjected to prolonged inflammation and fibrosis, the isolation of small groups of individually normal mucosal cells is not an uncommon occurrence. When examining an ulcer suspected of malignant change, the greatest care must be taken not to confuse these misplaced cells with cancer cells. It must be remembered that misplaced cells remain relatively true to type and usually show clear evidence of acinus formation. Moreover, although they may be below the muscularis mucosæ, and are sometimes found actually within the granulation

and fibrous tissue at the edge of the ulcer, they rarely invade the muscular wall of the stomach.

On the other hand, if the process be truly carcinomatous the cells may be found infiltrating the wall to any depth and showing their characteristic appearances. In true ulcer-cancer they clearly show the tendency for the normal epithelial cells to change into spheroidal and

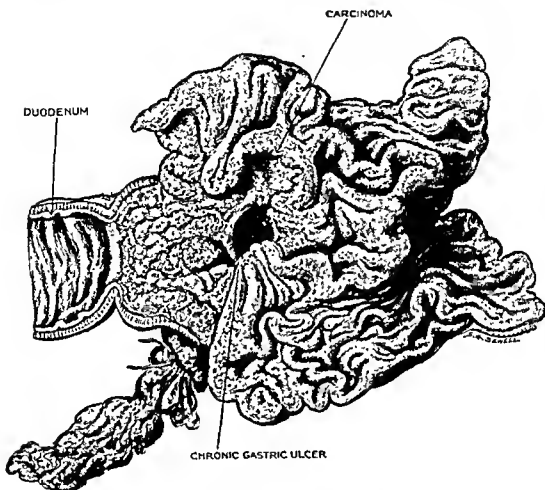


Fig. 335.—ULCER-CANCER OF THE STOMACH. GASTRECTOMY SPECIMEN. (Mr. Rodney Mairngot's case.)  
(Museum, Royal College of Surgeons)

polygonal types, and to disclose their augmented rate of growth by showing increased and irregular mitosis, hyperchromatic nuclei, and the formation of solid cell masses (figs. 336 and 337).

In the section of the ulcer itself we have, therefore, carefully to consider both the position and the nature of any suspicious cells. Before it can be confidently asserted that the case is one of true ulcer-cancer we have to obtain clear histological evidence of the pre-existing chronic ulcer as well as proof that the secondary change is truly carcinomatous. In a case of early neoplasm, the latter is the more difficult

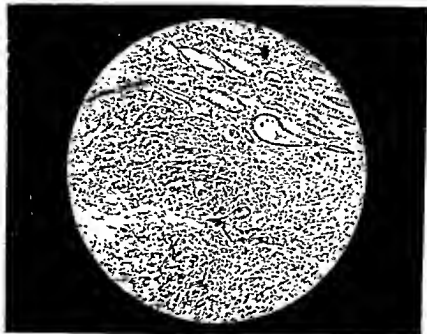


Fig. 227.—HIGHER MAGNIFICATION OF CANCER CELLS SEEN IN FIG. 225.



Fig. 226.—LOW POWER SECTION OF EDGE OF ULCER CANCER SHOWING POINT OF TRANSITION OF CELLS BETWEEN THE TWO CENTRAL UNIFORM MASS LINE.

to decide. When the malignant process is more advanced, its histology will be characteristic and the finding of metastases in the gastric glands may leave no doubt as to the presence of carcinoma. But at this late stage it is not so easy to demonstrate conclusively that the original lesion was simply a chronic ulcer. It is clear then that investigation of a suspected instance of ulcer-cancer calls for a good deal more than the cutting of a single section. The whole lesion must be examined with the greatest care so that both the original ulcerative and secondary carcinomatous processes can be separately and clearly demonstrated.

*Symptomatology.* See page 436.

*Treatment.* See page 414.

SECTION 4

MEDICAL TREATMENT OF PEPTIC ULCER  
AND OF HÆMATEMESIS

CHAPTER I

The Medical Treatment of Gastric, Duodenal and  
Anastomotic Ulcer

by

ARTHUR F. HURST

CHAPTER II

HÆMATEMESIS

A. Principles of Treatment of Hæmorrhage from Peptic Ulceration

by

R. SLEIGH JOHNSON

B. Treatment of Gastric and Duodenal Hæmorrhage

by

ARTHUR F. HURST

C. Choice of Operation in Cases of Hæmatemesis

by

RODNEY MAINGOT

## SECTION 4

# MEDICAL TREATMENT OF PEPTIC ULCER AND OF HÆMATEMESIS

## CHAPTER I

### THE MEDICAL TREATMENT OF GASTRIC, DUODENAL, AND ANASTOMOTIC ULCER

by

ARTHUR F. HURST

CHRONIC ulcers require prolonged treatment. The smallest ulcer rarely heals in less than four weeks, and large ones may require as long as three months. If suitable treatment is given for a sufficient period nearly every gastric ulcer eventually heals, as well as about 75 per cent of duodenal ulcers and 50 per cent of the much more intractable anastomotic ulcers which follow gastric operations.

The systems of treatment which give a different diet for each successive week are most illogical, as what is suitable for the first week remains suitable as long as healing is incomplete. The patient should be kept in bed and the strict regime should be continued without alteration until all spontaneous discomfort and all tenderness and rigidity have disappeared, no occult blood has been found in three consecutive stools by the most delicate form of guaiac test and by the spectroscope, and the X-rays show complete disappearance of the crater. When the ulcer has healed the patient can pass at once to an intermediate diet, which he should follow for two or three weeks; he should then begin the post-ulcer regime, which he should keep to for the rest of his life. No one develops a chronic ulcer unless he is born with a certain type of stomach, which is part of his physical make-up and remains unaltered as long as he lives. When, however, an ulcer has healed soundly and is not merely latent, as it is after the insufficiently strict and insufficiently prolonged treatment usually given, he is



unlikely ever to have a recurrence if he keeps strictly to the post-ulcer regime. There is no need for any restriction of exercise.

There is no doubt that the presence of free hydrochloric acid is an essential factor in the production of a peptic ulcer, and that healing is promoted by keeping the contents of the stomach neutral or nearly neutral for as great a part of the day and night as possible. This can be accomplished by means of a diet which calls forth a minimal secretion of gastric juice and by its alkalinity neutralises as much free acid as possible, and by giving olive oil and atropine before feeds in order to inhibit the secretion of acid, and alkalis between feeds to neutralise any free acid which appears in spite of these precautions.

A suitable diet should be completely fluid when swallowed and should not form clots in the stomach, it should be sufficiently nourishing to prevent loss of weight and in thin patients should lead to an increase in weight, and it should contain a sufficient supply of vitamins. The feeds should be given hourly. Milk should be the main ingredient. It is a very efficient alkali, as it neutralises an equal volume of gastric juice containing 0.3 per cent hydrochloric acid. If five ounces are given every hour, the gastric contents remain neutral during the greater part of the day. As the acid of the gastric juice curdles milk, which is further clotted by the action of rennin, sodium citrate should be added to it in the proportion of two grains to each ounce. Sodium citrate is a powerful alkali which neutralises any free acid which is present as well as making rennin inactive by combining with the calcium of the milk.

Milk of magnesia (*emulsio magnesice*), which contains five grains of magnesium oxide to the drachm, is another useful alkali, which should be given in just sufficient quantity to keep the bowels regular. In the unlikely event of any discomfort or heartburn being experienced with the diet and drugs already mentioned half to one teaspoonful of prepared chalk can be taken as often as necessary between feeds. It has the advantage of being insoluble, so that after complete neutralisation has occurred the excess of alkali is not absorbed, and there is thus less tendency for alkalosis to develop than with soluble alkaline salts. The popular triple carbonate powder has many disadvantages: sodium bicarbonate is the most powerful of all stimulants of gastric secretion and produces an enormous rise in acidity after the initial neutralisation, and bismuth oxy carbonate does not act as an alkali at all in the weak hydrochloric acid of the gastric juice.

It is important to avoid giving the excessive doses of alkalis sometimes recommended, as they frequently cause alkalosis. When a

patient with ulcer becomes irritable and complains of anorexia, nausea, and headache, it is almost certain that his blood urea has risen, and that the temporary omission of all alkalis will make him comfortable again, after which he should only be given half of his previous dose.

Individuals with chronic ulcer secrete large quantities of very acid gastric juice throughout the night; they should therefore be instructed to take additional feeds every time they wake.

Any active focus of infection should be removed when the patient has been about a fortnight under treatment, but more thorough treatment should be postponed until healing is complete. Pyorrhœa should be thoroughly treated, if possible by conservative means, and dead teeth with infected roots should be extracted. Infected tonsils should be enucleated, and sinusitis should be treated. In my experience the importance of intra-abdominal foci of infection has been much exaggerated, and it is very rarely necessary to remove either the appendix or gall-bladder apart from acute or sub-acute attacks, which are no more common in patients with ulcer than among the general public.

More important than foci of chronic infection are acute infections, as a recurrence of ulceration not uncommonly follows an acute infection, especially tonsillitis. The patient should be warned of the importance of going to bed at once at the onset of an acute infection and remaining at rest on an unirritating diet until recovery is complete. If more than one acute attack of tonsillitis should occur the tonsils should be enucleated.

It is impossible to go into every detail of treatment, but the following summaries give an idea of the strict treatment and the post-ulcer regime I use. Naturally, both have to be modified to suit each individual patient.

#### STRICT ULCER TREATMENT

*To be followed without alteration until healing is complete.*

(1) Every alternate hour from 8 a.m. to 10 p.m. 5 oz. of milk. This can be warm or cold and may be flavoured with tea.

(2) Every other hour, alternating with (1), from 9 a.m. to 9 p.m. a 5-oz. feed, which may be made of any of the following:

(a) Arrowroot, farola, Benger, junket, custard; to any of these red currant, apple or other fruit jelly can be added, and the junket may be flavoured with chocolate.

(b) At least two should consist of a thick soup or semi-solid purée of potato, artichoke, cauliflower or parsnip.

During the night the patient should have citrated milk by his bedside so that whenever he wakes he can take a feed.

(3) A rusk with butter should be eaten with three feeds. A "coddled egg" and some thin bread and butter may be taken once or twice a day by patients who can be relied upon to chew them thoroughly.

(4) Small quantities of water may be drunk between feeds. An ounce of strained orange or tomato juice should be taken with three or four of the drinks.

(5) One ounce of erecani should be added to the 11 a.m., 1 p.m. and 5 p.m. feeds, and  $\frac{1}{2}$  oz. of olive oil should be taken before the 9 a.m., 2 p.m. and 7 p.m. feeds.

(6) Ten grs. of sodium citrate in a teaspoonful of water should be added to each milk feed.

(7) One drachm of *emulsio magnesiæ* should be taken before feeds sufficiently often to keep the bowels regular.

(8) Atropine sulphate,  $\gamma\frac{1}{2}$  gr., in a drachm of water before the 8 a.m. and 3 p.m. feeds, and 2 drachms of the same mixture before the 10 p.m. feed. The dose should be increased by 10 minims every day until an unpleasant degree of dryness of the mouth or paralysis of accommodation occurs; the dose should then be reduced to that of the previous day.

(9) One drachm of prepared chalk half-way between feeds when any heartburn or discomfort is present.

(10) In chronic cases and especially with very large gastric ulcers  $\frac{1}{2}$  oz. of bismuth oxy carbonate should be given in a thick mucilage made of 1 dr. agar-agar stirred with 2 oz. of warm water.

(11) Wash the mouth out after each feed.

(12) No smoking during the strict treatment.

The patient should be weighed once a week. If he is too thin and has not gained weight, as often happens with gastric ulcers, the feeds should be increased to 6 or 7 oz. If he is too fat and has not lost weight, as sometimes happens with duodenal ulcers, the feeds should be reduced to 4 or 3 oz.

#### POST-ULCER REGIME

##### *To be followed permanently.*

Avoid alcohol except, if desired later on, a small quantity of light wine or diluted whisky at meals. Avoid effervescing drinks and coffee.

Avoid all pups and skins of fruit (whether raw, cooked or in jam, and currants, raisins and lemon-peel in puddings and cake), nuts, and all unripe fruit. For example, an orange may be sucked but not eaten. Currants, raisins and figs are particularly undesirable. Avoid all raw vegetables, whether taken alone (celery, tomatoes, cucumber, watercress), or in pickles and salad; green vegetables must be passed through a sieve and mixed with butter in the form of a purée. Porridge is only allowed if made with the finest oatmeal.

Avoid vinegar, lemon-juice, sour fruit; fried fish; pepper, mustard, curry, chutney, excess of salt; new bread; tough meat; pork, made-up and fried dishes, high game, clear and thick meat soup. During the first six months after recovery from an ulcer it is best to avoid butcher's meat altogether.

Take plenty of butter and cream, and a tablespoonful of olive oil before each meal.

Eat slowly and chew very thoroughly. An adequate time should be allowed for

meals, and rest for at least a quarter of an hour before and after meals. Meals must be punctual.

Do not smoke excessively. No smoking at all if any indigestion.

For the first six months a meal or feed should be taken at intervals of not more than two hours from waking till retiring, and again if awake during the night. The feeds should consist of a glass of the following mixture, which should be prepared each morning: 35 oz. of milk, 5 oz. of cream, and 120 grains of sodium citrate.

After six months of complete freedom a feed should be taken in the middle of the morning, on going to bed, and again if awake during the night, in addition to breakfast, lunch, tea, and dinner.

A teaspoonful of an "alkaline powder"<sup>1</sup> in a little water should at first be taken an hour after meals, but subsequently only when there is indigestion or heartburn. If the curve of acidity is high, the atropine mixture should be taken before meals.

The bowels should be kept regular by means of the magnesia in the alkaline powder, and, if necessary, liquid paraffin, but no other aperients should be taken.

Have your teeth attended to every six months.

Take no drugs in tablet form.

If you have the slightest return of symptoms, go to bed on a strict diet and consult your doctor, and do not wait for the symptoms to get serious

Pyloric obstruction is generally regarded as an absolute indication for surgery. Many cases, however, are caused by reflex achalasia or spasm resulting from a duodenal or, much more rarely, a pre-pyloric or even lesser curvature ulcer, together with congestion and oedema of the mucous membrane in the immediate neighbourhood of the ulcer. It is therefore always worth while trying the effect of medical treatment combined with evacuation of the stomach with a stomach tube every evening. Lavage is unnecessary, but no food should be given for three or four hours before the tube is passed. The quantity evacuated often becomes rapidly smaller; when it no longer exceeds 5 oz. the use of the tube should be discontinued. If the gastric stasis remains unaltered, organic pyloric obstruction must be present, but this preliminary treatment for two or three weeks renders the subsequent operation much safer, especially if at the same time plenty of fluid and salt are given by the rectum to overcome the dehydration and hypochloræmia.

<sup>1</sup> The "alkaline powder": 5 parts of prepared chalk with 1 part of light oxide of magnesia. The proportion should be altered according to the state of the bowels.

## CHAPTER II

### HÆMATEMESIS

#### (A) PRINCIPLES OF TREATMENT OF HÆMORRHAGE FROM PEPTIC ULCERATION

by

R. SLEIGH JOHNSON

THE complication of bleeding from a peptic ulcer has long provided a problem of treatment of unusual difficulty, in view of its frequency of occurrence and the urgent nature of its symptoms. Controversy in the past has prevailed in the main between the advocates of immediate operation and those of studied restraint and surgical inactivity, and while with progressive experience the issue is becoming clarified in the opinion of most of those best qualified to judge, there still remains a nucleus of dissension in favour of immediate radical measures. It may be of some value, therefore, before detailed consideration of different aspects and varieties of treatment, to review the principles underlying them, and to attempt by weighing up the pros and cons of each measure to lay down a working scheme of practical treatment.

The problem is simplified by separate consideration from the clinical standpoint according to the severity and frequency of the hæmorrhage. The commoner group is formed by those cases where hæmorrhage is of sudden dramatic onset, severe or torrential in degree, and danger to life demands immediate decisions as to treatment. The other natural sub-division consists of the cases where recurrent smaller bleedings occur, sufficient collectively to require special consideration because of their accentuation of illness and resultant anemia, but not individually serious in the sense of immediate risk to life.

The distinction between bleeding from an acute and that from a chronic ulcer is not one that can be made with any degree of safety or reliability on clinical data alone, at least in the circumstances attending a severe and sudden hæmorrhage. History is the only available evidence, may in any event be misleading, and, as elicited with difficulty from an exsanguined patient or his anxious relatives, must

be given proportionately less weight. Such a distinction attempted on clinical grounds should not, then, enter into the pros and cons of operation.

(1) *Acute Hæmorrhage.* An abrupt hæmorrhage from an ulcer is usually shown by hæmatemesis or melæna, or both, together with the general phenomena of collapse from rapid loss of blood, often assuming a condition of grave illness and anxiety. Such a state, other things being equal, would on fundamental principles be treated by absolute rest and the utmost conservatism with regard to active interference or disturbance of the patient. Loss of blood with accompanying fall of systemic pressure and increased coagulability directly favours arrest of hæmorrhage, while a condition of shock and collapse is of all states the least desirable for surgical procedures.

On the other hand, if it could be clearly shown that advantages accrued with certainty from surgical interference which were denied the patient by medical means alone, and which outweighed those of the latter in importance and results, then a policy of routine surgical intervention in such cases would be justified. Certain criteria of justification would, however, have to be fulfilled, of which the following are perhaps of chief import :

(a) *Diagnosis.* Although the most common, ulcer is not the sole cause of hæmatemesis, and its presence cannot necessarily be assumed, since some of the most severe instances arise from other lesions. These include bleeding from engorged and ruptured œsophageal varices in cirrhosis of the liver, or from splenic or gastric radicles, the subject of phlebitis in splenic anæmia ; rarely, severe hæmorrhage may occur in malignant disease of the stomach or neighbouring organs, in rupture of an aneurysm into the œsophagus, in acute toxic states with gastric erosion, in congestive heart failure, and in a number of primary blood conditions such as pernicious anæmia, the leukæmias and hæmophilia ; hæmorrhagic purpura forms yet another group.

In some of these cases the ætiology of the hæmatemesis will not be difficult to determine, but in others its origin will at the time be obscure. The patient is in no state for a detailed examination, history may be unreliable or unobtainable, and special investigations, such as X-ray or blood examinations, upon which normally the diagnosis is largely based, are for the same reason inapplicable, at least until a later stage of recovery. The clinical picture is the only guide and in a number of cases, therefore, uncertainty must prevail, for one or another of such causes apart from ulcer cannot definitely be excluded.

It not infrequently arises, then, that in approaching a case of hæmatemesis some doubt remains after examination and collection of all available data as to the presence or absence of an underlying ulcer. With the exception of purpura and (later) splenic anæmia, non-ulcer causes of hæmatemesis have no surgical indications—the immediate remedies are entirely medical and directed towards combating the hæmorrhage. Far from helping, indeed, surgery may well prove fatal, and the lack of certainty as to diagnosis provides, therefore, a cogent reason against operating in any case of severe hæmatemesis.

(b) *Comparative Mortality.* The respective merits of surgical and medical treatment are further influenced by a consideration of the risk and mortality accompanying each method. In the first place, although in ulcer cases death after hæmatemesis treated on medical lines is by no means negligible, it but seldom follows a single bleeding however profuse. Rather does the danger of fatality lie in a repetition of hæmorrhages at short intervals before recovery has taken place from the initial shock of a first severe attack. Apart from recurrence of bleeding, the majority of cases of hæmatemesis do become arrested by medical treatment. Comparing the medical with the surgical risk, the mortality rate, on the other hand, of immediate operation upon patients weak and shocked from gastric hæmorrhage, even with the added help of transfusion, is agreed by nearly all observers to be far higher than when medical measures alone are adopted. Were the operation itself technically a success in every case, which as noted below is an unreachèd ideal, the safer procedure as regards recovery would still lie in medical treatment.

(c) *Amenability of the Lesion to Surgical Measures.* The question may now be considered as to whether, assuming an operation be carried out, bleeding may thereby be arrested with certainty. The presence of hæmorrhage from the field of operation adds in no small measure to the difficulties in technique of gastric or duodenal surgery. Before operation it is an arbitrary point whether the ulcer is more likely to be gastric or duodenal, for although hæmatemesis is met with more often in the former, it is nevertheless still of such frequent occurrence with an ulcer in the duodenum as to be of no distinguishing value in diagnosis. It is, however, an indication that the rate of bleeding is rapid enough to produce an acute distension of the stomach and the resulting rejection of its contents. At operation not only may the exact source of hæmorrhage be obscured by blood and difficult to identify, but, when found, control may be no less difficult, the bleeding

point from its location and surroundings often not allowing of efficient ligature.

In a number of cases, moreover, a steady general ooze of blood may be found from a wide area of gastric mucosa. Where the site of bleeding is more restricted the offending vessel can sometimes be identified and tied and the bleeding arrested. More often it lies deeply buried in the floor of a penetrating ulcer and is difficult of access, while frequently the affected region is sodden and friable from inflammatory œdema around the ulcer, and attempt at ligature is frustrated by a prompt cutting-out of the stitches. It follows then that operation in such circumstances is at the best a hazardous and sometimes a blind undertaking, entered upon with no guarantee that bleeding can thereby be arrested.

In some cases the source of bleeding is found to be an acute ulcer, identified only after opening and searching the interior of the stomach and duodenum, the peritoneal covering not being involved. Even after exposure of the ulcer, an isolated point of bleeding may be equally difficult to find. It is in this type of lesion, moreover, that from its acute nature a ready healing on medical treatment may reasonably be expected. Bleeding from such an acute ulcer may be quite as severe as from one of many years' standing.

It is not disputed that in the hands of a few operators of unusual experience and skill successful surgical results under such difficult conditions may nevertheless be obtained. Considerations of technique and choice of operation are made elsewhere; briefly it may be noted that resection of the whole ulcer-bearing area as by a partial gastrectomy is advocated by some, by others a simple gastro-enterostomy or again ligature of the bleeding vessel. Granting these exceptions, from the standpoint of general policy, however, it must be conceded, and is in fact widely agreed, that judged by immediate operative results surgical intervention in acute hæmatemesis is unwise.

The factor of age in its association with arterial disease has been put forward by some authorities as a special indication for operating upon the elderly patient with a long-standing history of ulcer suffering from severe hæmatemesis. It is argued that under such circumstances vessels which are degenerate and sclerotic cannot be expected to contract and retract sufficiently for arrest of hæmorrhage and subsequent firm thrombosis. However this may be, the opposing argument holds good that the death-rate from operation in hæmatemesis rises steadily with increasing years and any hypothetical advantage in operating is outweighed.



From these several aspects, therefore, the conclusion may be drawn that in most hands surgical treatment in the presence of severe hæmorrhage from a peptic ulcer is unjustifiable, the danger to life from operation being greater than the danger from bleeding when left alone. The chance of recovery under medical measures is good, hæmorrhage usually becoming arrested, sometimes even after the patient's condition may have appeared hopeless.

Where the hæmorrhage fails to be controlled by medical treatment, it requires courage and conviction to pursue a path of apparent inaction. It is in such circumstances that surgery is sometimes undertaken as a last desperate measure from an illogical sentiment that "something must be done." Operation thus postponed until a state of exsanguination is reached becomes still more dangerous. Even when it is undertaken early the risk of fatality is increased thereby and the scale perhaps turned against an otherwise recovery.

*Transfusion.* The position with regard to blood-transfusion may now be considered. As with operation, this mode of treatment may either be employed as an immediate measure or delayed until severe bleeding has ceased.

(a) *Immediate Transfusion.* Blood-transfusion from a suitable donor may in some circumstances be clearly indicated in acute hæmatemesis or melaena of a grave order as a life-saving measure to frustrate death from hæmorrhage. It is not, however, to be lightly undertaken, being not entirely free from danger, while fallacious arguments have been brought to bear in its favour. It is to be remembered that bleeding in hæmatemesis subsides largely from a fall of systemic pressure and cardiac output, and that the fresh introduction of fluid, whether blood or saline, into the circulation has the counter-effect of raising the blood-pressure and thereby making recurrence of hæmorrhage more likely. Neither has it been proved that, on the grounds of increasing the coagulability of the blood, transfusion has an intrinsic hæmostatic effect, although hæmorrhage in itself may facilitate clotting in this way. As the chief danger of hæmatemesis lies in recurrence of bleeding, any measure likely to increase such a risk is, unless otherwise indicated, to be avoided. It is, moreover, not always possible to avoid a febrile reaction or even a rigor after transfusion, the general disturbance of which may in a sick patient be enough to endanger life.

In some cases, however, of torrential hæmorrhage the blood-volume may undergo so sudden and extensive a reduction that unless it be immediately restored death from collapse and anoxæmia will rapidly

ensue. Apart from general symptoms of blood-loss, a rough guide may be obtained from repeated estimation of the hæmoglobin percentage, which in severe cases should be made daily. Should the reading fall below 35 per cent, transfusion should be carried out without delay, and, if necessary, be repeated, although bleeding be still in progress. It is to be remembered that even after hæmorrhage has ceased a further drop in the hæmoglobin content may be found merely from dilution of the blood-volume with body-fluids, although the clinical condition has improved. Thereafter a steady rise in the hæmoglobin and red cell count should be noted, accelerated by giving iron.

(b) *Delayed Transfusion.* The routine employment of blood-transfusion in acute hæmatemesis has thus but little in its favour. The greater value of transfusion lies in its reservation until an interval after gross bleeding has ceased, when the blood picture may be restored without risk of recurrence of bleeding. Cases so treated do better than those transfused during or directly after the hæmorrhage. Even a small transfusion at this stage will by stimulation of the bone-marrow promote recovery of strength and accelerate healing of the ulcer.

Transfusion has similarly an important place in the treatment of chronic or recurrent hæmorrhage, as will be considered below.

To summarise, the acute hæmorrhage from ulcer remains the province of the physician. Such a statement perhaps needs the qualifying remark that medical means, if to be entrusted, must not be half-hearted. They imply absolute and complete rest of the patient, physically, mentally, and locally of his gastric functions, motor and secretory, by the various means described in the section on medical treatment (see page 584).

If the case is on other grounds considered ultimately suitable for surgery, the time for this procedure is after the stage of recovery, when by careful medical means, including transfusion where necessary, the patient is restored to a condition where operation may safely be carried out. Normally this interval should be not less than three months.

(2) *Chronic or Recurrent Hæmorrhage.* Here the problem between medical and surgical treatment is a simpler one, although the same general principles hold good. In the greater number of cases of hæmatemesis, a prompt and adequate regime of medical treatment will control the hæmorrhage and avoid its recurrence. In a proportion, however, despite complete rest and appropriate dieting small recurrent losses of blood continue, which while not individually alarming delay recovery and lead to a considerable degree of secondary anæmia and consequent ill-health.

The question again arises whether it is to the patient's best advantage to adhere to medical treatment or to consider surgical intervention, and if so whether this should be immediate or delayed.

With regard to the first point, persistence of bleeding may be taken to indicate continued activity of the ulcerative process or possible malignancy. Such a failure of the ulcer to heal in spite of proper medical treatment carries, moreover, the risk of a further and more severe hæmorrhage at any time. If then the losses of blood have been sufficient in amount or frequency to maintain a state of definite anæmia, and operation is not contra-indicated on other grounds, their persistence is a call for surgery. Removal where possible of the entire lesion is the best safeguard against each of these dangers.

In the choice of time for operation the same considerations will apply as in dealing with the acute case. Surgical intervention at the actual time of bleeding is equally undesirable. The lesser urgency of the case, moreover, will give correspondingly less weight to the cry for haste and more readily permit of a safe choice of time for operation.

The immediate treatment will therefore be medical, supplemented by iron, and where indicated by blood-transfusion after the cessation of bleeding, operation being carried out after an interval when as a result of these combined methods the patient has been restored to the best possible condition to withstand it. When this state is once reached, operation should not be longer delayed, as without surgical treatment the liability to recurrent bleeding persists, clinical records showing that cases with a number of hæmatemeses in the past usually continue to bleed.

As already noted, it is in the recurrent type of hæmatemesis that blood-transfusion is of the greatest help in treatment. A number of small transfusions, carried out at intervals after gross bleeding has ceased, will so aid improvement of the general state that, supplemented perhaps by a further transfusion during the operation, a major surgical procedure for the radical treatment of the ulcer may then be undertaken with comparative safety.

## (B) TREATMENT OF GASTRIC AND DUODENAL HÆMORRHAGE

by

ARTHUR F. HUEST

COMPLETE immobility is the first essential in the treatment of gastric and duodenal hæmorrhage. Immobilisation of the patient keeps the

blood-pressure down ; immobilisation of the stomach prevents a newly formed clot from being dislodged. The patient is kept quiet in bed and reassured about his condition—a very important point too often neglected, especially in hospital. He is given  $\frac{1}{6}$  gr. of morphine hydrochloride subcutaneously to prevent mental and physical restlessness together with  $\frac{1}{100}$  gr. atropine sulphate to inhibit the secretion of gastric juice. No food or drink is given for 48 hours. During the period of starvation it is very important to keep the mouth absolutely clean, as otherwise parotitis may develop. The patient should be given 15 ounces of normal saline solution every six hours by rectum, and the colon should be kept empty by means of a daily enema of plain water. If much dehydration occurs, saline solution should be given subcutaneously. There is no object in giving ice to suck.

Hæmostatic serum, calcium, and other drugs given with the object of promoting coagulation are quite useless. A drachm of tribasic magnesium phosphate in a small quantity of water should be given every three or four hours in order to neutralise the free acid present in the stomach and so prevent the digestion of the clot.

The hæmoglobin percentage should be estimated daily. If it falls below 40 the patient should be transfused without delay. The blood-pressure should be measured at least twice daily, as a fall in blood-pressure precedes a fall in hæmoglobin when bleeding occurs. It may be necessary to repeat the transfusion two or three times. The slight rise in blood-pressure does not increase the danger of recurrent hæmorrhage. It should be remembered that death from hæmorrhage is due to anæmia, and it should always be possible to prevent this except in the rare cases in which death occurs actually during the first hæmorrhage, generally owing to the erosion of one of the main gastric or duodenal vessels. In the exceptional cases in which a sclerotic artery incapable of contracting has ruptured, transfusion tides the patient over sufficiently long for it to be possible to deal directly with the ulcer by operation.

Blood is as much food to the stomach as a meal, and its presence in the stomach leads to active secretion of gastric juice and peristalsis. The rapid distension of the stomach in severe hæmorrhage results in its evacuation by hæmatemesis, and smaller quantities pass out through the intestines and give rise to a mælena. But when severe hæmorrhage continues after hæmatemesis has occurred or when the bleeding is not sufficiently rapid to cause the sudden distension which results in its ejection, death may result unless steps are taken to empty the stomach. Just as uterine hæmorrhage can be arrested by emptying the uterus

and causing it to contract, so evacuation of the stomach with a tube and repeated lavage (with four ounces of ice-cold water until it comes back no longer blood-stained) almost invariably leads to cessation of gastric hæmorrhage. When the water has finally been evacuated a drachm of 1 in 1000 adrenalin chloride is passed into the stomach; it can now gain access to the bleeding point, which it cannot do when the stomach is full of blood. As it is not absorbed it does not cause any rise of blood-pressure. Although it requires courage to give this treatment to a desperately ill patient, I have seen several cases in which I am sure that it has saved lives. It is particularly valuable when the hæmorrhage is from an acute ulcer, in which no operation can be of any use.

When there has been no bleeding for 48 hours the usual treatment of a chronic ulcer by hourly feeds, alkalis, and atropine should be begun. When there have been no symptoms pointing to the presence of a chronic ulcer the alkalis and atropine should be omitted, but the strict diet should be continued until the occult blood has disappeared from the stools. This generally occurs much more rapidly than with a chronic ulcer.

In all cases 30 grains of iron and ammonium citrate should be given three times a day as soon as feeding is begun, as although the spontaneous regeneration of blood is often rapid, in some cases it is extremely slow, and, although the ulcer may heal, the patient may remain unfit for many months if the anemia is not recognised and treated.

### (C) CHOICE OF OPERATION IN CASES OF HÆMATEMESIS DUE TO PEPTIC ULCERATION

by

RODNEY MAINGOT

I AM in agreement with the principles of treatment of hæmatemesis due to peptic ulceration as outlined in the preceding pages.

In a case of severe hæmatemesis known to be due to a chronic peptic ulcer, in my opinion operation should not be performed *during a severe bout of bleeding or immediately afterwards* when the patient is collapsed and exsanguinated. But the question as to *when* operation should be performed is so frequently raised that it is essential to have some plan of action definitely in mind with regard to such cases.

As the exsanguinated patient is a very bad operative risk it is usually wiser to defer surgical measures until he has recovered from the effects of his hæmorrhage, and this may entail a lapse of weeks or even months.

The alternative method of giving blood-transfusions directly after a torrential hæmorrhage and operating immediately is not one which appeals to me. On the other hand, the persistent recurrence of mild attacks of hæmorrhage is a clear indication for operative interference, always provided that a diagnosis of peptic ulcer has been established beyond dispute, and that intensive medical treatment has been given a fair trial.

*Operation.* The abdomen is explored through a paramedian or preferably a mid-line incision. In cases of hæmorrhage due to a *chronic duodenal ulcer* the following operations have been practised :

(1) Underrunning and ligature of the blood-vessels in the region of the pylorus and first part of the duodenum.

(2) Opening the pylorus and anterior wall of the duodenum to expose, and cauterise or otherwise obliterate the ulcer.

(3) Gastro-jejunostomy combined with (1) or (2), or with :

(a) Pyloric occlusion, or

(b) Infolding of the anterior wall of the duodenum in such a manner as to produce stenosis and plugging of the ulcer bed.

(4) Attack upon the ulcer combined with gastro-duodenostomy.

(5) Finsterer's operation of *partial gastrectomy with pyloric occlusion*, nothing being done to the ulcer itself.

(6) Gastro-duodenal resection *with duodenal occlusion*. Here the ulcer is resected together with the first part of the duodenum and at least three-quarters of the stomach, the operation being completed by either the posterior or anterior type of Polya anastomosis.

In practice the actual operative procedure will in large measure depend upon the state of the patient, the condition of the ulcerated area, and the technical skill of the surgeon.

Such measures as underrunning and ligature of the blood-vessels in the involved region or direct attack upon the ulcer, neither method being combined with any form of short-circuit operation, will rarely have more than a temporary effect in arresting the hæmorrhage.

If the patient's condition is poor it is my practice, and that of many

other surgeons, merely to infold the first part of the duodenum as efficiently as possible, occlude the pylorus with kangaroo tendon, and perform a posterior gastro-jejunostomy.

If, however, the patient's condition is satisfactory and the parts lend themselves readily to excision, the radical operation of gastro-duodenal resection is to be preferred as it is the only method likely to be followed by permanently satisfactory results, at the same time ensuring freedom from secondary peptic ulceration and recurrence of the hæmorrhage.

In certain cases where the ulcer is posteriorly placed, inaccessible, deeply excavating the pancreas, and surrounded by œdematous adhesions, Finsterer's operation of partial gastrectomy with pyloric occlusion would seem to offer a fair prospect of cure.



Fig 234. PYLORIC OCCLUSION BY MEANS OF KANGAROO TENDON.

In cases of *gastric ulcer* the ideal treatment is partial gastrectomy, and this operation should be carried out wherever possible.

An alternative to gastrectomy is wedge resection of the ulcer combined with gastro-jejunostomy.

Any *indirect* operation, such as pyloroplasty or gastro-jejunostomy for a bleeding gastric ulcer, is unsound in principle and often doomed to failure. There must be a *direct* attack upon the ulcer, and partial gastrectomy fulfils this requirement in the most satisfactory manner.

If on exploration of the abdomen in a case of severe hæmatemesis the stomach and duodenum appear to be normal, there being no localised induration or crater to be felt which would indicate the presence of an ulcer, no useful purpose will be served by opening the stomach or duodenum and searching for the bleeding point, as, if this is attempted, more often than not the whole mucous surface will

be found to be weeping blood. In these cases, where no visible or palpable organic lesion is found in the stomach or duodenum, neither partial gastro-duodenal resection nor a short-circuit operation is, in my opinion, justifiable. It is better to carry out a careful examination of the other abdominal viscera, such as the gall-bladder, the appendix, the spleen, etc., to ascertain whether there is any pathological condition in any of these organs to account for the gastrostaxis. If any gross disease is found here, the surgeon should not hesitate to deal radically with the condition present.

During and after operation every means of resuscitation should be readily available.



SECTION 5  
DUODENUM AND SMALL INTESTINE  
by  
J. EWART SCHOFIELD

CHAPTER I  
Tumours of the Duodenum and Small Intestine

CHAPTER II  
Injuries to the Small Intestine

CHAPTER III  
Chronic Duodenal Ileus

## SECTION 5

### DUODENUM AND SMALL INTESTINE

#### CHAPTER I

##### TUMOURS OF THE DUODENUM AND SMALL INTESTINE

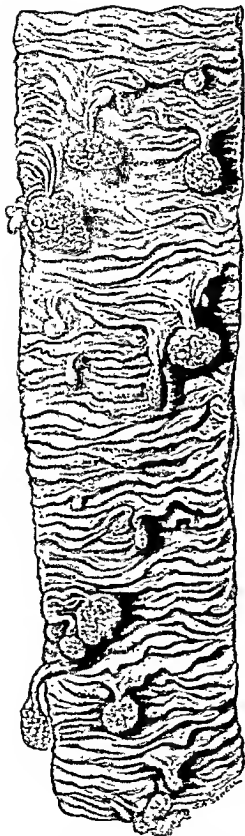
SMALL intestinal neoplasms are seldom met with in surgical practice, and their rarity can be judged by the paucity of recorded cases. Every student of surgery is taught that carcinoma, which incidentally is the commonest of small intestinal tumours, practically never occurs in the small bowel. There are very few surgeons, moreover, who, when investigating a vague abdominal condition, would include these tumours in their differential diagnosis. Growths in this situation, therefore, are liable to be regarded as pathological curiosities of only academic interest. Thus they remain, until the occasion arises when the surgeon is suddenly confronted by one of them, undiagnosed, at operation. The tumour then ceases to be of mere theoretical interest, assumes a practical importance, and demands immediate attention.

##### BENIGN TUMOURS

Benign tumours are to be found more frequently in the large than in the small intestine. A comprehensive survey of these tumours was made by King, who was only able to collect 119 cases in literature. Of these, 47 were found in the small intestine.

In 1933, Rankin and Newell published a list of 35 cases of benign intestinal new growths, which comprised the total number of these cases appearing in the files of the Mayo Clinic. They were arranged as follows :

Adenoma	.	.	.	.	.	.	11
Myoma	.	.	.	.	.	.	11
Fibroma	.	.	.	.	.	.	6
Lipoma	.	.	.	.	.	.	2
Hæmangioma	.	.	.	.	.	.	2
Indefinite	.	.	.	.	.	.	3



The rarity of these benign tumours is demonstrated by the fact that Rankin and Newell, in collecting 35 cases, were able at the same time to find 60 cases of small intestinal carcinoma.

Allowance must be made, however, for a number of cases which undoubtedly, on account of the absence of symptoms, remain unobserved, so that in all probability they are of more frequent occurrence than statistics would lead one to believe.

*Pathology.* The commonest of these tumours in order of frequency are adenoma, myoma, fibroma, lipoma, hæmangioma. Other extremely raro forms occur, such as myxoma, myxofibroma, neurofibroma, endothelioma, and teratoblastoma, but only isolated cases of these have been described. Such tumours affect all parts of the small bowel, but the ileum is the commonest site. They may be subserous or submucous, but they usually grow in the direction of the lumen, which is probably due to the fact that the mucosa offers less resistance than the muscular coats of the bowel.

They vary greatly in size from that of a pea to that of a tumour which may be palpated through the abdominal wall. They may be

Fig. 339.—MULTIPLE ADENOMATA OF JEJUNUM.  
NUMEROUS SPHEROIDAL TUMOURS RISE FROM THE  
VALVULE CONJUGATES BY NARROW PEDICLES.  
(*"British Journ. Surg."* Hunterian Museum, R.C.S.)

single or multiple, and in the latter case may affect a considerable portion of the intestinal tract. This is best exemplified in the condition of multiple polyposis.

Finally, the tumours may be sessile or pedunculated, the submucous variety taking on the latter form. Of these growths, the adenomata and lipomata usually become pedunculated. The wall of the intestine is composed of a variety of tissues, viz., the outer peritoneal coat of endothelium, unstripped muscle, areolar tissue, and mucous membrane



Fig. 340.—TYPICAL ADENOMATOUS POLYP OF THE ILEUM,  
COMPOSED OF MUCOUS GLANDS. ( $\times 115$ .)  
(Rankin & Newell. "Surg., Gynec., Obstet.")

with vascular supply, from which, with the exception of the outer endothelial coat, growths may arise.

They have all the microscopical characteristics of similar tumours found in other parts of the body.

*Adenomata.* Although the commonest benign tumours of the small intestine, they are found more often in the large intestine. They arise from the intestinal glandular epithelium, and usually grow towards the lumen. They occur in all parts of the small bowel with a slight predilection for the duodenum, where they have a second site of origin

from Brunner's glands, a fact which can be demonstrated under the microscope. Adenomata frequently become pedunculated, and a few instances of malignant change supervening are known, although this is an extremely rare complication. There is a tendency for these tumours in their polypoidal form to disappear spontaneously.

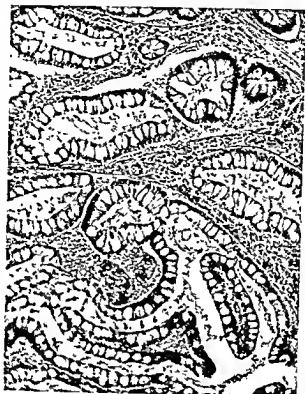


Fig 341.—ADENOMA OF BRUNNER'S GLANDS. ( $\times 115$ ).  
(Rankin & Newell. "Surg., Gynec., Obstet.")

*Myomata.* Under this heading are included the fibromyomata, their nomenclature depending on the relative proportions of muscle and fibrous tissue present. All portions of the small intestine are affected, and these tumours have an equal distribution in both large and small bowel. They arise from the muscle coat of the intestine, rarely become pedunculated and are subserous and submucous in equal proportion. They contain smooth muscle-fibres, and a salient feature of their microscopical appearance is the presence of large numbers of red blood-corpuscles. Small intestinal myomata show a marked tendency to bleed, and may be the cause of serious intestinal hæmorrhage.

*Fibromata.* Pure fibromata rarely occur, as usually there is sufficient fibrous tissue present to warrant the term fibromyomata.

They spring from the connective tissue of the submucous coat of the intestine, tend to become pedunculated, and are prone to myxomatous or chalky changes. Fibromata are most commonly found in the ileum, and of all the benign tumours are the most likely to give rise to intussusception.

*Lipomata.* They are probably commoner than the above statistics of Rankin and Newell show. They tend to become pedunculated, and cases have been reported in which spontaneous expulsion of the tumour has occurred per rectum. A yellow tinge is often imparted to the mucous membrane or peritoneal coat by the presence of the underlying growth. They are often multiple, and of all the benign tumours are the most susceptible to rapid growth, sometimes attaining a size sufficient to allow them to be palpated through the abdominal wall.

*Hæmangiomata.* These tumours are more often met with in the small than in the large intestine. They appear reddish in colour under the mucosa and blue under the serous coat. They are formed of sinus-like spaces containing blood, lined by a definite layer of endothelium. Rankin and Newell's cases both occurred in the duodenum, one giving rise to serious gastro-intestinal hæmorrhage, the other growing so large as to occlude the lumen of the bowel. MacCullen in 1906 reported a case of multiple cavernous hæmangiomata occurring throughout the length of the small intestine which were most marked in the jejunum. Saint has also described a case of small multiple hæmangiomata occurring in the jejunum.

*Glandular Hyperplasia.* This condition, though not strictly coming within the classification of benign tumours, should be mentioned. According to Saint it is probably the commonest cause of polypi of the small intestine. The pedicles are extremely small, being 1-2 centimetres in length. Saint, in his study of this condition, believes it to be one of local hyperplasia, probably of the lymphoid follicles of the intestinal mucosa. The polypi are most commonly found in the ileum. Clinically this condition is unimportant.

*Diagnosis.* Many cases of benign intestinal tumour remain undiagnosed; they may be found accidentally at operation or at autopsy. Undoubtedly there must be a number of cases which remain undiscovered.

Vague abdominal symptoms such as epigastric pain, vomiting,

nausea and indigestion may occur, mimicking many other abdominal conditions, e.g. disease of the gall-bladder or appendix, duodenal ulcer, constipation, etc. These symptoms by themselves contribute little to the diagnosis of benign tumour. Possibly a diagnosis of one of the above-mentioned conditions may be made, and a tumour be found at operation. Rarely does the tumour grow so large as to be felt on physical examination.

It has been claimed that a few cases have been diagnosed as a result of X-ray examination and more particularly cases which have occurred close to the pylorus or the ileo-cæcal valve. On the whole, however, the results are most unconvincing, and little help can be expected in this direction.

It must be admitted, therefore, that the diagnosis of these tumours is a difficult matter; actually it is rarely made except in the presence of a complication.

*Benign Tumours of the Duodenum.* The general symptoms of epigastric pain, vomiting and indigestion may be present, and if there is ulceration of the mucous membrane over the tumour, a diagnosis of duodenal ulcer is likely to be made. Should the ulceration be severe, serious hæmorrhage may occur resulting in hæmatemesis, melæna and anæmia. This is more likely to occur should the tumour be a myoma. This complication, however, would only tend to strengthen the diagnosis of duodenal ulcer. Should the tumour grow to such a size as to cause obstruction, the true nature of the disease may come to light. The case would probably be regarded as one of pyloric stenosis, and if the growth occurred below the ampulla of Vater, the patient would vomit large quantities of bile. This important diagnostic factor together with an X-ray examination may result in unmasking the condition.

The most valuable complication as an aid to diagnosis in tumours of the small intestine is undoubtedly the onset of intussusception. Owing to its fixity this rarely occurs in the duodenum. Kellogg, however, has recorded a case of intussusception which occurred in the duodenum as a result of adenoma arising in Brunner's glands.

*Benign Tumours of the Jejunum.* Tumours in this region may give rise to no symptoms at all; but, as in the duodenum, vague abdominal symptoms may be present which are common to other abdominal conditions.

Bleeding may occur resulting in melæna, and occasionally bright red blood is passed per rectum.

The tumour may grow to such a size as to cause chronic obstruction. The patient suffers from threatened attacks of acute intestinal obstruction which subside temporarily, only to recur later.

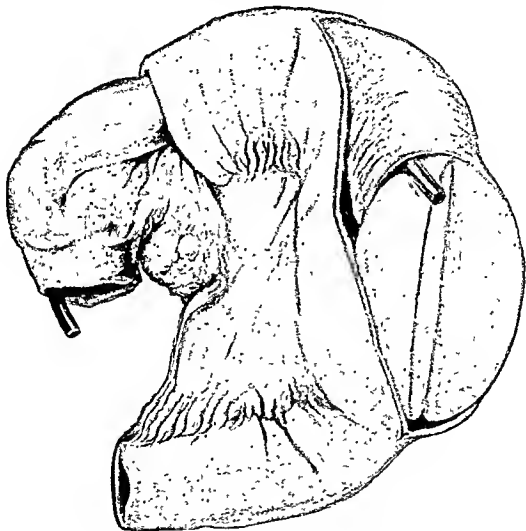


Fig. 342.—INTUSSUSCEPTION DUE TO FIBROMA OF SMALL INTESTINE.  
(*Hunterian Museum, R.C.S.*)

The complication responsible for unmasking many of these tumours is intussusception. The commonest cause of intussusception in the adult is benign small intestinal tumour. Other causes are: typhoid ulceration, including the period of convalescence, dysentery, stercoral ulceration, tuberculosis, Meckel's diverticulum, foreign bodies, parasites and intussusception without any known cause. Of these tumours which produce symptoms, the percentage in which intussusception occurs is considerable.



The patient, who may previously have been quite healthy, is seized with acute abdominal pain with dramatic suddenness. The pain, usually located in the epigastrium, is described by the patient as a cramp or a knot in the pit of the stomach. Vomiting is severe.

The abdomen is distended and a lump can usually be felt. Constipation is marked, but blood may be passed per rectum. These attacks may be severe enough to bring the patient to the surgeon, or they may pass off suddenly, only to recur later. Intussusception may in some cases be found on opening the abdomen for some other condition.

A typical feature is the sausage-shaped tumour which is nearly always present and which becomes visibly larger with each successive attack of colic. Unlike intussusception in the infant, the condition is not necessarily progressive, the intussusciptens remaining viable for some time. Cases have been recorded of a spontaneous cure following the passage of a slough per rectum.

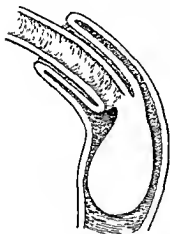


FIG 342.—DIAGRAMMATIC REPRESENTATION OF FIG. 342 REDUCED TWO THIRDS.

*Treatment.* If a small tumour is present, local excision will probably be all that is required. If the growth is a subserous one, an attempt should be made to shell out the tumour through a longitudinal incision, if possible without dividing the mucous membrane. The bowel should be closed with a transverse suture line.

The tumour may be large enough to render resection necessary with end-to-end anastomosis, or closure with lateral anastomosis.

The results of these operations are most encouraging. Rankin and Newell report that in their series of 35 cases there were no deaths as a result of operation. In some cases enucleation was performed; in others resection; and in following up the results, the permanence of cure by surgery was demonstrated. Balfour and Henderson treated six cases of benign duodenal tumour by excision and all the patients did well. Some surgeons prefer in addition to perform gastro-enterostomy in duodenal tumour. In those cases complicated by intussusception the treatment depends on the severity of the intussusception. It may prove to be easily reducible and local excision may be all that is necessary, aided perhaps by lateral anastomosis. On the other hand, the

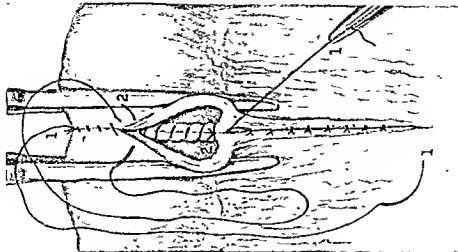


Fig. 316.—Return Intra Suture (All Coats)

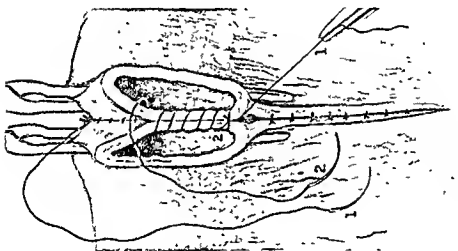


Fig. 315.—Intra Suture (All Coats)

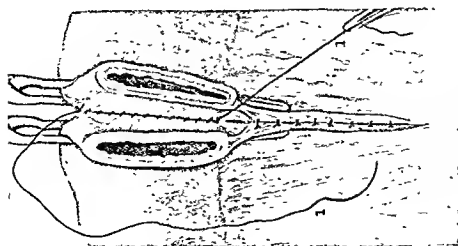


Fig. 314.—End to End Anastomosis of Small Intestine. Suture of Posterior Sphincter Muscular Suture. Suture of Posterior Layer of Mesentery.

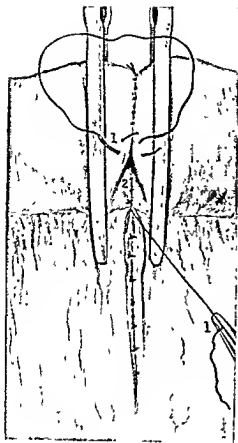


Fig. 347—ANTERIOR SERO-MUSCULAR SUTURE.



Fig. 348—ANASTOMOSIS COMPLETE. SUTURE OF ANTERIOR LAYER OF MESENTERY.

surgeon may prefer to resect a portion of bowel, but each case must be treated on its merits. Usually, however, it is not possible to obtain complete reduction. This should not be too energetically attempted, as damage may be done to the delicate bowel, and much valuable time wasted. It is better to reduce the intussusception as far as safety will allow, and then to resect the unreduced portion followed by anastomosis.

#### MALIGNANT TUMOURS

*Carcinoma.* Small intestinal carcinoma constitutes only 2 to 3 per cent of all intestinal cancer. This is a curious fact when one considers the relative frequency of cancer of the stomach and large bowel. If constant irritation were accepted as a predisposing cause of cancer, then this discrepancy might well be explained by the fluid content of the small intestine proving less irritable than the more solid contents of the stomach and colon.

With regard to regional incidence, the duodenum is most commonly affected, the ileum to a lesser degree, while jejunal cancer is practically unknown. Johnson, in a report from the Vienna General Hospital, found 343 cases of intestinal cancer in 41,883 autopsies, but not a single case of jejunal cancer was included. Of 71 cases of small intestinal carcinoma, Jefferson found the duodenum to be affected in 34, that is 48 per cent, and states, "Considering the shortness of the duodenum it is evident that, inch for inch, it is more liable to carcinoma than the rest of the small intestine."

*Carcinoma of the Duodenum.* The duodenum is divided, for descriptive purposes, into supra-ampullary, ampullary and infra-ampullary regions. Table 1 shows the relative frequency with which they are affected.

TABLE 1. DISTRIBUTION OF DUODENAL CANCER

<i>Author.</i>	<i>Supra-Ampullary.</i>	<i>Ampullary.</i>	<i>Infra-Ampullary.</i>
Rolleston	8	24	3
Geiser	11	51	9
Fenwick	11	29	7

It will be noticed that by far the greatest number of cancers occurs in the region of the ampulla of Vater.

Although the duodenum is highly resistant to the disease, it is not the pylorus which forms the actual barrier. It has been shown that the duodenum does not always escape in pyloric cancer, but the growth seldom invades it for more than a few centimetres. Nagel has demonstrated cases of pyloric cancer which to the naked eye involve the duodenum, but he has added to this number after microscopical examination. Table 2 shows the evidence of other writers on this point.

TABLE 2. FREQUENCY OF DUODENAL INVOLVEMENT IN CANCER OF THE PYLORUS

<i>Author.</i>	<i>Cancer of Pylorus.</i>	<i>Duodenum Microscopically Involved.</i>
Konjetzny	54	23
Brinton	125	10
Maragliano	12	2
Borrmann	63	20

*Pathology.* Duodenal carcinoma usually takes the form of a cylindrical-cell adeno-carcinoma arising from the mucosa. It has a

definite tendency to change to the squamous or spheroidal type. At the ampulla of Vater three types of epithelium are met with. There are the high cylindrical cells of the choledochus, the clear flat cells and mucous cells of the duodenum, and the low cuboidal cells of the pancreatic duct of Wirsung. This differentiation should not be unduly stressed, since from a practical point of view the problem facing the surgeon is the same. If the growth extends in the form of a plaque, the duodenum is probably the seat of origin, whereas a pedunculated

carcinoma protruding from the ampulla probably has its origin in the common bile-duct.

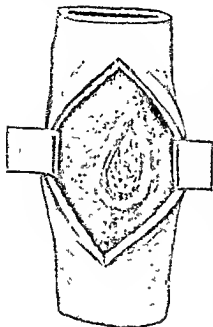


Fig 349.—CARCINOMA OF DUODENUM. DUODENUM OPENED, EDGES RETRACTED, EXPOSING MALIGNANT ULCER, ON POSTERIO MEDIAL WALL, ENCHANCING PROXIMALLY ON AMPULLA.

(J. E. Schofield "British Journ. Surg.")

Many theories have been advanced as to the origin of these tumours. By some it was thought that the condition arose in aberrant stomach glands, by others that the primary site was in pancreatic rests, which are known to occur in the first part of the duodenum. Orth favoured the view that the origin was in Brunner's glands. Morrison and Feldman have reported a case of carcinoma arising in a diverticulum of the first part of the duodenum. The question as to whether carcinoma arises in a duodenal ulcer has been for years a subject of controversy. In the stomach Walton thinks that 20 per cent of carcinomata are preceded by ulcer, but there is almost complete absence of evidence so far as the

duodenum is concerned. Isolated cases have been recorded from time to time, but most of them fail to survive a rigid investigation. One cannot fail to observe the disparity in the numbers of duodenal ulcers and duodenal cancers. Furthermore, cancer in the duodenum is most frequently found at the ampulla of Vater, whereas duodenal ulcer is almost exclusively confined to the first portion of the duodenum.

The growth tends either to spread along the duodenum or to encircle it, eventually leading to obstructive symptoms. In the ampullary region early obstruction of the common bile-duct occurs, producing a rapidly progressive jaundice, with dilatation of the common

bile-duct and gall-bladder, which become filled with aseptic bile and mucus. Perforation is rare, but local abscess has occurred. Involvement of the portal vein has led to ascites, whilst external fistula or internal fistula with the gall-bladder or colon is an occasional complication. Metastases occur late in the disease, a fact which at first sight might lead one to consider a case of duodenal cancer in a favourable light. When metastases do occur it is usually the neighbouring organs, such as liver and pancreas, which are involved.

*Diagnosis.* The diagnosis of duodenal cancer is undoubtedly a matter of great difficulty. Naturally the symptoms and signs depend to a great extent on the particular portion of the duodenum affected. There are, of course, vague abdominal symptoms which are common to many other abdominal diseases. Indigestion, epigastric discomfort and loss of appetite are among these.

Little need be said concerning supra-ampullary cancer, since the symptoms closely resemble those of pyloric cancer. Suspicion of a supra-ampullary growth may arise in the mind of the radiologist, when investigating a case referred to him for gastro-duodenal investigation.

Ampullary cancer, on the other hand, produces symptoms quite peculiar to itself, the most important of which is jaundice. This appears early on in the disease and is usually progressive in character. Occasionally the jaundice disappears but returns later. It requires only the smallest growth to produce obstruction of the common bile-duct and a correspondingly serious condition of the patient. Carcinoma above or below the ampulla of Vater may progress until the lumen of the bowel is occluded, without giving rise to serious symptoms; but a small ampullary growth can produce rapid emaciation and death. One very constant feature is enlargement of the gall-bladder and liver. Should infection of the stagnant bile occur, it will result in intermittent fever, with sweats and rigors. Wasting occurs early on in the disease, due to obstruction of the flow of the pancreatic juice, and fat may show itself in the faeces. Pain is not a marked feature, but as a rule vomiting occurs. One would naturally consider carcinoma of the head of the pancreas and stone in the common bile-duct, in view of the symptoms, but their rapid progress and the absence of pain might lead one to suspect the true nature of the condition. It should certainly encourage one to examine the ampullary region at operation.

Apart from vague upper abdominal symptoms, the disease in the infra-ampullary region may progress unheeded until the onset of obstruction. The condition is then characterised by copious vomiting,

the vomit containing large quantities of bile. Rapid emaciation follows. The stomach and duodenum proximal to the growth become enormously dilated, and the true site of the obstruction will be revealed by a barium meal examination. Even then the diagnosis is liable to be confused with tumour pressing from outside, or obstruction due to the superior mesenteric vessels.

*Treatment. Pre-operative.* This is of paramount importance since the jaundiced and debilitated condition of these patients has been to a large extent responsible for the appallingly high operative mortality in peri-ampullary cancer. Particular attention should be paid to the jaundice, for hæmorrhage following the operation has repeatedly defeated the surgeon in his purpose. Blood-transfusion, which will decrease the clotting time of the blood, should be undertaken, and calcium chloride in doses of 5 cc. of a 10 per cent solution should be administered intravenously for three days before the operation. In addition, the patient's condition will be improved by the administration of large quantities of saline, subcutaneously, intravenously and per rectum for some days prior to operation.

*Supra-Ampullary Cancer.* This extremely rare condition is treated on the same lines as pyloric cancer.

*Ampullary Cancer.* When confronted by a cancer in this situation, the surgeon has to decide on the question of operability. This is determined by the size of the growth, its fixity, the involvement of local structures, the presence or otherwise of metastases, and the condition of the patient.

If the growth is considered inoperable, much relief can be afforded to the patient by means of a palliative operation. Obstruction of the passage of the stomach contents can be overcome by gastro-enterostomy, and jaundice relieved by drainage of the gall-bladder. Anastomosis of the gall-bladder to the stomach is preferred, since it gives the patient the benefit of the bile, and avoids a copious discharge on to the surface of the abdomen which is distressing to the patient.

A preliminary palliative operation should be performed in those cases which are regarded as suitable for an attempted radical cure. It is preferable to perform the palliative operation and await the recovery of the general condition of the patient before attempting the radical operation two or three weeks later. Tremendous benefit accrues to the patient in these few weeks of waiting, for the jaundice rapidly

disappears and emaciation is considerably relieved. When the palliative procedures have been completed, the surgeon turns his attention to the growth itself.

The duodenum should be carefully mobilised and opened through an anterior vertical incision. The tumour may be exceedingly small and removable by local excision. Such a minute procedure should not be undertaken lightly. Of 53 cases reported by Cohen and Colp which were treated by local excision, 50 by the trans-duodenal, and 3 by the retro-duodenal route, only 18 survived the operation and its immediate after-effects. Death may result from shock, hæmorrhage, peritonitis, fistula, etc. Moreover, in about 50 per cent of cases it is necessary to re-insert the common bile-duct into the duodenum. Drainage of bile will be delayed for a few days owing to the inflammatory reaction at the site of re-insertion. If a preliminary cholecyst-gastrostomy has been performed, its value will be appreciated at this stage.

Should the growth have extended beyond the papilla, so that simple papillectomy fails to remove the whole of it, then the surgeon is faced with the problem of performing a circular resection of the duodenum, including a portion of the pancreas. This is an operation of considerable magnitude to be undertaken in a patient who by this time is probably in an extremely poor condition. The unsatisfactory nature of this operation can be judged from the account of Cohen and Colp, who report that only four persons are known to have survived this ultra-radical procedure. Such a mode of treatment therefore deserves little or no consideration.

Results from radium treatment of cancer in this particularly difficult situation would be welcomed, but so far little is known concerning the reaction to radium of malignant disease of the small intestine. The writer recorded a case of cancer occurring immediately below and involving the ampulla of Vater, which he treated by the implantation of radon seeds, after having performed cholecyst-gastrostomy and gastro-enterostomy. The patient died 29 days later from duodenal fistula. At the same time it must be admitted that the surgical treatment of peri-ampullary cancer is an extremely precarious adventure, so that radium treatment combined with a palliative operation might for the present be regarded as a more justifiable choice of treatment.

*Infra-Ampullary Cancer.* In this region resection is preferred to local excision. The third and fourth portions of the duodenum are



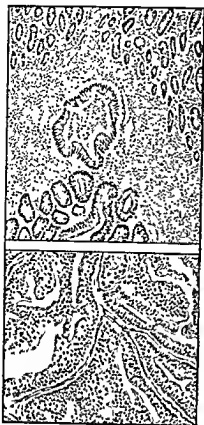


FIG. 350.—THE LOWER FIGURE SHOWS SECTION OF CARCINOMA REMOVED AT OPERATION. THE ALVEOLI ARE LINED IN PARTS BY COLUMNAR CELLS, IN OTHERS THE CELLS PROLIFERATE IRREGULARLY AND PARTLY FILL THE LUMEN, STROMA SCARCY. ABOVE, NORMAL DUODENUM AT EDGE OF GROWTH. ( $\times 65$ )

(J. E. Schofield : *British Journ. Surg.*)

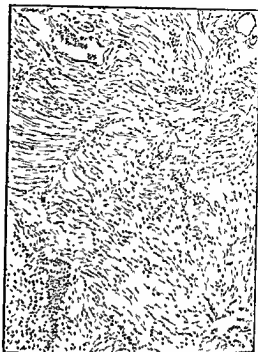


FIG. 351.—SECTION OF SAME GROWTH AFTER INTRODUCTION OF RADON SEEDS, CONSISTING MAINLY OF FIBROUS TISSUE AND EPITHELIAL DEBRIS. SOME CELL PROLIFERATION PRESENT, PROBABLY CHRONIC INFLAMMATORY. NO EVIDENCE OF ORIGINAL TYPICAL CARCINOMATOUS PICTURE. ( $\times 63$ )

(J. K. Schofield : *British Journ. Surg.*)

mobilised, and the proximal portion anastomosed to the jejunum, either by end-to-end or end-to-side anastomosis. Pauchet and Luquet reported such a case in a woman of 61 years of age, who made an uninterrupted recovery and later returned to her work.

#### CANCER OF THE JEJUNO-ILEUM

*Pathology.* The growth in nearly all cases is adeno-carcinoma, the scirrhus or colloid variety occasionally occurring. Although the tumour may project into the lumen in some cases, it is usual for it to encircle the bowel, stenosis eventually resulting. Obstruction, however, does not show itself until stenosis becomes almost complete, owing to the fluid nature of the bowel contents. Metastases occur late

in the disease. It is not uncommon for the proliferating non-stenotic type of carcinoma to be mistaken, on naked-eye examination, for tuberculosis or sarcoma.

*Diagnosis.* It is impossible to enumerate any definite symptoms of the early stage of cancer in this situation. Vague symptoms may

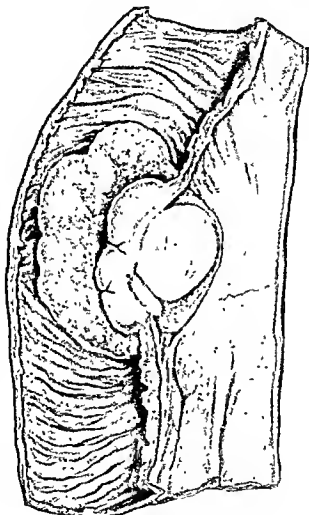


Fig. 352.—CARCINOMA OF JEJUNUM.  
(Hunterian Museum, R.C.S.)

precede the onset of stenosis. This is characterised by colicky pains, vomiting, constipation, and in some cases loss of weight, but this is not constant. As the obstruction becomes more pronounced, the coils of intestine proximal to the growth become tremendously distended, producing a ladder pattern centrally placed in the abdomen. In jejunal carcinoma, vomiting is an earlier and marked symptom.

The non-stenotic type proceeds in subtle fashion without subjective

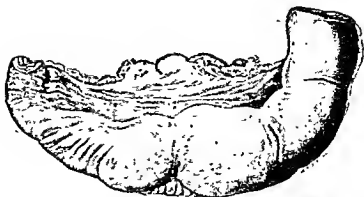


Fig. 353a.



Fig. 353b.



Fig. 353c.

**CARCINOMA OF ILEUM. SPECIMEN REMOVED AT OPERATION.**

(a) VIEW OF SPECIMEN SHOWING CONSTRICTION DUE TO GROWTH AND DILATATION AND HYPERTROPHY OF THE GUT PROXIMAL TO THE LESION.

(b) SECTION OF THE SPECIMEN SHOWING THE LUMEN OF THE GUT CONSTRICTED BY AN ANNULAR GROWTH.

(c) MICROSCOPIC SECTION OF THE GROWTH SHOWING COLONNAR-CELLED CARCINOMA (ADENO-CARCINOMA).

The gland shown in (c) has deposits of a character similar to those of the primary growth.

(Mr. Rodney Mowbray's case; specimen now in the Museum of the Royal College of Surgeons.)

symptoms. Meanwhile the patient suffers from fatigue, emaciation and anæmia. The latter symptom may result in the condition being mistaken for one of pernicious anæmia, and is due to bleeding from the growth into the lumen of the bowel.

A lump may be felt on palpating the abdomen and, in the case of the ileum, the growth may weigh down the affected coil of bowel, so that it can be felt on rectal examination. Ascites is rarely present.

X-ray has been claimed to be of value in the diagnosis, but very few cases come to light through this medium. Grant, however, has demonstrated, by means of barium series, two cases of jejunal obstruction due to carcinoma, which was confirmed at operation. The cardinal feature in both cases was the enormous dilatation of the bowel proximal to the obstruction. Some radiologists claim to recognise small intestinal tumour from a "straight" film of the abdomen by the evidence of gaseous obstruction, but this has not proved altogether reliable.

*Treatment.* It is because of the tendency for carcinoma of the jejuno-ileum to metastasise late, that early diagnosis is important.



Fig. 354A.—X-RAY DIAGNOSIS IN JEJUNAL OBSTRUCTION DUE TO CARCINOMA.

NOTE DILATED JEJUNUM. BARIUM HELD UP ABOUT ONE FOOT FROM DUCODENO-JEJUNAL FLEXURE.



*Fig. 354b.*—THREE HOURS LATER. MARKED DILATATION OF JEJUNUM.



*Fig. 354c.*—SIX HOURS LATER. THE BARIUM IS STILL ACCUMULATED IN THE FIRST FOOT OF THE JEJUNUM AND NONE HAS PASSED BEYOND THAT POINT.

(Case of Dr. J. L. A. Groll.)

Most of the cases are met with at operation in the presence of acute intestinal obstruction. It is advisable, therefore, not to attempt a radical cure *ab initio*, but to perform either a lateral anastomosis or jejunostomy. When the danger of obstruction has passed, a resection of the growth should be undertaken, together with a considerable portion of healthy bowel, both above and below the growth. The surgeon should not be deterred from his purpose by the presence of glands in the mesentery. It has already been pointed out that metastases occur late, and if glands are present, they may be only inflammatory in character. Nevertheless, although the results of the operation are poor, the percentage of cases which recover renders the procedure justifiable.

An important point to be observed is the necessity for a complete examination of the abdominal organs. What at first sight may appear to be a primary carcinoma of the small intestine may prove on further investigation to be a secondary deposit from primary carcinoma of the gall-bladder, ovary, etc.

*Globocellularis* or *Carcinoid Tumour*. This tumour, first described by Lubarsch in 1888, has given rise to a good deal of controversy. Whilst resembling carcinoma histologically, it is clinically benign and characterised by an almost constant absence of metastases. Krompecher suggested the resemblance of this tumour to the basal-celled carcinoma, and it has received the title of "The Rodent Ulcer of the Small Intestine." Its commonest site is in the ileum and the appendix. The tumour grows slowly and rarely reaches a sufficient size to be of any clinical importance, although intussusception has been known to result. It is usually about the size of a pea, has a wide base and a smooth surface, and the mucous membrane remains intact. Recurrence does not occur after removal.

Recently the term "Argentaffin tumour" has been applied on account of the fact that granules in the tumour take the silver stain. In the light of our present knowledge, it would appear that the term "Carcinoid tumour" more aptly describes it.

*Sarcoma of the Small Intestine*. Although sarcoma is more common in the small than in the large intestine, very few cases have been known to occur. It is much rarer than carcinoma, and appears at an earlier age. Persons between 30 and 40 years of age are more liable to be affected, although the disease has occurred in infants and the aged. No portion of the small bowel is exempt from sarcoma. It arises in the

submucosa and rapidly proceeds to infiltrate the coats of the bowel. It may occur as a single growth or as nodules scattered throughout a considerable length of the intestine. Occasionally the mucous membrane remains intact, but often deep ulceration occurs, resulting in hæmorrhage. Erosion of the inferior pancreatico-duodenal artery has been known to occur in sarcoma of the duodenum. Metastases occur at an early date.

The symptoms tend to be general rather than local in character. Emaciation, weakness and anorexia occur early with vague abdominal pains, diarrhœa or constipation.

In contrast to carcinoma, stenosis rarely results, for, owing to involvement of the muscle coat of the intestine, dilatation occurs. The tumour therefore tends to grow to a sufficient size to allow of its being felt before obstruction supervenes. It is usually with a large abdominal tumour already present that patients seek surgical advice. Others seek treatment on account of threatened intestinal obstruction. In either case, with metastases occurring early on, the condition is nearly always hopelessly inoperable. Palliative lateral anastomosis is all that can be offered. Sufficient evidence of the results of radium and deep X-ray therapy treatment of so rare a condition is not yet available.

## CHAPTER II

### INJURIES TO THE SMALL INTESTINE

OF all the abdominal organs, the small intestine is the most liable to suffer in abdominal injury. This is on account of its centrally-placed position, and its proximity to the vertebral column. Only about one-tenth of intestinal injuries affect the large bowel.

*A. Closed Injury.* This type of injury is commonly produced by a blow with the fist, a kick, the passing of the wheel of a vehicle over the abdomen, or by the impinging of a blunt instrument against the anterior abdominal wall. The suddenness of the blow, taking the abdominal muscles off their guard, crushes the intestine against the bodies of the vertebræ or the promontory of the sacrum. The duodeno-jejunal flexure, on account of its fixity, is particularly vulnerable, and is liable to be torn away from its attachment. The so-called pneumatic rupture, due to compression of air in the intestine, is seldom encountered.

It must be remembered that serious damage to the bowel may be brought about by trivial injury, or by indirect violence such as a fall on the feet or buttocks.

*Diagnosis.* It cannot be unduly stressed that the amount of external injury to the abdominal wall is no criterion as to the degree of damage which the deeper structures may have sustained.

It is the rule rather than the exception to find, even in serious intra-abdominal injury, little or no evidence of trauma of the abdominal wall. The clothing is to some extent protective, and bruising does not immediately show itself. It is therefore important that a careful enquiry be made as to the details of the accident.

When seen soon after the accident, the patient will be found to be suffering from shock. If severe, this may result in death within a few minutes of injury. On the other hand, it may be completely absent.

The typical facies, showing pallor, anxious expression and apprehension are usually present, whilst vomiting and nausea are fairly constant.



Pain is a marked feature, and, though at first slight, becomes severe, localised and persistent. The persistency of the pain is of particular diagnostic importance.

Abdominal respiration is usually absent. Generalised tenderness and rigidity are present early on, eventually becoming localised. Little attention is paid nowadays to the disappearance of the liver dullness, which is easily mistaken for gaseous distension of the colon. In the following case treated by the author the majority of these features were evident, so that no diagnostic difficulty presented itself.

"A middle-aged man was admitted to hospital suffering from abdominal injury. On careful inquiry it was ascertained that he had been struck in the abdomen and pinned against a wall by the corner of a safe which weighed 30 cwts. He was suffering from severe shock, the facies were ashen grey and he appeared very frightened. He had been in severe pain since the accident and had vomited once. Breathing was embarrassed and there was marked abdominal tenderness and rigidity, especially in the region of the umbilicus. The slightest movement caused the patient to cry out. A diagnosis of ruptured bowel was made, morphia was administered and an operation performed three hours later. A paramedian incision was made to the right of the umbilicus, and on opening the peritoneum brown-coloured fluid escaped. A collapsed loop of bowel was found lying immediately beneath the incision, and contained a perforation, through which the mucous membrane was everted. The loop was gently lifted out of the wound and held in a swab by the assistant. A careful examination was made for other perforations but none was found. Close to the perforation was a small hæmatoma of the mesentery which did not require attention. The perforation was closed by means of a purse-string suture and the abdomen drained by the suprapubic route. The post-operative course was uneventful and the patient left hospital three weeks later."

Such then is the classical description of the clinical picture of injury to the intestine. It cannot, however, be relied upon, nor will it stand the surgeon in good stead. Only bitter experience teaches us how deceptive these cases can be, and in how subtle a fashion can a serious condition progress without betraying itself by physical signs.

This is fully illustrated in two cases supplied by Mr. R. St. Leger Brockman :

*Case 1.* "A boy, six years of age, was running down a hill with a toy barrow. He ran into a cab, and the handle of his barrow hit him in the upper abdomen. He was admitted to hospital at 11 a.m. and kept under close observation until 8 p.m. When

seen then, he was playing football with his cap with other boys in the ward, and on the strength of this was allowed to go home. He got up at 7 a.m. on the following day and an hour later complained of acute abdominal pain. He was brought to hospital immediately, but was found to be dead on arrival. The post-mortem examination showed that the first part of the duodenum was torn completely across. It was thought that leakage had not occurred until a few minutes before he died."

*Case 2.* (Followed case 1.) "A boy of 17 years of age was flung from one end of a swing boat to the other in a fair ground at 8 p.m. He arrived at hospital at 8.15 p.m. The upper abdomen did not move on respiration and his breathing was short and jerky. There was no other physical sign present. Bearing in mind Case 1, the abdomen was opened. The duodenum was found to be torn completely across, immediately distal to the pylorus, which was tightly closed by spasm. There was no leakage. The two ends of the duodenum were sutured together, and a posterior gastro-enterostomy performed. The boy made an uninterrupted recovery, and when seen six months later was quite well."

It is obvious, therefore, that the surgeon must not await the development of the classical picture. Decision must be influenced by one's personal experience, the presence of even one physical sign, and, above all, by a careful enquiry into the nature of the accident. It is better to view every abdominal injury as a case of potential intestinal rupture, and if, after careful consideration, suspicion lingers, the abdomen should be explored.

If the case is seen some time after the accident, the picture will have changed. Shock will have passed off, the temperature will be raised and the pulse-rate once again be rapid. Muscular rigidity will have given place to abdominal distension, and there may be dullness of the flanks. In other words, the picture is now one of peritonitis.

*Treatment.* Shock must always be treated from the outset, and it is wise to delay the operation for a few hours until this has passed off. One should not wait longer than about six hours from the time of the injury, since by this time peristalsis will have returned to the bowel.

In dealing with a case of ruptured small intestine it must be borne in mind that other organs may be injured. About 20 per cent of cases are complicated by some other intra-abdominal injury.

A right paramedian incision should be made at the level of localising signs, but if these be absent the incision should be inclined to the upper abdomen so as to allow the examination of the fixed duodenum.

On opening the peritoneum, the escape of gas, intestinal contents or blood is likely. The nature of any free fluid is noted, since this may betray the site of injury. A preliminary investigation should be made, special attention being given to bowel which appears collapsed. The intestine must receive gentle manipulation so as not to disturb too much the site of rupture. If a cursory examination fails to reveal the

damaged bowel, a systematic investigation of the whole length of the intestine follows. The presence of bile in the free fluid would suggest beginning at the duodenum. Foul-smelling or brown-coloured fluid would indicate that the examination should commence at the ileo-cæcal valve. The bowel must be examined most carefully since a perforation is easily overlooked.

The injured portion of bowel should be withdrawn from the abdomen and held in a large swab by the assistant, whilst a further search is made for other perforations. These are frequently multiple and

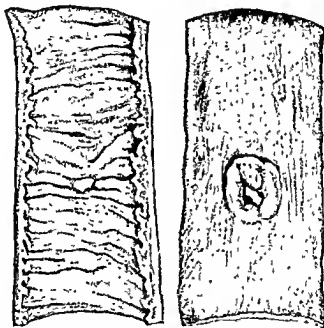


Fig. 355.—PORTION OF JEJUNUM IN WHICH THERE IS A TRANSVERSE OVAL RENT FROM A MAN OF 70 YEARS OF AGE WHO WAS KICKED IN THE ABDOMEN BY A HORSE. NOTE THE EVERSION OF THE MUCOUS MEMBRANE, THUS LIMITING ESCAPE OF FÆCAL CONTENTS.  
(*Hunterian Museum, R.C.S.*)

usually occur in adjacent loops of bowel. The edges of the perforations will be found to be everted, and in the first few hours after injury peristalsis has usually ceased.

Treatment will depend on :

(1) *The Extent of the Tear.* A small circular perforation may be closed by means of a purse-string suture, strengthened by stitching over a piece of omentum.

Larger tears will require suturing in a transverse direction. This procedure will be made easier by fixing a clamp above and below the site of rupture. Little fear need be entertained as to the patency of

the lumen, since this is rarely encroached upon and lateral anastomosis is seldom necessary. If the rupture is complete, the edges should be pared and an end-to-end anastomosis performed.

A large tear or multiple tears in a loop of howel may render resection necessary.

(2) *Involvement of the Mesentery.* This may be slight, requiring ligature of vessels and suture. On the other hand, it may be so extensive as to require resection of the corresponding loop of howel.

(3) *The Condition of the Patient.* Although resection of howel may be indicated, it often happens that the patient's condition is so grave that this cannot be undertaken without serious risk. In such a case an attempt at suture of howel of doubtful viability is justified.

(4) *Time which has elapsed since Injury.* Early cases will be treated as above. Unfortunately some patients come to operation after the onset of peritonitis. One may attempt to suture the howel and drain the abdomen, but often it is well to take advantage of the perforation and drain the intestine by inserting a Paul's tube.

It need hardly be added that repair of bowel should be undertaken outside the abdominal cavity, and all cases be given suprapubic drainage.

*Rupture of the Duodenum.* If the duodenum is ruptured, gastro-enterostomy may be performed in addition, provided the condition of the patient permits. Sometimes the rupture is extra-peritoneal, and such a possibility must always be borne in mind when exploring a case of abdominal injury. There is usually swelling beneath the parietal peritoneum in the region of the duodenum. Attempt at suture must be made, with drainage of the surrounding area. Duodenal fistula is liable to follow, so that, in addition, gastro-enterostomy with pyloric occlusion is recommended.

*B. Open Injury.* This type of injury, though just as serious as the closed variety, does not present the same diagnostic difficulty. It is usually produced by a stab with a knife or puncture with a sharp-pointed instrument. The injured bowel may or may not protrude through the wound.

The symptoms, whilst resembling those of the closed variety, are usually less marked.

In every case of penetrating wound of the abdomen, injury to the

intestine must be presumed until proved otherwise. This can only be done by opening up the wound and making a systematic search of the bowel. The wound is cleaned as much as possible before the peritoneum is opened. Usually one finds the injured loop immediately beneath the external wound.

The wounds, for they are often multiple, are dealt with and the peritoneum closed, with drainage.

The following case of the author is illustrative :

"A boy, 12 years of age, was impaled on the spike of some railings. When admitted to hospital he was found to have a wound in the right iliac fossa, very much resembling a grid-iron incision. At first sight it appeared that only the skin and subcutaneous tissues were involved. He was in no way alarmed at his misfortune, and there were no physical signs of injured bowel. He was taken to the operating theatre, the wound cleaned and the abdomen explored. There were no less than five punctured wounds of the small intestine within a few inches of each other. There was no leakage, probably due to contracture of the muscular coat of the bowel. Each perforation was closed by means of a purse-string suture, and the wound drained. The boy made a rapid recovery."

*Gunshot Wounds.* Wounds due to pistol or rifle bullets are sometimes encountered in civil practice. Usually the intestine is perforated in a number of places. No time should be wasted in searching for the bullet. Treatment is carried out on the lines already indicated for rupture. Often the condition is complicated by severe hæmorrhage from large blood-vessels.

C. *Injuries due to Foreign Bodies in the Intestine.* Very rarely does a foreign body, swallowed accidentally or intentionally, become lodged in the intestine. This is a rather remarkable fact considering the size of the objects which are swallowed. Should a foreign body perforate the intestine, the process is usually a slow one, resulting in either a localised abscess well shut off by adhesions, or an internal and sometimes external fistula. Occasionally a sharp object such as an open safety-pin causes a perforation which results in localised pain and rigidity and demands operative interference.

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## CHAPTER III

### CHRONIC DUODENAL ILEUS

THIS condition, first described by Petit in 1900, has since occupied the attention of numerous observers. Interest was aroused in this country by the writings of Wilkie in 1921, but some time elapsed before duodenal ileus came to be accepted as a definite pathological entity.

*Pathology.* Chronic dilatation of the duodenum may occur due to compression of the bowel between a tight root of the mesentery and the lumbar vertebræ. The dilatation extends as far as the superior mesenteric artery; the first part of the duodenum is usually the most dilated and may resemble, as Wilkie suggests, a second stomach. Associated with this condition is a degree of visceroptosis, which is thought to be responsible for the drag on the mesentery. The small intestine may be prolapsed into the pelvis, or the cæcum and ascending colon may pull in the line of the superior mesenteric artery, or even the right colic artery. This is the generally accepted view, but it has not met with unanimous approval. Robertson is of the opinion that the condition is one of chronic paralysis of the duodenum and considers the operation of duodeno-jejunostomy to be valueless.

Other causes have been cited in addition. Rowlands found a congenital band compressing the second part of the duodenum just before it passes under the transverse mesocolon. He also successfully treated a case of duodenal pouch affecting the fourth part, which when loaded pressed upon the duodenum and caused dilatation. Tuberculous calcified glands in the root of the mesentery, secondary carcinomatous glands in the same site, congenital adhesions obstructing the duodeno-jejunal flexure and acquired adhesions following gastro-enterostomy have all been mentioned as causes of duodenal ileus.

As a result of continued intermittent obstruction the duodenal wall becomes thickened and the pylorus dilated. Wilkie regarded chronic duodenal ileus as a predisposing cause of duodenal ulcer, and to a less extent, of gastric ulcer. This view has been substantiated by

many others. He also takes the view that regurgitant vomiting following the operation of gastro-enterostomy is in some cases due to an already existing duodenal ileus.

*Diagnosis.* The patient is usually a female in whom visceroptosis is present. Symptoms often date from childhood, and the patient regards herself as having a "weak stomach" easily upset by vagaries of diet. Nausea, indigestion and bilious attacks comprise the usual run of symptoms. When adult life is reached these take on a more definite character. Epigastric discomfort and distension come on during or after meals, and are aggravated by standing or exercise. The patient will often complain that she is unable to finish her meals. Every few weeks there is an exacerbation of symptoms described by the patient as "bilious attacks." These are usually preceded by constipation. By the time the patient has sought advice, she has usually discovered that relief can be obtained by lying face downwards or in the genu-pectoral position.

Physical examination rarely reveals any positive sign, but epigastric distension or hyperæsthesia of the skin may be observed in extreme cases. If X-ray diagnosis is to be of value, it is imperative that the examination should take place during an "attack." Dilatation of the duodenum will then be seen, with stasis, or it may be possible to observe reverse peristalsis. Evidence of duodenal or gastric ulcer may be found at the same time. In addition, ptosis of the small intestine or of the cæcum and ascending colon may be observed which will give an indication of the causal factor.

Duodenal ileus can be so easily confused with other conditions such as recurrent appendicitis, cholecystitis, gastric and duodenal ulcer, that



FIG. 356.—X-RAY DIAGNOSIS OF DUODENAL ILEUS. THE BARIUM WAS HELD UP IN THE SECOND AND THIRD PARTS OF THE DUODENUM. VIGOROUS ANTIPERISTALSIS WAS NOTED.

(Case of Dr. J. L. A. Groat)



the importance of the X-ray examination cannot be unduly emphasised, because it may prove to be the deciding factor in diagnosis.

*Treatment.* Medical treatment should be given a trial in every case, and since duodenal ileus has now come to be regarded as a complication of visceroptosis, attention should be devoted to its relief. Rest in bed with the foot of the bed elevated, abdominal massage, regulation of diet, gastric and duodenal lavage, exercises and supports should all be tried. Such remedies may cure or give considerable relief to milder cases. Should medical treatment fail to alleviate the symptoms, or the condition be complicated by duodenal or gastric ulcer, then operation is indicated. Despite the doubts which have been shared by a number of observers, duodeno-jejunostomy remains the operation of choice. It is easy to perform and the post-operative course is uneventful. The transverse colon is raised up, thus exposing the prominent third part of the duodenum. The peritoneum over it is incised, and the duodenum mobilised. The first loop of jejunum, about six to eight inches from the duodeno-jejunal flexure, is then brought over to the right and anastomosed to the mobile duodenum.

The operation of gastro-enterostomy is not advisable, because it not only fails to afford relief, but it may also cause the condition to become aggravated by adhesions or by kinking at the site of anastomosis. Where definite prolapse of the caecum and ascending colon is demonstrated, fixation is performed by some surgeons.

The after-treatment is carried out on the same lines as that for gastrectomy or gastro-enterostomy. Immediate relief is usually experienced by the patient and in the majority of cases this proves to be of a permanent nature.

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SECTION 6  
GALL-BLADDER AND BILE-DUCTS

CHAPTER I  
Treatment of Injuries and Diseases of the Gall-Bladder and  
Bile-Ducts  
by  
RODNEY MAINGOT

CHAPTER II  
Gall-Stones and Cholecystitis  
by  
A. M. A. MOORE

CHAPTER III  
Congenital Cystic Dilatation of the Common Bile-Duct  
by  
A. DICKSON WRIGHT

SECTION 6  
GALL-BLADDER AND BILE-DUCTS

CHAPTER 1

TREATMENT OF INJURIES AND DISEASES OF THE GALL-BLADDER AND  
BILE-DUCTS

by  
RODNEY MAINGOT

- (A) INDICATIONS FOR OPERATIVE TREATMENT.
- (B) PRE-OPERATIVE TREATMENT.
- (C) TECHNIQUE OF OPERATIONS UPON THE GALL-BLADDER AND  
BILE-DUCTS.
- (D) POST-OPERATIVE TREATMENT.
- (E) RESULTS OF OPERATIVE TREATMENT.

(A) INDICATIONS FOR OPERATIVE TREATMENT

- (1) Injuries.
  - (a) Operative trauma.
  - (b) Rupture of the gall-bladder or bile passages due to external violence, gunshot wounds, stabs, etc.
- (2) Primary carcinoma :
  - (a) Of the gall-bladder.
  - (b) Of the bile-ducts.
- (3) Gall-stones and their resulting complications.
- (4) Cholecystitis.
  - (a) Chronic.
  - (b) Acute.

- (5) Biliary fistula.
  - (a) Internal.
  - (b) External.
- (6) Obstruction of the common bile-duct.

### (1) INJURIES

*Operative Trauma.* During the performance of cholecystectomy, owing to the remarkable variations in the anatomy of the bile-ducts and the hepatic and cystic arteries, damage to vital structures can be prevented only by constantly bearing in mind that such variations are very common, and that nothing should be divided which has not first been clearly identified and demonstrated to an assistant. In all cases the cystic duct must be dissected free and traced upwards to the neck of the gall-bladder and downwards to its junction with the common ducts. The cystic artery, likewise, must be carefully isolated and traced to the point where it is clearly seen to enter the region of the neck of the gall-bladder (fig. 357).

Flint (*Brit. Jl. Surg.*, x, 509, 1923) draws attention to these abnormalities of the bile-ducts and associated blood-vessels, and emphasises their surgical importance. Normal arrangement of the ducts and blood-vessels was found in only 69 out of 200 consecutive dissections.

The *gall-bladder* may be absent or rudimentary, or it may be buried in the liver. On the other hand, it may be suspended from the liver by a long lax mesentery, and only in such cases can a *volvulus* of the organ occur.

The *cystic duct* may also be absent and the gall-bladder possess a wide opening into the common duct. The cystic duct may be very short or very long; when long it may enter the duodenum separately or may join the common duct low down in the substance of the head of the pancreas or in the region of the ampulla of Vater. It is often closely bound to the main duct. Again, the cystic duct may lie in a loop anterior to the common duct or may form a loop behind it.

*Accessory hepatic ducts* are present in some 20 per cent of cases, and originate from the right lobe of the liver. They may enter the gall-bladder itself, the cystic duct, the right hepatic duct, or the common hepatic duct just above the point where the cystic duct joins the common ducts. An accessory hepatic duct, which is often attenuated, is frequently unrecognised during the operation of cholecystectomy,

and leakage of bile from the divided end may be a troublesome or even fatal post-operative complication.

Anomalies of the *blood-vessels* are exceedingly common, the chief variations affecting the common and right hepatic arteries. These arteries may lie anterior to the common ducts. Again, the right hepatic artery may lie in very close proximity to the cystic duct or to

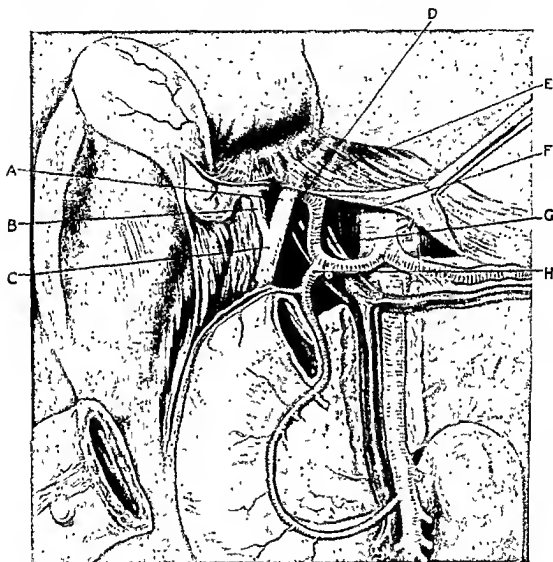


Fig 357.—ANATOMY OF THE BILIARY PASSAGES AND BLOOD-VESSELS IN THE REGION OF THE PORTAL FISSURE AND OF THE RIGHT BORDER OF THE GASTRO-HEPATIC OMENTUM.

- A = Common hepatic duct (hepatic duct). The right and left hepatic ducts are seen in the portal fissure  
 B = Cystic duct.  
 C = Common bile-duct  
 D = Right hepatic artery.  
 E = Left hepatic artery.  
 F = Common hepatic artery (hepatic artery).  
 G = Portal vein.  
 H = Gastro-duodenal artery.

(Adapted from Kirschner's "Operative Surgery," Lippincott, by kind permission of Julius Springer.)

the neck of the gall-bladder, and may be inadvertently ligatured instead of the cystic artery (fig. 358).

There may be more than one cystic artery, or this artery may spring from the common hepatic, from the left hepatic, from the gastroduodenal, from the superior pancreaticoduodenal, or even from the superior mesenteric artery. The plexiform arrangement of veins over the common ducts is often a source of troublesome hæmorrhage during dissection for exposure of the ducts. The *portal vein* may also be injured during operation upon the gall-bladder and bile-ducts. Again, it may be torn as a result of external violence. The bleeding from this important vein should be temporarily controlled by inserting a finger through the foramen of Winslow and compressing the structures in the free border of the gastro-hepatic omentum between the finger and the thumb. The structures here are lifted forwards while a careful dissection is undertaken. A small lateral tear in the vein should be clipped with a hæmostat and ligatured with silk. A long lateral tear is best closed by a continuous suture of fine silk which is carried on a small atraumatic needle. When the vein is divided across, it may be possible to repair it by end-to-end suture with fine silk or catgut.

Figure 358 illustrates the right hepatic artery crossing the common hepatic duct and coursing upwards to the right lobe of the liver behind the gall-bladder, where the cystic artery springs from it in an unusual position. The right hepatic artery, owing to its abnormal position, may easily be mistaken for the cystic artery and be ligatured.

Figure 359 shows buttonholing of the common hepatic duct. This injury can be recognised immediately after cholecystectomy has been performed, as bile wells up into the wound at the completion of the operation. In such cases the aperture should be snugly closed round a rubber tube which serves as a drain.

An accident such as is depicted in figure 360 may occur when, during the operation of cholecystectomy, the cystic artery, which lies behind and in very close proximity to the cystic duct, is unnoticed by the surgeon when the stump of the cystic duct is ligatured. Numerous adhesions, and perhaps oozing of blood, obscure the parts in the region of the right hepatic duct, causing it to be mistaken for the cystic artery. This is a very serious, though not necessarily fatal, mistake, and if recognised at once an end-to-end anastomosis of the divided duct should be attempted.

Figure 361 shows how the common ducts may be inadvertently clamped during cholecystectomy. Here the surgeon has mobilised the gall-bladder from the fundus and has exerted considerable traction

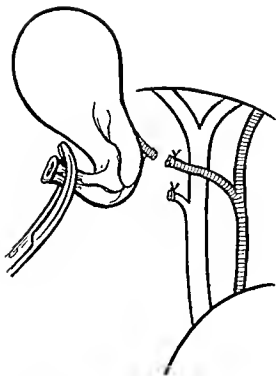


Fig. 358.—LIGATURE OF THE RIGHT HEPATIC ARTERY DURING THE PERFORMANCE OF A CHOLECYSTECTOMY.



Fig. 359.—BUTTONHOLING OF THE COMMON HEPATIC DUCT.

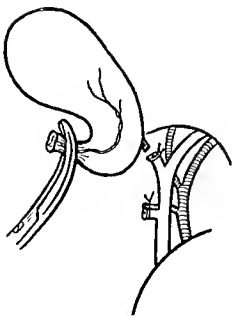


Fig. 360.—LIGATURE OF THE RIGHT HEPATIC DUCT DURING CHOLECYSTECTOMY.

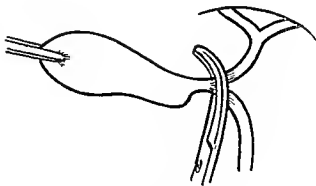


Fig. 361.—CLAMPING OF THE COMMON DUCTS DURING CHOLECYSTECTOMY. (After Kehr.)

which straightens out the cystic duct and draws the common hepatic and common bile-ducts together into an acute angle. This accident is prone to occur when the gall-bladder and bile-ducts are unduly mobile. The surgeon has failed to identify the three ducts and has applied the forceps in the manner shown.

Figure 362 shows Hartmann's pouch large and adherent to the common bile-duct for half an inch or more. The common bile-duct has been mistaken for the cystic duct and clamped.

Figure 363 represents a small sclerosed gall-bladder in which Hartmann's pouch overhangs the common duct; the cystic duct is very short, and is, to all intents and purposes, non-existent, and the body of the gall-bladder is tethered to the hepatic duct. Here, owing to lack of free exposure of the parts, the common duct has been mistaken for the cystic duct.

Figure 364 illustrates an unusual condition in which the cystic duct is long and firmly bound to the common hepatic duct. Here, again, owing to failure to identify each structure, the clamp has been applied both to the cystic duct and to the common hepatic duct.

Figure 365 depicts a frequent injury to the common hepatic duct. Here cholecystectomy is being performed from the cystic duct end of the gall-bladder, and a ligature applied to the cystic artery has slipped. The whole operative field has been flooded with blood, and a wild grasp through a pool of blood to pick up the divided end of the cystic artery has resulted in both artery and duct being crushed and possibly ligatured *en masse*.

*Other factors which contribute to injury* are a small incision with inadequate exposure in an obese patient, poor illumination, and a badly administered anæsthetic causing the patient to strain, thus making retraction difficult.

There is liability to injury also where the operation of cholecystectomy is commenced from the fundus; here the blood trickles from the liver during its separation from the gall-bladder, obscuring the field of vision when the most intricate part of the operation, i.e. ligature of the cystic duct and cystic artery, is about to be performed.

The use of large artery forceps for clamping the cystic duct and cystic artery may be another cause of injury, as such instruments are unsuitable for this delicate work and their points may consequently pick up more than is intended.

Furthermore, when the cystic artery is grasped by forceps the assistant may exercise undue traction to facilitate the application of the ligature, and in so doing the artery may be avulsed and cause a



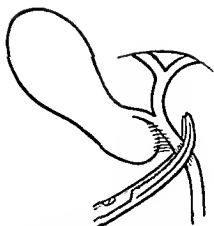


Fig. 362.—THE COMMON BILE-DUCT HAS BEEN CLAMPED IN MISTAKE FOR THE CYSTIC DUCT. (After Walton.)

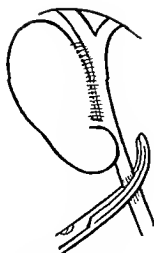


Fig. 363.—THE COMMON BILE-DUCT HAS BEEN CLAMPED IN MISTAKE FOR THE CYSTIC DUCT. HARTMANN'S POUCH OVERHANGS THE DUCT AND THE GALL-BLADDER IS ADHERENT AND SCLEROSSED.

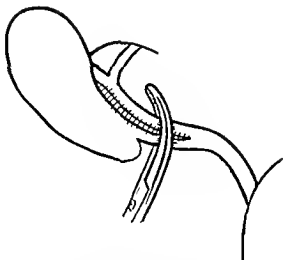


Fig. 364.—DIVISION OF THE COMMON HEPATIC DUCT WITH AN ADHERENT CYSTIC DUCT. (After Walton.)

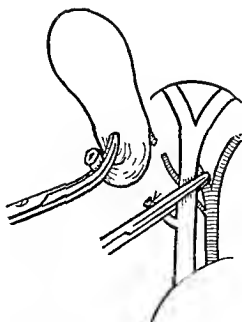


Fig. 365.—DUCT AND ARTERY CLAMPED BY A HÆMOSTAT FOLLOWING THE SLIPPING OF THE LIGATURE WHICH HAS BEEN APPLIED TO THE CYSTIC ARTERY. (After Walton.)

very serious and troublesome hæmorrhage. The search for the retracted end of the artery is always a tedious and hazardous procedure, and is fraught with danger of injury to the common ducts.

In old-standing cases of cholecystitis, where the gall-bladder is drawn to the under-surface of the liver, or where, in addition, there is a chronic duodenal ulcer with pseudo-diverticula, the duodenum is liable to injury. It may be torn during the separation of the two viscera, or a diverticulum may inadvertently be cut across where it lies hidden in a sheaf of adhesions. This accident should be recognised at the time by the bubbling of yellow frothy fluid through the small puncture. A few interrupted Lembert sutures will efficiently close the breach in the duodenum which should receive additional protection with an omental pad. Cholecystectomy is not advised in those cases where technical difficulties appear very formidable, or where prudence would dictate that the necessary surgical manipulations should be reduced to a minimum.

Clinically these cases of *operative trauma to the common ducts* fall into three groups :

- (a) Injuries recognised at the time of operation.
- (b) Post-operative fistula with escape of bile. With these cases there is no back pressure on the liver, no jaundice, and the tract is narrow.
- (c) Obstruction or obliteration of the duct. Here there is jaundice. Although the duct may be easy to find at operation the risks of operative interference are very great.

The *treatment* advised for these conditions is briefly outlined as follows :

(1) *Immediate operations.* When the injury inflicted upon the common ducts during the performance of a cholecystectomy is recognised at once, one of the following operations *may be necessary* :

- (a) End-to-end anastomosis (choledocho-choledochostomy), usually with the aid of rubber tubes.
- (b) Choledocho- or hepatico-enterostomy. Hepatico-enterostomy is anastomosis between the stump of the hepatic end of the bile-duct and the gastro-intestinal canal. In choledocho-enterostomy the *common duct* is anastomosed to the stomach, to the duodenum, or to the jejunum. The anastomosis may be *direct*, as in Mayo's operation, or *indirect*, as in Walton's operation.

- (c) A huttonholed common duct should be repaired by interrupted sutures, or the rent closed around a small rubber catheter or T-tube.

(2) *Secondary operations.* These may be the most difficult, time-consuming, and hazardous of all abdominal operations. They are usually performed some weeks or even months after the injury, when the patient is in poor health and in a bad condition to withstand the stress of a protracted operation. Jaundice may be intense, whilst wasting, muscular weakness, dehydration, and profound mental depression combined with a lack of desire to live, proclaim the gravity of the case. The greatest skill and the finest judgment are required during the dissection which is undertaken to restore the organs entangled in the mesh of adhesions as near as possible to their normal anatomical positions, to prevent serious or even irreparable damage to such vital structures as the portal vein or hepatic artery, and to identify and isolate the remaining proximal stump of the bile-duct before proceeding with the delicate operation of reconstruction of the duct. One of the following procedures may be indicated :

- (a) Choledocho- or hepatico-enterostomy.
- (b) Hepaticostomy (drainage of the dilated stump of the hepatic duct), possibly followed by (c) : or
- (c) Dissection of the fistula, followed by its anastomosis to the intestine.

*Rupture of the Gall-bladder or Bile Passages due to External Violence, Gunshot Wounds, Stabs, etc.* Traumatic rupture of the gall-bladder and bile-ducts is a rare condition and is usually associated with other intra-abdominal lesions, such as rupture of the liver or injury to the intestines. Such injuries are frequently due to run-over accidents, kicks in the abdomen, direct blows in the right hypochondriac region, stabs, gunshot wounds, etc. Pre-existing disease will make these structures more liable to injury.

Following the receipt of the injury there is often profound collapse, followed by shock and signs pointing to a serious abdominal catastrophe. Localisation of the exact site of the lesion is almost impossible, nor is it of great import as an exploratory operation is clearly indicated. The distended or pathological gall-bladder may rupture after very slight external violence, and in such cases a tender swelling localised to the right hypochondrium, may result. If the bile

is infected a localised abscess may form, and where there has been considerable extravasation of bile, general peritonitis usually supervenes after the lapse of three or four days.

In cases of injuries to the bile-ducts jaundice is usually present, and to a marked degree in 65 per cent of cases.

Rupture may involve the gall-bladder or the bile-ducts or both. Reference to figure 366 will show the sites at which rupture may occur.

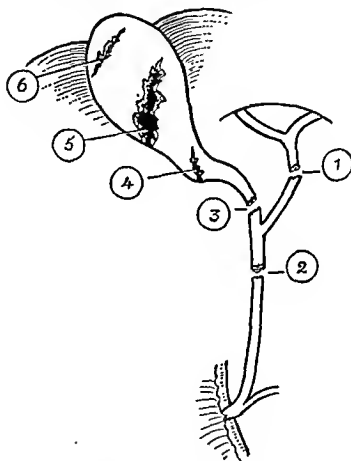


Fig. 366.—RUPTURE OF THE GALL-BLADDER AND DUCTS RESULTING FROM EXTERNAL VIOLENCE.

*Treatment.* When the *cystic duct* is torn across at *Point 3*, no useful purpose will be served by uniting the torn ends as subsequent stricture at this site will inevitably follow. The operation here advised is cholecystectomy. Likewise, when there is laceration of Hartmann's pouch or in the region of the neck of the gall-bladder (see *Point 4*), or where again the body of the gall-bladder is severely torn at *Point 5*, cholecystectomy should be performed. Rents involving the fundus, if small, may be closed by purse-string sutures or a series of interrupted Lembert sutures,

or the aperture may be used for draining the gall-bladder, i.e., cholecystostomy (see *Point 6*). After operations for rupture of the gall-bladder, whether the organ is removed or drained, a tube should be placed in Morison's pouch to deal with any leakage of bile or blood which may follow.

When the *common hepatic duct* is torn across (*Point 1*), the ends of the duct should be carefully mobilised and an end-to-end anastomosis performed over a rubber tube, or with the aid of a T-tube (fig. 367).

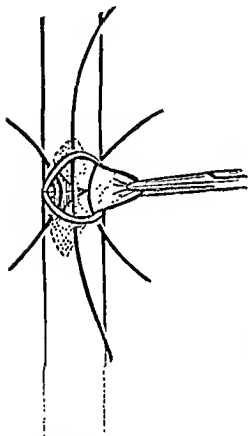


Fig. 367.—END TO-END ANASTOMOSIS OF SEVERED BILE DUCT WITH THE AID OF A RUBBER TUBE.

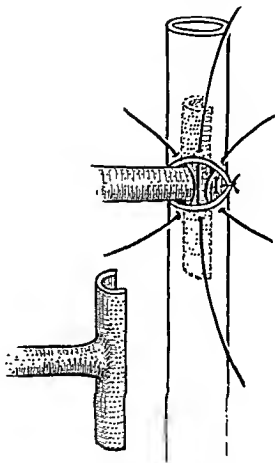


Fig. 368.—END TO-END ANASTOMOSIS OF SEVERED BILE DUCT WITH THE AID OF A T-TUBE.

When there is a complete rupture of the *common bile-duct*, as at *Point 2*, end-to-end anastomosis may be impossible as the lower portion of the common bile-duct often retracts behind the duodenum, and the anastomosis, when feasible, is often subjected to a great deal of tension. When, however, the gap is small and the parts are easily accessible, I would recommend end-to-end anastomosis over a T-tube, as depicted in figure 368. If it is not reasonably possible to suture the ruptured

common bile-duct, implantation of the proximal end of the duct into the duodenum by Walton's method is probably the best form of treatment. When the gall-bladder is healthy both ends of the duct may be ligatured, and the gall-bladder be anastomosed to the stomach (cholecysto-gastrostomy) or to the duodenum (cholecysto-duodenostomy).

## (2) PRIMARY CARCINOMA OF THE GALL-BLADDER AND BILE-DUCTS

*Gall-bladder.* Cancer of the gall-bladder is a commoner condition than it is generally estimated to be. Wilkie, in 1000 operations upon the gall-bladder and bile passages, records 32 cases of cancer. In two cases, in which a previous cholecystostomy had been performed for acute disease and in which gall-stones had been removed, he found at the second operation a recurrence of gall-stones and, in addition, cancer of the gall-bladder. Malignant disease of the gall-bladder is about three times more common in women than in men, and is most frequently seen after the age of 50, although cases occurring in younger patients have been recorded.

*It is often very difficult or even impossible to make a correct pre-operative diagnosis, as the condition in its clinical features so closely resembles gall-stones or chronic cholecystitis.*

Usually an operation is advised for cholelithiasis or chronic cholecystitis, and the true condition is only recognised when the gall-bladder is exposed.

Primary cancer of the gall-bladder may be of: (i) the villous or papillomatous type; or (ii) the infiltrating type. The growth may arise from: (i) the fundus; this is the part of the gall-bladder most liable to constant irritation from gall-stones, and certainly the commonest site for papillomata or adenomata to occur: (ii) the body; here an hour-glass gall-bladder may result: or (iii) the neck, where gall-stones so frequently become impacted. Where the disease is advanced the entire gall-bladder becomes involved in growth, and the exact site of origin is then impossible to determine (fig. 369).

Three varieties of malignant gall-bladder may be found at operation:

(i) The gall-bladder is chronically inflamed, and may or may not contain gall-stones. Cholecystectomy is performed and on slitting open the gall-bladder a small growth is detected. As might be expected, the results in such cases are excellent.

(ii) A portion of the gall-bladder is the seat of cancer which has spread and visibly involved an adjoining portion of the liver. Here

cholecystectomy combined with a wide wedge-excision of the adjoining portion of the liver is indicated, but the results of this operation are most discouraging. The majority of cases are dead within six months, and certainly not more than 5 per cent survive for longer than three years.

(iii) The gall-bladder is involved in growth which has already spread extensively into the liver or adjacent structures. In such cases nothing surgical can be attempted. The growth spreads very rapidly into the liver or involves neighbouring structures, and perforation into the colon, stomach, or duodenum may occur, or the gut become obstructed.

It is generally agreed that there is a definite relation between the incidence of gall-stones with cholecystitis and of primary cancer of the gall-bladder, and in approximately two-thirds of cases of cancer of this organ stones are found to be present. It is probable that the cholecystitis *per se* is a more important ætiological factor than the irritation produced by the gall-stones. Emphasis is again laid upon the fact that the symptoms in these cases are similar to those of cholecystitis or cholelithiasis. A diagnosis, however, may sometimes be aided by means of radiography. As a correct diagnosis is always difficult and the condition is by no means infrequent, cholecystectomy should, in the absence of obvious contra-indications, always be advised for patients who are suffering from gall-stones, recurrent biliary colic, or chronic cholecystitis. The early removal of diseased gall-bladders in such cases is the only sure method of preventing the onset of malignant degeneration.

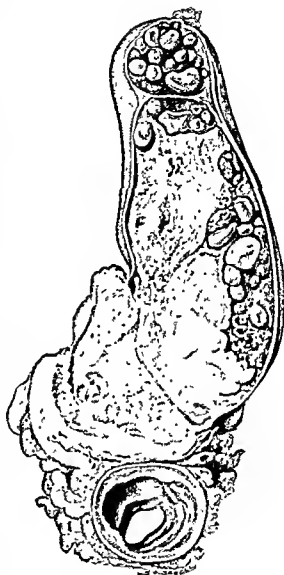


Fig 369.—CARCINOMA OF THE GALL-BLADDER.  
(Museum, Royal College of Surgeons)

*Bile-ducts.* Primary carcinoma of the bile-ducts is a rare disease. A number of cases, however, and particularly those in which the growth originates in the retro-duodenal portion of the common duct, have been labelled as cancer of the head of the pancreas. The signs and symptoms produced by cancer of the bile-ducts are often indistinguishable from those of stone in the common duct associated with obstruction, and all the complications which occur in such cases may be closely mimicked.

Reference to figure 370 will show the changes that usually occur when the growth is situated at various levels of the common ducts. Deepening and unrelenting jaundice is a constant feature, whilst the size of the gall-bladder will depend upon the position of the growth and the condition of the gall-bladder itself.

If previous to the onset of cancer the gall-bladder is the seat of chronic cholecystitis, it is incapable of distension. Again, it is collapsed when the growth is situated in the common hepatic duct and has not encroached upon the cystic duct. When the growth occurs at the junction of the three ducts the gall-bladder becomes filled with mucus. The distension of the gall-bladder may be enormous when it is not chronically inflamed, and the ducts may be distended to their fullest capacity above the point where the growth has produced complete obstruction.

The possibility of primary cancer of the bile-ducts should be borne in mind in all cases of obstructive jaundice which persist for over four weeks in patients who are at or past middle age. Whilst the occurrence of biliary colic in obstructive jaundice does not necessarily exclude the possibility of primary malignant disease of the ducts being present, the possible co-existence of gall-stones should be remembered. In a typical case the disease is evidenced by the rapid development of a complete afebrile jaundice in which there are bouts of epigastric distress, nausea, loss of appetite, and sometimes biliary colic.

In a patient past middle age presenting signs and symptoms of complete obstruction of the common bile-duct, where jaundice has persisted for more than one month, where the smooth rounded edge of the liver can be felt two or three finger-breadths below the costal margin, and where the distended gall-bladder—not tender on palpation—can be identified, the likelihood of malignant disease cannot be ruled out, and exploratory laparotomy should be undertaken without delay.

Treatment of the condition is advised as follows:

- (a) Cancer of the ampulla of Vater: (i) operable—excision; (ii) inoperable—cholecysto-jejunostomy.



(b) Growth situated in the common bile-duct: (i) operable—excision of growth, ligature of both ends of the duct, with cholecystogastrostomy or cholecysto-duodenostomy. If the gall-bladder has been removed previously or is the seat of advanced cholecystitis, the distal portion of the duct is ligatured and the proximal portion

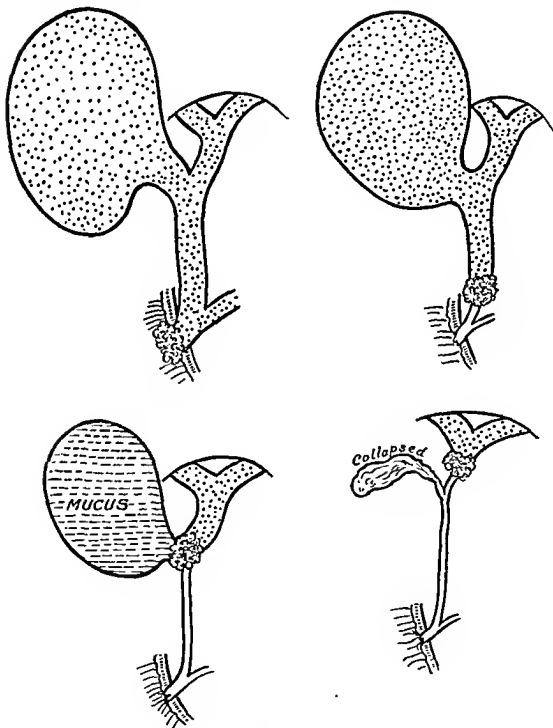


Fig. 370—CANCER OF THE BILE-DUCTS.

anastomosed to the duodenum—choleodocho-duodenostomy; (ii) inoperable—either cholecysto-gastrostomy, lateral choleodocho-duodenostomy, or hepaticostomy.

(c) Growth situated at the junction of the three ducts: (i) operable—excision of growth and gall-bladder, ligature of the distal end of the common bile-duct followed by hepatico-duodenostomy; (ii) inoperable—cholecysto-gastrostomy and hepaticostomy.

These cases (c, ii) are a great problem. The gall-bladder is ballooned and its walls are thin and friable, being stretched to their full capacity by the pent-up mucus. If a cholecystostomy and hepaticostomy are performed the patient will have two discharging sinuses, one of mucus from the gall-bladder and the other of bile from the hepatic ducts. In one such case upon which I operated I anastomosed the gall-bladder to the stomach and drained the common hepatic duct by means of a T-tube. The tube remained in position for four months, during which time the patient was free from jaundice and was comparatively comfortable.

Where the growth occludes the common hepatic duct very little can be done. Cancers situated here spread rapidly into the portal fissure and are nearly always inoperable. Hepaticostomy for the relief of jaundice is the most that can be attempted under the circumstances.

- (1) *The failure of medical treatment to prevent the onset of complications or to bring about a symptomatic cure.*

Surgery is here indicated to relieve troublesome or grave symptoms produced by a diseased gall-bladder and the complications which have resulted from the presence of stones.

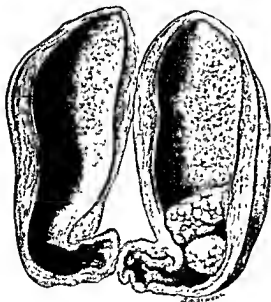


Fig. 371.—ACUTE CHOLECYSTITIS WITH GALL-STONES.  
(Museum, Royal College of Surgeons)



Fig. 372.—CALCULI IN THE BILIARY PASSAGES.  
(Museum, Royal College of Surgeons.)

- (2) *To remove a diseased organ which is potentially malignant.*
- (3) *To remove a focus of infection producing metastatic toxic effects upon distant organs and tissues.*

Wilkie has shown that in cases of chronic cholecystitis, with or without gall-stones, the heart muscle, the fascial planes, the joints, or the kidneys may be seriously damaged by being subjected to the infection which is present in the walls of the gall-bladder, and which is conveyed to distant structures via the blood stream.

*Contra-indications to Operation.*

- (1) *Senility and decrepitude.*
- (2) *Extreme obesity.*
- (3) *Advanced cardio-vascular disease or other serious complicating conditions such as severe diabetes, pulmonary tuberculosis, etc.*
- (4) *Pregnancy.*

*The choice of operation is, in part, governed by such considerations as :*

- (1) *The general condition of the patient.*
- (2) *The presence or absence of jaundice.*
- (3) *The presence or absence of acute inflammation of the gall-bladder and bile-ducts.*
- (4) *The position of the stones and the condition of the gall-bladder and bile-ducts.*
- (5) *The presence or absence of other intra-abdominal lesions.*
- (6) *The skill and experience of the surgeon.*

*The Condition of the Patient.* The choice of operation in cases of gall-stones is largely governed by the general condition of the patient. Where this is satisfactory, where there is no dehydration, no jaundice, no evidence of renal or hepatic insufficiency, and no marked emaciation, the risks of operation are slight. The majority of cases, however, are bad operative risks. They are fat, "flabby," breathless, dehydrated, and suffering from chronic toxæmia. In some the condition of the heart leaves much to be desired. A course of careful pre-operative treatment will do much to render these patients fit for surgery. It is common to find that the thin, tall type of patient fares well, whereas the fat and short type is a greater operative risk.

An icteric tinge will often denote the presence of stones in the common ducts, chronic sclerosing pancreatitis, growth, or cirrhosis. Wasting, too, is seen in these conditions, and may be very marked when there is a stone in the common duct, whether jaundice is present or not. Progressive loss of weight in the presence of unremitting jaundice is very suggestive of cancer of the head of the pancreas, but it is sometimes difficult to exclude the possibility of stone in the common



Fig. 373.—SKIAGRAPH SHOWING MULTIPLE OPAQUE GALL-STONES (CALCIUM), VISIBLE WITHOUT THE AID OF CHOLECYSTOGRAM.  
(Reproduced from "Modern Aspects of Gastro enterology," Baillière, Tindall and Cox. By kind permission of Dr. M. A. Arafat, Cairo University.)

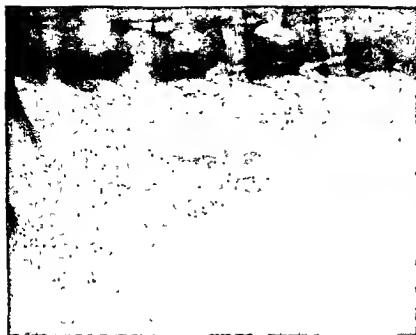


Fig. 374.—CHOLECYSTOGRAM. RADIOGRAM SHOWING TWO NON OPAQUE GALL-STONES (CHOLESTEROL), VISIBLE ONLY AFTER CHOLECYSTOGRAM.  
(Reproduced from "Modern Aspects of Gastro enterology," Baillière, Tindall and Cox. By kind permission of Dr. M. A. Arafat, Cairo University.)

bile-duct until the abdomen has been opened and a careful exploration conducted.

*The Presence or Absence of Jaundice.* In cases of gall-stones where there is no jaundice, the operative mortality is very low. Cholecystectomy in such cases has a death-rate of about 2-3 per cent. When jaundice is present, however, the death-rate is usually higher than 5 per cent, but it will, to a large extent, be governed by such factors as the skill and experience of the surgeon, the magnitude of the operation performed, and the general condition of the patient.

It is exceedingly difficult to decide exactly *when* such jaundiced cases should be submitted to operation. Is it preferable to wait until the jaundice is on the wane or has entirely subsided before operating, so as to diminish the risk of hæmorrhage and to avoid the dangers of hepatic insufficiency, or should operation be undertaken without delay? Although a few days' delay is usually advantageous, I consider that it is inadvisable to persevere with conservative measures under the following conditions :

- (1) When jaundice is ushered in *immediately* after a severe attack of colic. The danger of immediate operation in such cases is little, if at all, increased, provided that no other serious complications are present.
- (2) If jaundice persists and pain becomes more intense or is unrelieved by morphia, it is highly probable that stones are impacted. Where also there is additional evidence of suppurative cholangitis, immediate operative interference is advisable.
- (3) Where jaundice has been present for many days or weeks, where there have been no important diagnostic local signs, *but* where the patient's general condition is deteriorating, and the symptoms suggest that the dependent jaundice is due to obstruction of the duct by a stone, by a growth, or by a chronic sclerosing pancreatitis, further delay will augment the back pressure on the liver and the damage to the hepatic cells. If white bile is found in the common duct it will indicate that operation has already been unduly delayed.

A waiting policy may be recommended in *mild* cases of jaundice, particularly when there is a previous history of slight recurrent attacks of colic associated with short spells of jaundice. There is much to be gained in these cases by temporising and operating when jaundice is

declining or has subsided, as more radical and final operative measures are then safely possible.

*The Position of the Stones.* The choice of operation in cases of gall-stones is, in part, dependent upon the position of the stone or stones, the condition of the gall-bladder, and the presence or absence of jaundice. When a stone is impacted in the cystic duct the gall-bladder

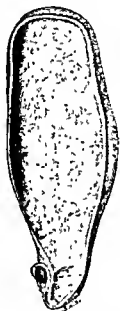


Fig. 375.—MUCOCELE OF THE GALL-BLADDER DUE TO IMPACTION OF A SMALL CALCULUS IN THE CYSTIC DUCT.

(Museum, Royal College of Surgeons.)



Fig. 376.—ACUTE OBSTRUCTIVE CHOLECYSTITIS DUE TO THE IMPACTION OF A STONE IN THE NECK OF THE GALL-BLADDER. CHOLECYSTECTOMY WAS PERFORMED UPON THIS CASE.

(Museum, Royal College of Surgeons.)

should always be removed if possible. If a cholecystectomy is not performed, but the stone is extracted by a process of "milking" the cystic duct, and the gall-bladder is drained, a stricture of the cystic duct at the site of the former impaction will frequently occur at a later date, producing symptoms indistinguishable from those caused by the impacted stone.

In certain cases of acute cholecystitis, where a stone is impacted in the neck of the gall-bladder, and where for one reason or another cholecystectomy is inadvisable, the stone may have to be extracted

by a process of "milking," usually with the index finger and thumb of the right hand working in unison with a scoop introduced into the gall-bladder and down to the site of impaction. Cholecystectomy will often be necessary at a later date in such cases.

*A mucocele of the gall-bladder* (fig. 375) is most frequently due to a stone impacted in the cystic duct, but occasionally to primary growth of the hiliary ducts or secondary malignant glands in the portal fissure. A mucocele is seen when a growth arises at the junction of the three ducts. When produced by a stone obstructing the cystic duct it is dealt with by cholecystectomy.

In cases of *acute obstructive cholecystitis* due to the impaction of a stone in the neck of the gall-bladder (fig. 376), the ideal operation is primary cholecystectomy. When, however, the patient is very ill, is toxæmic, the gall-bladder is much thickened and œdematous, and dissection of the cystic duct and artery is rendered impossible on account of dense inflammatory adhesions, the organ should be opened in the region of the fundus, the impacted stone or stones removed, and the gall-bladder drained. Morison's pouch should also be drained, and the abdominal wound only partly closed as subsequent suppuration is almost inevitable.

*Stones in the Gall-bladder in the Absence of Jaundice.* In cases of cholelithiasis in the absence of jaundice and of acute inflammation of the gall-bladder, cholecystectomy is now universally recognised as the operation of choice, and in such cases the common bile-duct should also be explored. There are certain cases where, although gall-stones can be palpated in the gall-bladder, the organ itself looks normal, and the surgeon is therefore tempted merely to perform a cholecystostomy. It has been proved beyond dispute, however, that better results are to be obtained by removal of the gall-bladder, as, in spite of its normal appearance, this organ will almost invariably be found to be the seat of chronic inflammatory changes when submitted to microscopic scrutiny.

*Stones in the Gall-bladder in the Presence of Jaundice.* In these cases cholecystectomy is often inadvisable, owing to the difficulty in controlling the oozing which takes place from the liver bed and from numerous vascular adhesions. When the gall-bladder is distended and contains stones a cholecystostomy should be performed, and if in addition calculi are felt to be impacted in the common duct, a supra-duodenal choledochostomy will also be required. If, however, the gall-bladder



is small, shrivelled, and fibrosed, numerous adhesions exist which hide the common duct, and a calculus can be felt in the common duct, the dissection of the structures over the duct should be limited to a minimum. In cases such as these, Moynihan's method of *rotation of the duct* may be employed with advantage for extraction of the stone.

*If there has been complete obstruction associated with deep jaundice and severe back pressure on the liver, particularly if on aspiration the duct is found to be distended with white bile, and the stone is firmly impacted, we agree with Wilkie that "discretion will dictate simple drainage of the duct, leaving the removal of the stone to a second operation when jaundice is relieved and hepatic function restored."*

When jaundice is slight or absent a deliberate operation can be undertaken. The exposure here must be complete, all adhesions must be carefully divided, and dissection carried out until the three ducts are clearly visible. The fundus of the gall-bladder and Hartmann's pouch are grasped with forceps and pulled upwards. The pylorus and duodenum are retracted downwards, and the common bile-duct is put on the stretch. Before opening the duct it is most essential to place a gauze pack underneath it into the foramen of Winslow, to prevent any septic bile from reaching the lesser sac.

The field of operation is carefully packed off, and an incision one-third of an inch long is made through the anterior wall of the common bile-duct, just below the point where the cystic duct joins the two common ducts. The infected bile that wells up in the wound is removed by suction or rapidly mopped up and the edges of the wound are retracted by two stay sutures.

The forefinger and thumb of the left hand being used as a guide and support, the stones are removed by scoops or forceps, and the patency of the papilla is established by passing a Liston sound or Desjardins forceps into the duodenum. *After the removal of the stone or stones from the common duct, the papilla should be well dilated with graduated sounds.*

If the duct contains biliary mud, gravel, or inflammatory debris, this should be removed by irrigating the duct with saline solution through a soft rubber catheter attached to a large Record syringe. Irrigation is continued until the fluid returns quite clear, and all this returning fluid is removed by suction. At the completion of the irrigation the gall-bladder is removed and the common bile-duct is drained.

I often use a large open-ended metal suction tube for passing down the common duct to the ampulla where stones are so commonly

overlooked, and this tube, which is attached to an electrical suction apparatus, is most useful in removing all debris and even impacted stones (fig. 377).

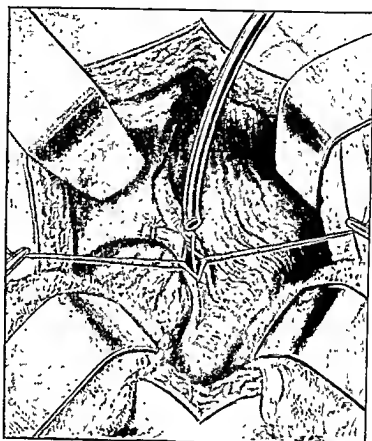


Fig. 377.—REMOVAL OF DEBRIS FROM THE COMMON DUCT. A LARGE OPEN-ENDED METAL SECTION TUBE IS ABOUT TO BE PASSED DOWN THE COMMON DUCT TO THE AMPULLA OF VATER WHERE STONES ARE SO COMMONLY OVERLOOKED. NOTE THE GAUZE PACK WHICH IS INSERTED UNDER THE DUCT AND INTO THE FORAMEN OF WINSLOW TO BLOCK THIS OPENING AND TO TRAP ANY INFECTED MATERIAL COMING FROM THE COMMON DUCT WHEN IT IS OPENED. THE PLACING OF THE SWAB IN THE POSITION SHOWN AND PROPER SUCTION TECHNIQUE PREVENT CONTAMINATION OF THE SUB-HEPATIC SPACES.

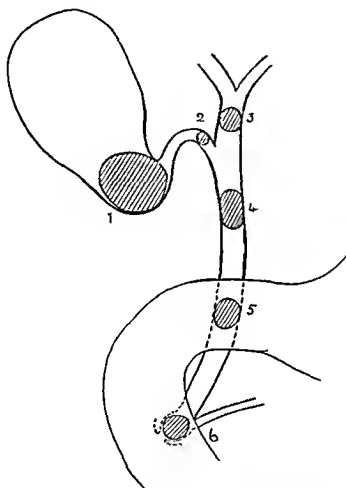
(After Lahey, modified.)

*Stones in the common bile-duct.* Gall-stones may become impacted in any portion of the biliary passages (fig. 378). They are found in the bile-ducts much more frequently than published statistics would suggest.

Stones in the common bile-duct will most frequently be found in cases of long-standing infection of the gall-bladder associated with gall-stones. In my experience it is the small stones lodged in the ampulla which are so often overlooked, and it is in just these cases that the employment of suction has proved so valuable.

The majority of stones in the common duct can be removed through an incision made in the supra-duodenal portion. It is only on rare occasions that the retro-duodenal or trans-duodenal approach becomes necessary.

The absence of jaundice is by no means a reliable argument against



*Fig. 77N.*—POSITIONS IN WHICH CALCULI MAY BECOME IMPACTED IN THE GALL-BLADDER OR BILE PASSAGES

- 1—A large calculus impacted in Hartmann's pouch, occluding the neck of the gall bladder.
- 2—Stone in the cystic duct.
- 3, 4 and 5—Calculi in the common ducts.
- 6—Stone in the ampulla of Vater.

exploration of the duct for possible stone, and the ducts should be explored upon suspicion, even when no calculi can be palpated. It should be remembered that some 25 per cent of patients with stones in the common bile-duct have never had jaundice, and some 5 per cent do not even give a history of pain.

*Whenever stones are discovered in the gall-bladder and cholecystectomy is indicated the surgeon should make it a practice to explore the common bile-duct.*

This is recommended on the following grounds :

- (a) In some 50 per cent of cases " recurrent " stones in the common bile-duct are actually stones which have been overlooked at the first operation.
- (b) It is often impossible to detect minute calculi or biliary sand, or soft pultaceous pigment stones, which may form a nidus for further deposition, even after the most painstaking and methodical palpation of the ducts.
- (c) Stones impacted in the lower portion of the common duct may be mistaken for indurated pancreatic nodules or lymphatic glands unless the duct is probed.
- (d) In some cases it is difficult to decide whether the duct is dilated or not until it has been incised.
- (e) Although a visual examination of bile aspirated from the common duct will often yield valuable information, it may, at times, be very misleading. Turbid bile is usually purulent, but clear bile also may be purulent.

When the exploration of the ducts and the dilatation of the ampulla is completed, the surgeon will have to decide whether the opening in the duct should be completely closed by suture (*internal biliary drainage*) or whether a choledochostomy (*external biliary drainage*) should be performed. In most cases it is wiser to adopt the latter procedure.

The only contra-indications to exploration of the bile-ducts in cases of cholelithiasis are :

- (a) Acute obstructive cholecystitis in some cases where, owing to marked vascularity and œdema of the parts, this added procedure proves impossible.
- (b) Severe jaundice associated with considerable back pressure and white bile, the stone being firmly impacted in the lower reaches of the common duct. (See page 649.)
- (c) Where at the completion of an obligatory cholecystectomy the patient's condition is unsatisfactory and it is obvious that the additional operation of exploration of the common duct is more than the patient's strength will allow.

The technique of the operations for exploring the common bile-ducts will be discussed in greater detail at the end of this chapter.

#### (4) THE TREATMENT OF CHOLECYSTITIS

*Chronic Cholecystitis.* There is no doubt that a large number of patients with *early* chronic cholecystitis can be rendered symptom-free by efficient medical or spa treatment. In a well-established case, however, operation is clearly indicated, as the disease is indolent, and the organisms which are lodged in the walls of the diseased gall-bladder are difficult or impossible to destroy by medical measures, and constitute a focus of infection which produces metastatic toxic effects upon distant organs and tissues (Wilkie, *Med. Soc. Trans.*, p. 105, 1930). The diseased gall-bladder is responsible for many disagreeable dyspeptic symptoms and much ill-health, and is a potentially malignant organ. In addition to this, chronic cholecystitis is very often associated with gall-stones, and the serious complications which result from their migration or impaction are by no means infrequent.

All available statistics prove quite conclusively that the ideal treatment for a case of chronic cholecystitis is removal of the gall-bladder. Cholecystostomy is reserved for those cases where excision would appear too hazardous, or where the patient's condition only warrants the simplest procedure possible under the circumstances.

The difficult cases are those in which the patient gives a definite history of recurrent attacks of severe colic, flatulent dyspepsia, and other symptoms pointing to disease of the gall-bladder, in which a dye test has given no gall-bladder shadow, and where at operation the gall-bladder appears to be normal. Early disease of the gall-bladder is evidenced by slight thickening of its wall, a change in its colour from sea-green to a greyish white, increase in the subserous fat, a disappearance from view of the fine blood-vessels which course over the viscus, enlargement of the cystic lymph gland, and the presence of adhesions.

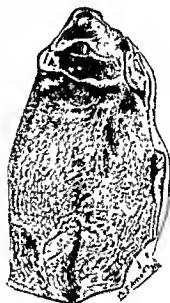


FIG. 379.—A TYPICAL SO CALLED STRAWBERRY GALL-BLADDER—CHOLESTEROSIS.

(Museum, London Hospital)

In cases where there is no evidence of chronic appendicitis, gastric or duodenal ulcer, or any other organic intra-abdominal lesion, it is better to remove the gall-bladder than to compromise by draining it. Cholecystostomy in such cases is quite useless. Such gall-bladders when removed and examined will often be found to be the seat of early cholesterosis (fig. 379). The late results in these cases, however, are not invariably satisfactory.

*Acute Cholecystitis.* It is generally necessary to consider each case as an individual problem. In some cases an immediate operation is advisable; in others a policy of delay is clearly indicated.

All surgeons are agreed that during an acute fulminating attack of cholecystitis in which the pain is controlled with difficulty or not at all, where peritonitis, localised to the right hypochondrium, is present, or where a swollen gall-bladder can be palpated, immediate operation should be performed to forestall perforation and to overcome the lethal effects of a protracted acute toxæmia.

Where acute cholecystitis occurs in cases of typhoid or paratyphoid fever immediate cholecystectomy should be performed, as perforation is such a common complication of this disease.

Young patients in good condition should, as a rule, be subjected to operation after a brief preparation without waiting for the attack to subside.

A plea for *immediate operation* is made by those who have been impressed by the great variation in the pathological changes discovered in patients presenting apparently the same condition clinically, and by the difficulty in deciding which cases will subside under conservative treatment and which will become progressively worse. There is also abundant proof that *at the time of operation* advanced grades of inflammation of the gall-bladder may exist in the complete absence of clinical signs and symptoms, and that it is often very difficult, or even impossible, to determine the exact nature and extent of the inflammatory lesion before the abdomen has been opened.

*The advantages of immediate cholecystectomy in cases of acute cholecystitis are claimed to be:*

- (a) The prompt removal of a useless, damaged, and infected organ.
- (b) That the operation in experienced hands presents no particular difficulty as the gall-bladder can be shelled out with ease on account of the surrounding peri-cholecystic œdema.
- (c) An otherwise necessary secondary operation is avoided.

- (d) Convalescence is shorter, smoother, and attended by fewer complications. There is, in addition, less pain, and fewer dressings are required.

There are, however, many surgeons who hold the view that it is usually inadvisable to operate during the acute phase of an attack of cholecystitis owing to the oedema and congestion of the base of the right lung which is almost invariably present, to active peri-cholecystitis, to associated hepatitis, and to increased vascularity of the parts. The exceptions are the fulminating cases and where the acute symptoms become progressively worse or do not subside within 24 hours, under which circumstances immediate operation is advised.

*The outstanding indications for adopting conservative measures are as follows :*

- (a) Very obese patients and those who are poor operative risks. In these poorer risks, usually the aged, the obese, and those with serious cardiac conditions, it is wiser to allow the acute attack to subside; but the surgeon should nevertheless be ready to intervene if the progress of the case is unfavourable.

Many cases in which expectant measures fail to yield satisfactory results are the most difficult of all at operation.

- (b) The attack has been very mild and the patient is seen many days after the onset of the illness. In these cases where perhaps there are certain evidences that the disease is subsiding, much will be gained without any particular danger by waiting for the process to resolve. A few weeks (not less than four) after the subsidence of the acute condition a more deliberate operation—often cholecystectomy—can be undertaken; whereas, during the acute stage of the disease, more often than not only drainage of the gall-bladder is possible.
- (c) There are serious intra-thoracic complications such as pneumonia or pleurisy.
- (d) There is a palpable mass over the gall-bladder region, but the pain is not severe, toxæmia is either slight or absent, and the discase, which may have been present for several days, is running a more or less apyrexial course. In such cases delay rather than immediate operation is advised, as the inflammatory mass will usually subside, thus diminishing the difficulties of subsequent operation.

In cases where conservative measures have been adopted the following *danger signals* would indicate the necessity for proceeding with immediate operation :

- (a) There is a persistence of temperature, local tenderness, and rigidity.
- (b) There is severe pain which is not easily controlled by morphia or other measures.
- (c) The toxæmia is increasing.
- (d) There is a rising leucocyte count ; the white blood count is over 15,000 per cubic millimetre.
- (e) The pulse-rate is also steadily rising.

*Uncertainty of diagnosis is one of the main indications for abandoning delayed treatment.*

In summing up, it may be said that the mild cases will usually subside under expectant treatment ; that patients presenting more acute symptoms should be kept under observation for 24 hours before any decision as to operation is made ; and that the very severe cases should be submitted to operation without delay.

Wherever possible, cholecystectomy is the operation of choice, cholecystostomy being performed only where the patient's general condition makes it imperative that the simplest and quickest surgical procedure should be undertaken, or where removal of the gall-bladder is rendered impossible on account of surrounding vascular adhesions and œdema.

*Perforation of the gall-bladder* has been estimated to occur in from 1-3 per cent of all cases of biliary disease.

Judd and Phillips have reported a series of 61 cases of perforation of the gall-bladder ; in all but two cases there was an associated localised abscess. Cholecystectomy was performed in 48 of the 61 cases with five deaths, and cholecystostomy in 13 cases with one death. The mortality, therefore, for operations performed upon the perforated gall-bladder is high, as the above figures show.

Perforation of the gall-bladder in acute cholecystitis associated with gall-stones is a comparatively rare condition and will often have to be distinguished from a high-lying appendix, perforated peptic ulcer, or acute pulmonary disease. Perforation is due to ulceration or local gangrene of a part of the wall of the gall-bladder. The gall-bladder itself never perforates as a result of distension with bile, but



may as a result of the thrombosis of the blood-vessels supplying its walls become gangrenous and perforate in acute cholecystitis in the absence of stones.

Perforation is usually associated with localised peritonitis. A local abscess forms in the region of the gall-bladder and is well shut off from the general peritoneal cavity. General peritonitis very rarely occurs.

Perforation of the gall-bladder may be subdivided into three groups :

(a) Chronic perforation in which there is a fistulous communication between the gall-bladder and some other viscus, usually the duodenum or colon.

(b) Sub-acute perforation. Here the perforated gall-bladder is surrounded by an abscess which is walled off by adhesions from the general peritoneal cavity.

(c) Acute perforation in which rupture has occurred with leakage into the general peritoneal cavity. The peritonitis which has resulted is due to a lack of protective adhesions.

It is very difficult to make a correct pre-operative diagnosis as there are no pathognomonic signs or symptoms of a perforated gall-bladder. If a localised abscess is present most surgeons prefer in the first instance to drain the abscess, deferring any set operation upon the gall-bladder until some future occasion.

#### (5) BILIARY FISTULA

*Internal.* Here the gall-bladder communicates with the duodenum, stomach, transverse colon, or common bile-duct. The commonest causes of internal biliary fistula are :

- (a) Gall-stones.
- (b) Carcinoma of the gall-bladder, pyloric region of the stomach or hepatic flexure of the colon.
- (c) Chronic duodenal ulcer.

*External.* A temporary external biliary fistula follows the operation of cholecystostomy. If, however, the fistula does not close spontaneously within a few days after removal of the tube it should be inferred that the common bile-duct is kinked owing to the gall-bladder being attached to the anterior abdominal wall, or that a stone is

present in the cystic duct or in the common bile-duct, obstructing the normal outflow of bile.

Other conditions, such as stricture of the common bile-duct and growth involving this duct, likewise maintain an external discharge of bile after cholecystostomy. If, on the other hand, the bile passages are free an external biliary fistula should close spontaneously within a few days. An external biliary fistula also results when a gall-stone ulcerates through the parietes.

*The Treatment of Internal Biliary Fistula.* When the condition found at exploratory laparotomy is due to cancer of the gall-bladder, stomach or colon, it is only on the most exceptional occasions that any prospect of cure by radical operation is possible. The primary disease is usually widespread, the matting extensive, and any attempt at separation of the viscera will cause severe hæmorrhage, diffusion of cancer cells, and possibly a fatal peritonitis from leakage of bile or gastro-intestinal contents.

When the fistula is due to gall-stones the two viscera which are in communication should be carefully separated, and the aperture in the stomach duodenum or colon closed in such a manner that no narrowing of the gut results. After exposing the common ducts and probing them to make sure that they are patent, the gall-bladder is removed and the common duct is drained or, alternatively, cholecystostomy alone is performed.

*The Treatment of External Biliary Fistula.* If a fistula has followed the operation of cholecystostomy for gall-stones, excision of the gall-bladder and exploration of the ducts is indicated. If, however, the obstruction is due to an impassable stricture of the common ducts, such as might result from an inoperable cancer of the common bile-duct or an extensive sclerosing pancreatitis, after the gall-bladder has been freed from its attachments to the anterior abdominal wall and sufficiently mobilised, it should be anastomosed to the stomach or duodenum.

#### (6) OBSTRUCTION OF THE COMMON BILE-DUCT

Causes :

*Intrinsic.*

- (a) Conditions *inside* the lumen of the gut : gall-stones, biliary mud, inflammatory debris, mucus, intestinal parasites, foreign bodies, etc.

- (b) Conditions in the wall of the duct: (i) new growths—innocent and malignant; (ii) stricture resulting from the ulceration produced by a calculus or external trauma; (iii) obliteration of the duct due to operative trauma; (iv) cholangitis.

*Extrinsic.*

- (a) Chronic sclerosing pancreatitis.  
(b) New growths, such as cancer of the pyloric end of the stomach or of the head of the pancreas.  
(c) Pressure on the duct by abdominal tumours, e.g. hydatid cyst or hepatoma.

*Treatment.* When the common bile-duct is obstructed by conditions inside the lumen of the gut, such as gall-stones, foreign bodies (e.g. a portion of a drainage-tube), intestinal parasites, etc., these objects should be removed by supra-duodenal or trans-duodenal choledochotomy. In the majority of cases the obstructing body can be coaxed to the accessible first portion of the duct, but if this proves to be impossible it is wiser to extract it by Kocher's operation of trans-duodenal choledochostomy.

When obstruction is due to conditions in the wall of the duct itself, such as new growths, stricture, or obliteration of the duct, a variety of methods is available whereby to restore the flow of bile into the alimentary tract. Waltman Walters (*Surg., Gynec., and Obstet.*, p. 235, Feb., 1933) offers the following conclusions as to the treatment of these conditions:

"If sufficient normal duct remains above the stricture to enable its accurate anastomosis without tension to an opening made in the duodenum, thus obtaining union of mucous membrane to mucous membrane, results will be excellent, provided the liver is in satisfactory condition. Failure to obtain such a result can be attributed directly to inaccurate anastomosis, to severe infection of the parenchyma of the liver, or to infection in the walls of the biliary tract itself; infection in either of the structures mentioned may be accompanied by sand-like calculi within the biliary passages.

"If the stricture or tumor is small, or is situated directly adjacent to the liver, with normal duct both proximal and distal to the structure or tumor, excision of the lesion, with subsequent direct anastomosis of the ends of the duct, probably will be a satisfactory procedure, and can be expected to be followed in most cases by good results. Such is

not usually the case, however, if the scarred portion is merely incised and allowed to remain, even though the lumen of the duct at this point is increased by a plastic procedure of the Heimeke-Mikulicz type. This may be explained by the fact that the remaining scar tissue continues to contract.

"If the amount of duct which remains exterior to the liver is not sufficient to allow of either procedure, an external biliary fistula can be established and later coned out and transplanted into the stomach or duodenum. This may be expected to be followed with good results in some instances and fairly good results in others.

"The frequency with which small tumors of the ampulla of Vater cause obstructive jaundice should not be forgotten. Since the tumor is usually small and of low degree of malignancy, producing symptoms early and metastasis late, it thereby lends itself readily to transduodenal removal. . . .

"Excellent results have been obtained in many cases in which accurate anastomosis has been made between the remaining portion of the duct and the duodenum or between the severed ends of the duct itself, and in which biliary fistulas have been transplanted into the stomach or duodenum. For such procedures to be successful too much infection must not exist in the hepatic parenchyma, in the intra-hepatic ducts, or in the remainder of the extra-hepatic duct above the stricture.

"Failure to obtain lasting good results could almost be predicted at the time of anastomosis, if the liver has a marked cirrhotic appearance, if the stump of the duct is unduly thickened, or if purulent bile or granular stones are present within the hepatic ducts."

Where there is an irremovable, impermeable stricture of the common bile-duct, whether this be of inflammatory, traumatic, or of neoplastic origin, if the gall-bladder is present, contains bile, and its walls are flexible, this organ should be anastomosed to the stomach or duodenum whichever seems the more accessible.

In cases of long-standing cholangitis the bile passages may become sclerosed and strictured. At operation on such a case, a woman of fifty-three who had had a cholecystectomy performed for gall-stones three years previously, the cord-like common bile-duct and hepatic duct were isolated after laborious dissection, and the attenuated, thick-walled contracted common duct felt like the vas deferens and proved very difficult to incise; but when this was done it was obvious that only the finest silver-wire probe could be passed into its lumen. It was incapable of dilatation, so an attempt was made to drain the duct by passing the smallest ureteric catheter upwards within its lumen

towards the liver, in order to establish external hiliary drainage ; but even this proved an impossibility. A drainage-tube was therefore led down to the incision in the duct to afford biliary drainage, after which the abdominal wound was closed. An external hiliary fistula resulted, with disappearance of the previously very pronounced jaundice and general improvement in the patient's health.

When obstruction is due to extrinsic causes, such as a chronic sclerosing pancreatitis or carcinoma of the head of the pancreas, the operation of cholecysto-gastrostomy or cholecysto-duodenostomy is advised.

There is a relatively common group of complicated cases which sometimes cause difficulty in the selection of the most appropriate treatment. Stones are found in a gall-bladder which is not sclerosed and which contains bile, and after extracting calculi from the gall-bladder and common duct, the head of the pancreas is felt to be definitely thickened. In such cases it is advisable to anastomose the gall-bladder to the duodenum or stomach after draining the common duct.

#### (B) PRE-OPERATIVE TREATMENT

*Expectant or Delayed Treatment for Acute Cholecystitis.* Here the patient is put to bed and maintained in the Fowler position. Complete starvation is enforced during the first 24-36 hours of the treatment. Hot fomentations are applied to the right hypogastrium, or heat is applied to the liver and gall-bladder region by a diathermy apparatus. No purgatives or enemata are allowed. The urine and feces are examined daily. The temperature and respiration- and pulse-rates are recorded two-hourly on a separate chart. Numerous examinations of the blood are required to detect the presence of acidosis or alkalosis and to determine any degree of leucocytosis which may be present. The blood urea should be estimated and liver function tests performed. The results of these tests will influence the amount of fluid, and the choice of the drugs which should be introduced into the circulation.

When toxæmia or dehydration is marked, *intravenous* salines should be administered. The necessary amount of water, sugar, and salts should be introduced into the circulation by the intravenous drip method. Intravenous injections of *cylotropin* (Schering), one ampoule (5 cc.) once a day for three days and then on alternate days, may be helpful and should be given a trial. Fluids by mouth and glucose are prescribed when there are evidences that the condition is subsiding.

*For Chronic Cholecystitis with or without Gall-stones.* These patients should be carefully prepared for operation and all septic foci eradicated. If there is marked obesity a systematic reduction in weight should be effected by dieting and exercises. Such patients may lose two or three stone in the course of a few months, which, in addition to considerably improving their general condition, greatly facilitates the performance of the operation. Thyroid extract may be administered in suitable cases.

Prior to operation, cases of cholelithiasis should be kept on a *cholesterol-free diet* in which the following are avoided: yolk of eggs in any form, including cakes and sweets made with eggs; cream, cheese, kidney, liver, sweetbread, brain, duck and goose; the fat of meat, suet, pork, sausages, high game. As little butter as possible is taken. The administration of hexamine and magnesium sulphate for at least a week before operation and for a few weeks afterwards not only makes the operation safer, but greatly reduces the risk of post-operative complications, both immediate and remote.

There are many cases of *mild* chronic cholecystitis which can be rendered symptom-free by means of biliary antiseptics and the stimulation of biliary drainage on the lines laid down by Hurst, and I have found his treatment very useful in the management of cases awaiting operation and in the immediate post-operative period. This treatment may be outlined as follows:

(a) *Hexamine.*

Hexamine, 100 grs.

Sod. bicarb., 60 grs.

Sod. citrate, 60 grs.

t.d.s. in water or milk.

The irritation of the urinary bladder with doses of hexamine exceeding 30-60 grs. a day is due to formalin being set free by the acid urine. This can be prevented by making the urine alkaline. As hexamine acts as a biliary antiseptic in spite of the alkalinity of the bile, its efficacy is not reduced by giving alkalis although these prevent it from acting as a urinary antiseptic.

(b) *Epsom Salts.* These should be taken in concentrated solution, fasting, one hour before breakfast every morning. The largest possible dose should be given short of causing diarrhoea, and no other aperient should be taken. This causes reflex contraction of the gall-bladder and bile-ducts with relaxation of the sphincter of Oddi.

(c) *Olive Oil*, two tablespoonfuls three times a day before meals, has the same effect as Epsom salts.

*The Pre-operative Treatment of the Jaundiced Patient.* Large quantities of fluid and glucose should be given by mouth and intravenously, as dehydration and hepatic insufficiency are common in jaundiced patients. Where large quantities of sugar have been administered it is the practice of some surgeons to give suitable doses of insulin. The intravenous drip method, in which Ringer's solution and 5-10 per cent glucose is administered, is excellent in combating cholæmia, in restoring the function of the liver, and in flushing out the kidneys. Heat applied to the liver by hot stupes or by a diathermy apparatus to promote hepatic activity is also beneficial.

In cases of jaundice associated with cholangitis I have found intravenous injections of cytotropin efficacious. In a severe case 5 cc. is injected intravenously daily for the first three days, after which 5 cc. is injected on alternate days. It is important when giving the injection intravenously to warm the ampoule up to blood heat and to inject the solution very slowly. These injections often have a marked effect in lowering the temperature and in diminishing the jaundice in cases of acute suppurative cholangitis.

The coagulation time of the blood should be decreased by :

- (a) Intravenous injections of 5 cc. of 10 per cent calcium chloride, or 10 cc. of a 10 per cent solution calcium gluconate (Sandoz) given intravenously daily on three consecutive days.
- (b) 30 cc. of 30 per cent sodium citrate injected intramuscularly immediately before operation.
- (c) Blood-transfusions. Small doses—250-300 cc.—are given on one or two occasions.
- (d) Auto-hæmatotherapy. 10 cc. of blood should be withdrawn from the patient's arm and injected into the gluteal region once daily for three days.

## (C) TECHNIQUE OF THE OPERATIONS UPON THE GALL-BLADDER AND BILE-DUCTS

The following operations will be described :

- (1) *Operations upon the Gall-bladder.*
  - (a) Cholecystotomy (cholecystendysis).
  - (b) Cholecystostomy.

- (c) Cholecystectomy.
    - (i) Partial.
    - (ii) Complete : *a.* from the fundus.  
*b.* from the cystic duct end.
  - (d) Cholecysto-enterostomy.
    - (i) Cholecysto-gastrostomy.
    - (ii) Cholecysto-duodenostomy.
    - (iii) Cholecysto-jejunostomy.
- (2) *Operations upon the Bile-Ducts.*
- (a) Choledochotomy and choledochostomy.
    - (i) Rotation of the duct by Moynihan's method.
    - (ii) Supra-duodenal choledochotomy.
    - (iii) Retro-duodenal choledochotomy.
    - (iv) Trans-duodenal choledochotomy.
      - a.* MacBurney's operation—duodeno-choledochotomy or ampullary choledochostomy.
      - b.* Kocher's operation—trans-duodenal choledochostomy.
  - (b) End-to-end anastomosis—choledocho-choledochostomy.
  - (c) Side-to-side anastomosis—lateral choledocho-enterostomy.
  - (d) End-to-side anastomosis—choledocho- or hepatico-duodenostomy and reconstruction operations.

#### GENERAL CONSIDERATIONS

(1) *Instruments.* Some of the more important instruments that are used in gall-bladder surgery, such as Moynihan's cholecystectomy forceps, Desjardins' forceps, Deaver's retractors, Mayo's trocar and cannula, gall-bladder scoops, rubber and other catheters, etc., need no special description, but are here illustrated. (See figs. 380, 381, 382, 383, 384, 385 and 386.)

Silk or linen thread is very rarely used in gall-bladder surgery, except for ligaturing the cystic duct and the cystic artery, and for the continuous sero-muscular stitch in cholecysto-gastrostomy.

(2) *The Position of the Patient upon the Operating Table.* Usually the same position is adopted as in other abdominal operations, but it is advantageous to insert a sand-bag or tripartite rubber bag (fig. 387) under the lower ribs, or to elevate the bridge which is present in some



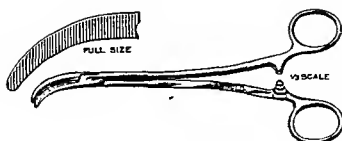


Fig. 390.—MOYNIHAN'S CHOLECYSTECTOMY FORCEPS.



Fig. 391.—DESJARDINS' GALL-STONE FORCEPS.



Fig. 392.—DEAVER'S RETRACTOR.



Fig. 393.—MAYO'S TROCAR AND CANNULA.



Fig. 394.—CHEATE'S GALL-STONE SCOOP.

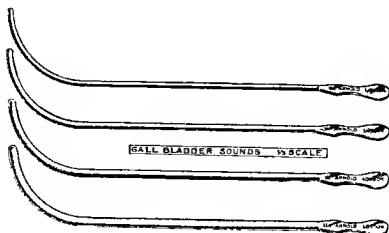


Fig 385.—LISTON SOUNDS FOR DILATING THE COMMON DUCT.

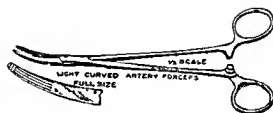


Fig 386.—SMALL ARTERY FORCEPS.

operating tables so as to arch the body slightly and throw out the epigastric region, and thus bring the lower edge of the liver and gall-bladder forward. In thin patients the intestines sink downwards towards the pelvis, and when the liver is rotated the gall-bladder and bile-ducts can be brought almost to the level of the abdominal wound. The whole table may be tilted to about  $5^{\circ}$  or  $10^{\circ}$  so that either the head or the feet are raised a few inches. Marked hyperextension of the body is to be avoided as it tends to cause respiratory embarrassment and chest complications.

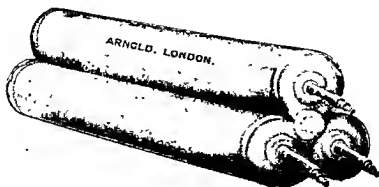


Fig 387.—TRIPARTITE RUBBER BAG.

(3) *Incisions* (fig. 388).

- (a) Paramedian incision.
- (b) Kocher's oblique subcostal incision.
- (c) Right transverse incision.
- (d) Vertical transrectus or muscle-split incision.

The two most popular incisions are : (a) the *paramedian incision*, in which the upper half of the right rectus muscle is retracted outwards ; and (b) *Kocher's incision*.

I prefer the paramedian incision in thin patients or where the costal angle is very narrow. Kocher's incision is employed for all other cases and is especially suitable for obese patients.

For secondary operations also, or where there has been a previous paramedian incision, Kocher's subcostal incision would be the one of choice. This incision starts just below the tip of the xiphisternum and proceeds downwards and outwards for 4-6 inches, 1½ inches below the right costal margin. In order to give ample access it is essential to divide the right rectus muscle completely. After the skin incision has been made, the muscles and extra-peritoneal tissues are infiltrated with a local anæsthetic in order to facilitate the division of the muscles of the lateral abdominal wall and to aid relaxation (fig. 389). The eighth and ninth dorsal nerves will be encountered at the outer border of the rectus muscle. It is often impossible to save the small eighth nerve, but the larger ninth must be dissected out for an inch or two and carefully preserved.

At the completion of the operation, to permit of easy suture of the wound, the bridge in the operating table is lowered, or the sand-bag or tripartite bag is removed. The wound is closed with two continuous

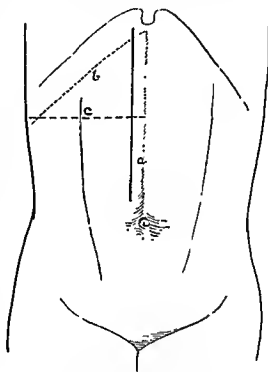


Fig. 388.—INCISIONS USED IN GALL-BLADDER AND BILE-DUCT SURGERY.

- a = Right paramedian incision.
- b = Kocher's incision.
- c = Right transverse incision.

sutures of No. 1 or No. 2 20-day chromic catgut, and a few tension sutures may be required.

When the patient is obese, in order to facilitate exposure of the ducts and to make all intra-abdominal manipulations easier, the incision through the skin and subcutaneous tissues is made very much longer than that through the muscles.

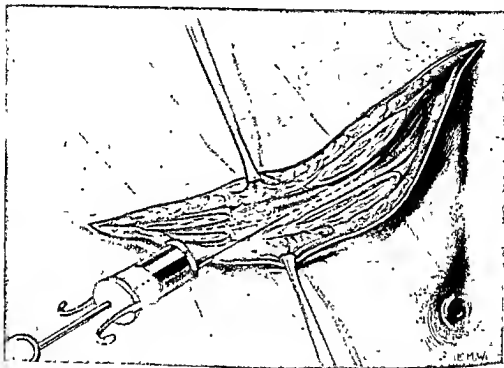


Fig. 389.—Kocher's Incision.

(4) *Rotation of the Liver.* Moynihan has shown that a much better approach to the ducts can be obtained by rotating the liver. The edge of the liver is seized and pulled firmly downwards and inwards towards the umbilicus, and then turned upwards and slightly outwards so as to expose its under-surface. A hand slipped between the liver and the diaphragm will, on occasions, permit of easy rotation of the liver. Should rotation be difficult or impossible, the gall-bladder and bile-ducts can be rendered more accessible by packing the space between the liver and diaphragm with gauze.

(5) *Exploratory Laparotomy.* Just before opening the peritoneal cavity tetra-cloths are fixed to the skin edges. As soon as the peritoneal cavity is opened, a general exploration should at once be carried out and the gall-bladder and bile-ducts be examined last of all.

Where the patient is very ill, is jaundiced, or where there is acute inflammation of the gall-bladder, this general exploration is omitted. Adhesions between the gall-bladder and adjacent viscera are separated and three packs are placed in position; the one on the right-hand side fills Morison's pouch, and the one on the left is so placed that the pylorus and duodenum are covered and retracted out of the way. The middle swab is placed over the colon (fig. 390).

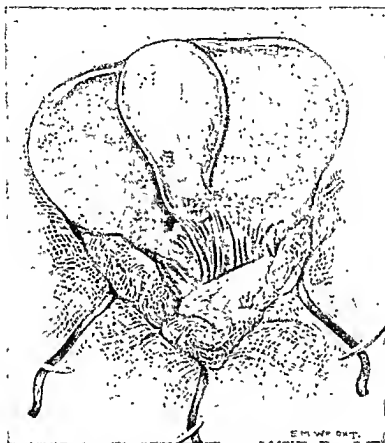


Fig 390.—EXPOSURE OF THE GALL-BLADDER. NOTE THE POSITION OF THE THREE SWABS USED FOR PACKING OFF THE VISCERA

In operations upon the common duct another swab is carefully placed so as to occlude the foramen of Winslow and to prevent any contamination of the lesser sac. In introducing these swabs great pains must be taken to place them into position gently so that they do not in any way traumatise the parietal peritoneum. Trauma to the parietal peritoneum produces a great deal of shock and encourages post-operative adhesions.

The wound edges are protected with mackintosh squares, and the three large packs are themselves often protected by a further layer of dental dam or mackintosh. The operative field should be isolated as

far as possible, and all the surrounding structures well protected from infectious material which may come from the gall-bladder or bile-ducts during the operations performed upon them. The best retractor in gall-bladder surgery is an assistant's hand well placed (fig. 391).

The assistant should pass his left hand into the wound and retract the stomach and duodenum to the left so as to keep these structures out of the way and to put the common duct on the stretch. Deaver's retractors are useful; one may take the place of the hand, whilst the other keeps the intestines away from the field of operation (fig. 392).

There must be no chilling and no undue exposure of the liver during a protracted operation upon the gall-bladder or bile-ducts. All adhesions must be very carefully separated or divided between ligatures

#### OPERATIONS UPON THE GALL-BLADDER

##### *Cholecystotomy*

In this operation, which is sometimes termed *cholecystendysis*, after the contents of the gall-bladder have been evacuated through an incision in the fundus, the wound is closed and the gall-bladder is returned to the abdomen without drainage.

The operation is sometimes employed, even at the present time, in cases where gall-stones are discovered in the course of some abdominal operation, such as ovariectomy. It is, however, only feasible where the walls of the gall-bladder, although possibly diseased, are still flexible.

After the gall-bladder region has been isolated with gauze swabs, a purse-string suture is inserted around the circumference of the fundus and an incision is made within the space thus demarcated. The contents of the gall-bladder are removed, and due care is taken in determining that the neck of the gall-bladder and the cystic duct are free from calculi. If stones are discovered here, they should be "milked" back into the gall-bladder.

After the stones have been removed through the incision in the fundus, the interior of the gall-bladder is explored with the finger, and any biliary mud or debris is washed out with warm saline solution, after which the incision in the fundus is closed by tying the purse-string suture. A few interrupted Lembert sutures should also be inserted to reinforce the invaginated area.

"The patient is thus relieved of her gall-stones with a minimum addition of trauma to the original operation, whereas *cholecystectomy* piles one major procedure on top of another." (Victor Bonney.)

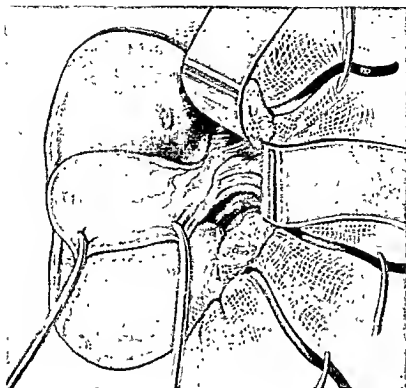


FIG. 392.—EXPOSURE OF THE GALL-BLADDER AND OPERATIVE AREA. DEANER'S RETRACTORS ARE SHOWN IN POSITION. CHOLECYSTECTOMY IS ABOUT TO BE PERFORMED.

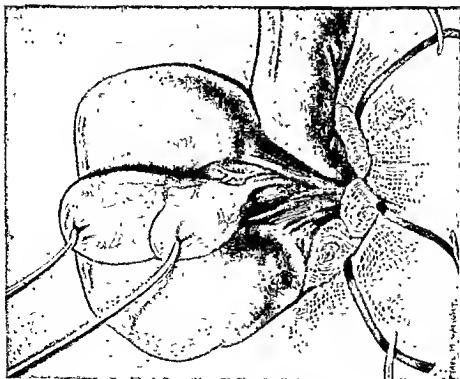


FIG. 391.—THE BEST RETRACTOR IN SIGHT OF THE GALL-BLADDER AND DUCTS IS AN ASSISTANT'S HAND WELL PLACED. CHOLECYSTECTOMY IS ABOUT TO BE PERFORMED.

*Cholecystostomy*

The main indications for performing this operation may be summarised as follows :

- (a) Acute or chronic cholecystitis, with or without gall-stones:
  - (i) Where the patient is very aged and infirm.
  - (ii) Where cholecystectomy presents great technical difficulties or involves undue risk.
  - (iii) Where the condition of the patient is grave, toxæmia being pronounced or other complications being present.
- (b) As an additional therapeutic measure in acute hæmorrhagic pancreatitis.
- (c) As a preliminary measure in cases of acute cholecystitis and suppurative cholangitis associated with obstruction of the common bile-duct.

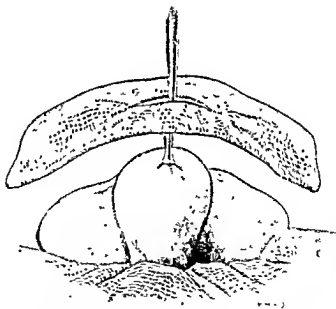


Fig 293.—CHOLECYSTOSTOMY. MOYNIHAN'S METHOD OF ISOLATING THE SITE OF THE INTENDED CHOLECYSTOSTOMY FROM THE REMAINDER OF THE OPERATIVE FIELD.

The edges of the wound and the region around the gall-bladder should be well protected with abdominal pads and mackintosh squares, as the bile is often highly infective in cases in which cholecystostomy is performed. If the gall-bladder is enlarged another mackintosh sheet, with a hole of sufficient size to admit the fundus, is taken and



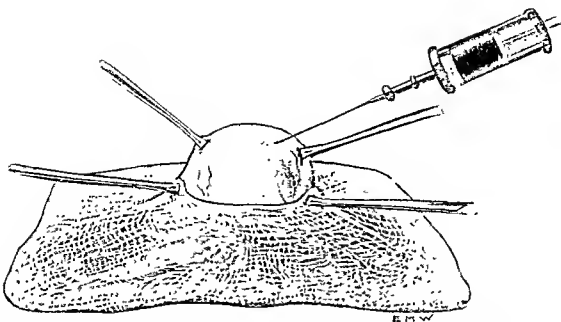


Fig. 331.—CHOLECYSTOSTOMY. THE CONTENTS OF THE GALL-BLADDER ARE ASPIRATED WITH A WIDE BORE NEEDLE ATTACHED TO A LARGE SYRINGE, PRIOR TO THE PERFORMANCE OF CHOLECYSTOSTOMY.

fitted snugly around the gall-bladder where it is fastened by Allis forceps, thus completely isolating the site of the intended cholecystostomy from the remainder of the operation area.

The contents of the gall-bladder are now aspirated with a wide-bore aspirating needle attached to a large Record syringe, or if the gall-bladder is unduly distended the isolated fundus should be pierced by Mayo's trocar and cannula and the bile drained into a small receptacle.

The fundus must be seized with Allis forceps on either side of the puncture spot to prevent the gall-bladder from retracting when it is empty, and also to prevent any leakage when the needle is withdrawn (fig. 334). An incision of sufficient length to admit the finger is then

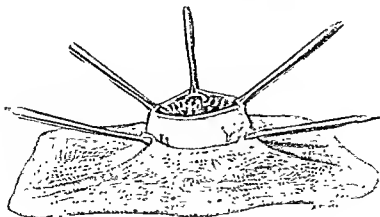


Fig. 335.—CHOLECYSTOSTOMY. MOYSHIN'S METHOD.

made through the fundus (fig. 395). A suction tube is introduced into the gall-bladder and the septic bile and inflammatory debris are withdrawn, or the contents may be mopped up with small gauze swabs. The gall-bladder should now be empty and collapsed unless its walls are thickened and rigid with inflammatory exudate. The index finger of the left hand is then passed under the cystic duct and neck of the gall-bladder, and the stones are worked upwards with the fingers towards

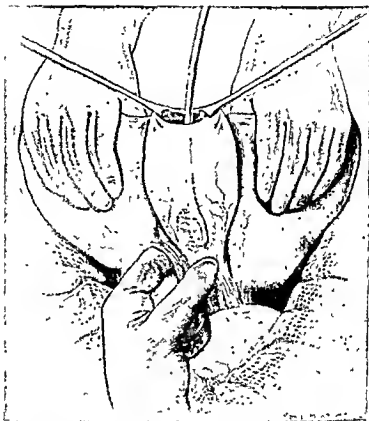


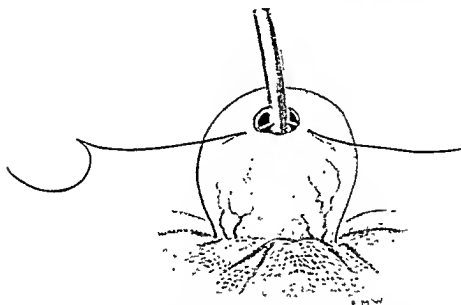
Fig. 396.—CHOLECISTOSTOMY. EXTRACTION OF STONE FROM HARTMANN'S POUCH BY MEANS OF DESJARDINS FORCEPS.

the opening. The calculi may be expressed through this opening or extracted from the gall-bladder with special scoops or forceps (fig. 396). When no further stones can be palpated, either in the cystic duct or in the gall-bladder, the forefinger of the left hand is introduced into the gall-bladder to feel if any fragments or grit remain. If so, these may be removed by passing strips of gauze into the gall-bladder, packing them in firmly and then withdrawing them. Small particles will become entangled in the gauze meshes and can thus be extracted.

If there is much inflammatory debris or putty-like substance, the

gall-bladder should be gently irrigated with warm saline by means of a rubber catheter attached to a syringe. The returning fluid should be mopped up at once or aspirated with a suction tube.

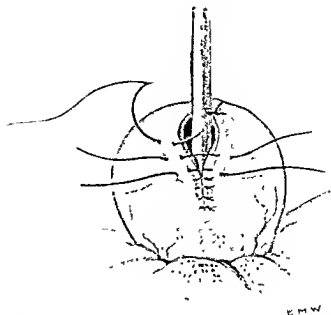
When calculi are felt to be impacted in the cystic duct, it is usually possible to "milk" them back into the gall-bladder. This is done by passing the finger and thumb of the right hand down and along the outer side of the neck of the gall-bladder until the finger-tip enters the foramen of Winslow. In this way the lowest part of the cystic duct is reached. From this point the fingers are worked gently upwards, pushing any stones which may be encountered back into the gall-



*Fig. 397.*—CHOLECYSTOSTOMY. A RUBBER TUBE HAS BEEN INSERTED INTO THE GALL-BLADDER AND ANCHORED IN POSITION BY A STITCH WHICH IS INTRODUCED IN THE MANNER DEPICTED IN THIS FIGURE.

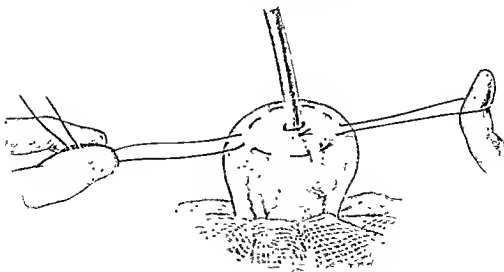
bladder. If a stone is firmly lodged in the cystic duct, firm pressure is applied with the finger and thumb to its lower end, thus coaxing it back into the gall-bladder. If the cystic duct or the neck of the gall-bladder is inadvertently torn during this manoeuvre cholecystectomy should be performed.

When the surgeon has made quite sure that no more stones remain in the gall-bladder and that the cystic duct is patent, a rubber tube with an outside diameter of about a third to half an inch is passed for two or three inches into the gall-bladder and secured by a stitch to the opening, in the manner shown in figure 397. This stitch, when tied, anchors the rubber tube to the gall-bladder, and further tends to invaginate a portion of the opening. The incision in the gall-bladder



*Fig. 398.*—CHOLECYSTOSTOMY. IN CASES WHERE THE WALL OF THE GALL-BLADDER IS CONSIDERABLY THICKENED, THE INCISION IS BEST CLOSED BY A SERIES OF CLOSELY APPLIED INTERRUPTED LEMBERT SUTURES.

may now be closed by a series of interrupted Lembert sutures (fig. 398), or by means of one or two purse-string sutures. When the walls of the incision are very thick or friable, interrupted sutures will have to be used. When, on the other hand, the walls are flexible and firm, purse-string sutures are to be preferred (figs. 399 and 400).



*Fig. 399.*—CHOLECYSTOSTOMY. INTRODUCTION OF THE FIRST PURSE-STRING SUTURE.

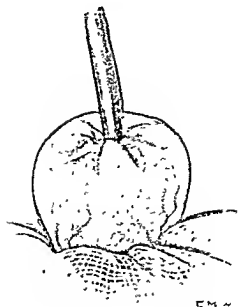


Fig. 400.—CHOLECYSTOSTOMY. THE PURSE STRING SUTURE HAS BEEN TIED.

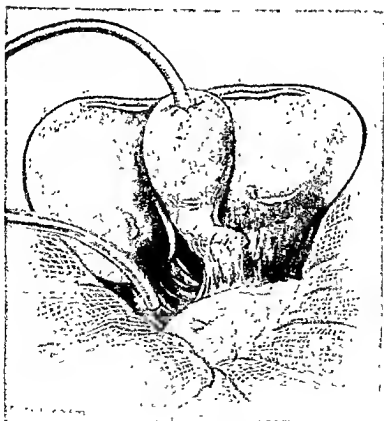


Fig. 401.—CHOLECYSTOSTOMY. THE OPERATION IS COMPLETED BY INSERTING A DRAINAGE TUBE BELOW THE GALL-BLADDER TOWARDS MORISON'S POUCH.

Another rubber tube or a piece of corrugated rubber is then placed below the gall-bladder in case there is any leakage (fig. 401), and the abdominal wound is closed.

### *Cholecystectomy*

#### *Indications :*

- (a) Most cases of traumatic rupture of the gall-bladder.
- (b) Mucocoele of the gall-bladder due to stone impacted in the cystic duct.
- (c) Carcinoma of the gall-bladder. Here the operation often includes a wedge-excision of a portion of the liver.
- (d) Most cases of internal and external biliary fistula, and especially mucous fistula following cholecystostomy.
- (e) Cholecystitis, with or without gall-stones :
  - (i) Strawberry gall-bladder.
  - (ii) Most cases of chronic cholecystitis.
  - (iii) Most cases of acute cholecystitis in the absence of complications.
  - (iv) Volvulus of the gall-bladder.
  - (v) Empyema of the gall-bladder.
  - (vi) Gangrene of the gall-bladder.

*Partial cholecystectomy* may be advised in certain cases where gangrene appears to be limited to the fundus and body of the gall-bladder. Here the separation commences at the fundus but no attempt is made to dissect out the cystic duct or to identify the important structures in that region. The dissection and mobilisation proceeds no further than to the neck of the gall-bladder, and very often only to a point proximal to this. Here two pairs of Kocher forceps are applied transversely side by side to the gall-bladder, which is divided between them and removed. The remaining stump is closed by a few interrupted mattress sutures, which are introduced in such a manner as to control the bleeding (fig. 402). A rubber tube is inserted towards the cystic duct through that portion of the gall-bladder which is not closed by the sutures (fig. 403). A corrugated rubber drain is laid over the suture line and led out through the abdominal incision.

*Technique of Cholecystectomy.* The gall-bladder may be removed by starting the dissection from the fundus end or from the cystic

duct end. When the operation commences by dissection from the fundus end, blood oozes from the raw surface of the liver and obscures the field of operation, rendering the dissection and isolation of the duct and artery extremely difficult. I prefer, therefore, to start the operation by isolating the cystic duct.

After dividing any adhesions which may exist between the gall-bladder and adjacent viscera, the three abdominal packs are placed in position. The liver is rotated and the wound is retracted in such a way that a clear view of the field of operation is afforded. If the gall-bladder is distended its contents should be aspirated.

Either two ring forceps or two hæmostats are then applied to the fundus and to Hartmann's pouch, these being held in the surgeon's left

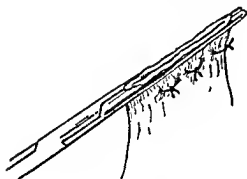


Fig. 402.—PARTIAL CHOLECYSTECTOMY.

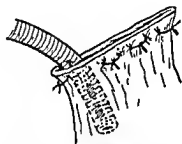


Fig. 403.—PARTIAL CHOLECYSTECTOMY. THE STUMP OF THE GALL-BLADDER IS PARTLY CLOSED, AND A TUBE HAS BEEN INSERTED DOWN TO THE NECK OF THE GALL-BLADDER.

hand and drawn upwards and on wards so as to put the gall-bladder and cystic duct on the stretch. (See fig. 392.) A small incision is now made in the peritoneum over the cystic duct, and the sero-fatty tissues in this region are carefully dissected away from the duct until the whole length of the duct can be clearly defined (fig. 404).

The dissection now proceeds a little further in order to display the common hepatic duct, the common bile-duct, and the point where the cystic duct joins the common ducts. The cystic duct is never ligatured until it has been traced upwards into the neck of the gall-bladder and inwards to the point where it unmistakably joins the common ducts.

*At this stage the three ducts must be clearly displayed and demonstrated to the assistant.*

The common ducts are then carefully palpated to determine the presence of stones, and the head of the pancreas is likewise examined for any evidence of chronic pancreatitis.

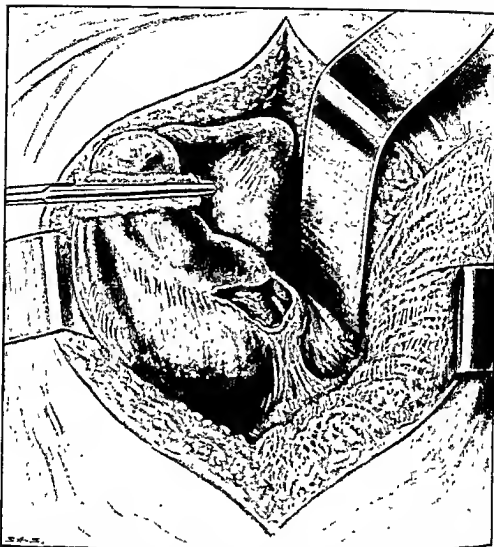


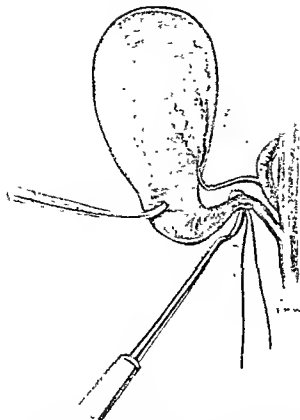
Fig. 404 —CHOLECYSTECTOMY IN A CASE OF ACUTE CHOLELITHIASIS. DISSECTION OF THE CYSTIC DUCT HAS BEEN COMMENCED, BUT IS NOT COMPLETED. BEFORE LIGATURING THE CYSTIC DUCT ITS JUNCTION WITH THE COMMON HEPATIC DUCT AND COMMON BILE-DUCT MUST BE CLEARLY DEMONSTRATED. THE ARTERY ABOVE THE CYSTIC DUCT IS, IN ALL PROBABILITY, THE CYSTIC ARTERY; BUT THIS TOO MUST BE CLEARLY DEFINED BEFORE IT IS LIGATED.

NOTE THE METHOD OF GRASPING THE FUNDS FOR PURPOSES OF RETRACTION IN CASES WHERE THE GALL-BLADDER IS ACUTELY INFLAMED.

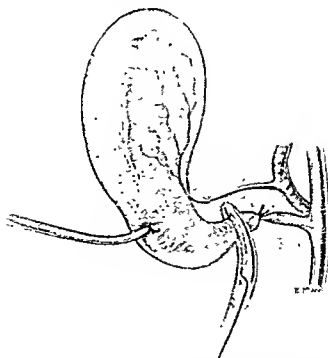
The cystic duct is then rendered as taut as possible, and an aneurysm needle, threaded with a long strand of No. 1 20-day chromic catgut, silk, or linen thread, is passed underneath the duct as near to the neck of the gall-bladder as possible (fig. 405), and the duct is tied securely in two places. The proximal ligature is then cut short, the distal one being left long.

Moynihan's cholecystectomy forceps are applied to the neck of the gall-bladder (fig. 406) and the duct is severed as close to the forceps as

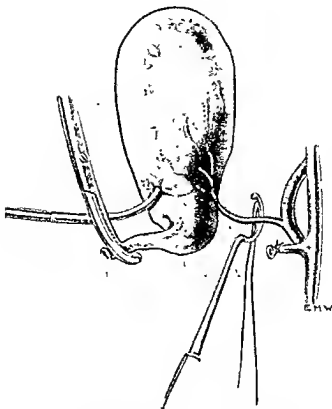




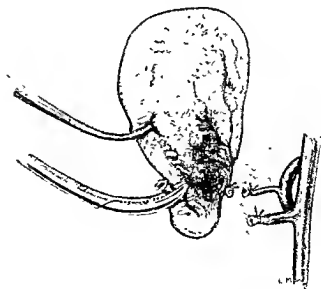
*Fig. 403.*—CHOLECYSTECTOMY. AN ANESTHESM NEEDLE, THREADED WITH CATGUT, SILK, OR LINEN THREAD, IS PASSED UNDER THE CYSTIC DUCT WHICH IS THEN LIGATURED CLOSE TO THE GALL-BLADDER.



*Fig. 404.*—CHOLECYSTECTOMY. LIGATURE OF THE CYSTIC DUCT WITH MOYNIHAN'S CHOLECYSTECTOMY FORCEPS IN POSITION, PRIOR TO THE DIVISION OF THE DUCT.



*Fig. 407.*—CHOLECYSTECTOMY. METHOD OF LIGATURING THE CYSTIC ARTERY.



*Fig. 408.*—CHOLECYSTECTOMY. THE CYSTIC DUCT AND CYSTIC ARTERY HAVE BEEN TIED AND DIVIDED, PRIOR TO THE REMOVAL OF THE GALL-BLADDER.

possible. The neck of the gall-bladder is then dissected free for a short distance and drawn upwards out of the way while a careful search is made for the cystic artery.

We have already discussed the various anatomical points in connection with the cystic artery, and how the right hepatic artery, or even the right hepatic duct, may be mistaken for it and be inadvertently ligatured.

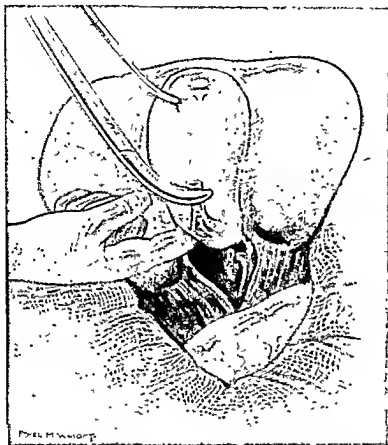
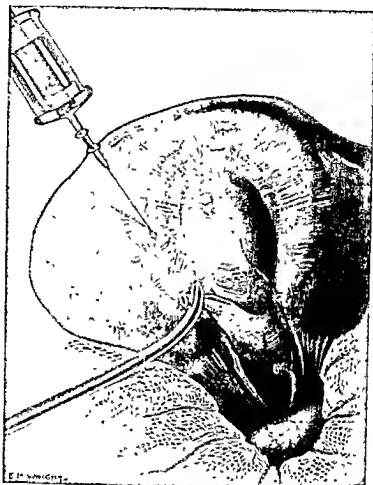


Fig. 402.—CHOLECYSTECTOMY. THE GALL-BLADDER IS STRIPPED FROM ITS BED BY THE INDEX FINGER OF THE RIGHT HAND.

As a rule the cystic artery lies in a more posterior plane than the cystic duct, slightly above it, and in close proximity to the liver. A little dissection in this obscure part will reveal the artery travelling towards the gall-bladder. *It must be traced to the point where it enters the wall of the gall-bladder, usually near the neck.* When it has been isolated an aneurysm needle, threaded with No. 1 20-day chromic catgut or linen thread, is passed behind it and tied in two places, well away from the right hepatic artery and common hepatic duct and as close to the gall-bladder as possible (fig. 407).

The artery is then divided between the ligatures (fig. 408). The artery should not be grasped with forceps, as in their use there is danger of picking up a portion of the ducts with the points and thus leading to button-holing or some other injury to the ducts.



*Fig. 410.*—CHOLECYSTECTOMY. SALINE IS INJECTED UNDER THE PERITONEAL REFLECTION OF THE GALL-BLADDER TO FACILITATE ITS SEPARATION FROM THE LIVER, AND TO SIMPLIFY THE SUTURING OF THE EDGES OF THE PERITONEUM ACROSS THE GALL-BLADDER FOSSA.

The forceps which grasp the cystic duct are then drawn upwards and the index finger of the right hand is introduced under the gall-bladder to strip it away from its bed (fig. 409).

As the separation of the gall-bladder proceeds upwards, the peritoneal reflections on either side are divided with scissors or with a scalpel, and in order to facilitate the division of this peritoneal reflection saline solution may be injected under the peritoneum where it is reflected from the gall-bladder on to the liver. The saline lifts up the

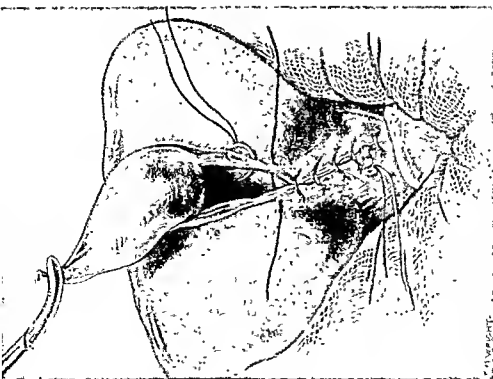


Fig. 411.—CHOLECYSTECTOMY. THE GALL-BLADDER IS USED AS A RETRACTOR WHILE THE PERITONEUM IS SUTURED OVER THE GALL-BLADDER FOSSA. A CONTINUOUS SUTURE MAY BE USED INSTEAD OF THE INTERRUPTED STITCHES.

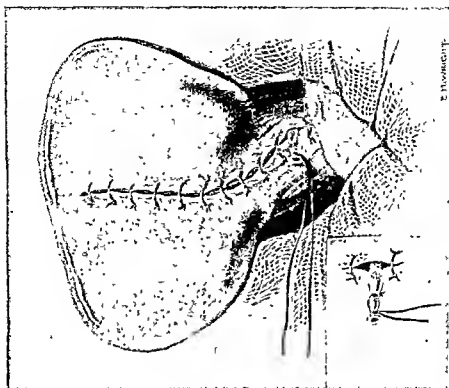
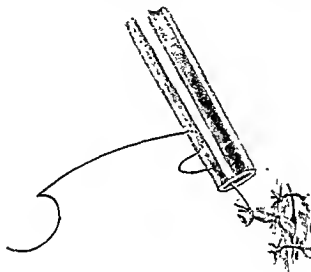


Fig. 412.—CHOLECYSTECTOMY. THE GALL-BLADDER FOSSA HAS BEEN PERITONIZED. NOTE THE CYSTIC DUCT TIED IN TWO PLACES, AND THE DISTAL LIGATURE LEFT LONG, EITHER TO BE TIED AROUND OR STITCHED TO A LARGE DRAINAGE-TUBE.

peritoneum all around the attached portion of the gall-bladder in a large wheal and makes its separation a simple and bloodless procedure (fig. 410).

A few large veins which are usually situated about the middle and on either side of the body of the gall-bladder are divided during this peritoneal separation, the bleeding points being picked up with mosquito forceps and ligatured.

The gall-bladder fossa is covered over by stitching the cut edges of peritoneum together by a series of interrupted sutures, whilst the raw surface over the common ducts is likewise peritonised, but the



*Fig. 413.—CHOLECYSTECTOMY. THE DRAINAGE TUBE IS STITCHED TO THE STUMP OF THE CYSTIC DUCT IN SUCH A WAY THAT WHEN THE SUTURE IS DRAWN TIGHT THE STUMP OF THE CYSTIC DUCT WILL LIE WITHIN THE LUMEN OF THE TUBE.*

stump of the cystic duct should be allowed to protrude between the lower sutures. These interrupted sutures are introduced as the gall-bladder is being dissected free, the gall-bladder itself acting as a retractor (fig. 411). The distal ligature on the cystic duct, which is long (fig. 412), is either tied round the lower portion of a rubber drainage-tube or is stitched to it in the manner shown in figure 413.

This tube, which has an outside diameter of about  $\frac{1}{2}$ – $\frac{1}{4}$  inch, and which has either lateral openings or has been slit down one side, is placed against the sutured gall-bladder fossa down to the stump of the cystic duct.

Drainage of the sutured gall-bladder fossa is never omitted in this operation, as if bile escapes into the general peritoneal cavity it is prone

to become infected and give rise to septic peritonitis. The tube is left *in situ* for four or five days, as there may be leakage of bile from an unrecognised severed accessory hepatic duct or from the raw surface of the liver, and however carefully the operation is performed, such leakage is apt to occur from time to time.

If the common duct has been drained the two tubes are brought out, either through the top or bottom end of the incision, or through a separate stab wound. Both tubes are stitched to the skin to prevent their being accidentally withdrawn.

### *Cholecysto-enterostomy*

In this operation an anastomosis is made between the gall-bladder and some portion of the intestinal tract in order to overcome an obstruction of the common bile-duct. It is an efficient way of dealing with the obstruction, but the immediate and the late results will depend upon the exact nature of the lesion. For instance, when the operation is performed for obstruction of the common duct due to a chronic sclerosing pancreatitis, the resulting benefit is immediate and lasting; whereas, in cases of cancer of the head of the pancreas the operative mortality is high (over 30 per cent) and the improvement is seldom maintained for more than a few weeks.

In the following order of preference the gall-bladder may be anastomosed to:

- (a) The stomach—cholecysto-gastrostomy.
- (b) The duodenum—cholecysto-duodenostomy.
- (c) The jejunum—cholecysto-jejunostomy.

In certain cases of inoperable cancer of the ampulla of Vater, of the duodenum, or of the pyloric end of the stomach, cholecysto-jejunostomy may be the only operation possible. It may also be indicated where approximation of the stomach or duodenum to the gall-bladder, for purposes of anastomosis, presents unusual difficulty.

The main *complications* of the operation of cholecysto-enterostomy are:

- (a) Leakage of bile from an inaccurately sutured line or on account of some of the sutures cutting out when tension is excessive. Septic peritonitis soon follows such leakage.
- (b) Serious hæmorrhage, especially in jaundice cases where the gall-bladder or duodenum has been mobilised prior to anastomosis.

*Indications.* The operation is indicated for irremovable or impermeable stricture of the common bile-duct, which may be due to:

- (1) Diseases of the head of the pancreas.
  - (a) Chronic pancreatitis.
  - (b) Carcinoma.
- (2) Lesions of the common bile-duct.
  - (a) Stricture following the impaction of a calculus.
  - (b) Primary malignant disease.
  - (c) Obliteration of the duct following injury.

In early cases of *chronic sclerosing pancreatitis* where jaundice is mild and the patient's condition is good, the results of the operation are very satisfactory. Jaundice disappears, appetite is regained, and the patient puts on weight. In certain cases it is impossible to determine whether the thickening of the head of the pancreas is inflammatory or neoplastic in nature. If on aspirating the contents of the gall-bladder thick black bile is withdrawn, it will indicate that there has been no serious hepatic damage as the result of back pressure, and that the operation offers a good prospect of relief. In late cases, however, where thin, limpid, white bile is withdrawn into the syringe, the outlook is exceedingly grave, and the operative mortality after cholecysto-enterostomy in such cases is not less than 30 per cent.

If on aspiration of the gall-bladder white bile is found, the surgeon will have to make sure that there is no obstruction of the cystic duct, as, if obstruction is present, there will, of course, be no amelioration of the jaundice following the short-circuit operation.

In suspected cases of *carcinoma of the head of the pancreas* associated with obstruction of the common bile-duct, the performance of cholecysto-gastrostomy is always worth while, as there is the possibility that the diagnosis may be wrong. The hard, craggy mass felt in the head of the pancreas may be due to a localised chronic pancreatitis, to a gumma, or to secondary inflammatory chronic fibrosis consequent upon an extensive, deeply-excavating, posteriorly-placed duodenal ulcer.

In cases of impassable *stricture of the common bile-ducts*, if the gall-bladder is present and is not grossly diseased, cholecysto-enterostomy should be performed. Where, however, the gall-bladder has been previously removed or is markedly thickened, choledcho-enterostomy is recommended.



(1) *Cholecysto-gastrostomy*. Approach to the gall-bladder is made through a right paramedian or Kocher incision. The distended gall-bladder will at once present in the wound, and before an examination is conducted of the three ducts, of the head of the pancreas, and of other viscera, the gall-bladder must be emptied of its contents. The gall-bladder is isolated with abdominal swabs and mackintosh squares, and Mayo's trocar and cannula is introduced through the ventral aspect of the fundus at the site chosen for the anastomosis. The pent-up fluid contained in the gall-bladder is allowed to drain into a receptacle at the patient's side, and this fluid is examined and its physical characters noted. White bile indicates that an unrelieved obstruction has produced severe back pressure effects and damage to the hepatic cells, and is an ominous sign. When white bile is found, the cystic duct will have to be displayed and carefully examined to make sure that it is not occluded. The presence of normal or thick turbid dark bile within the gall-bladder indicates that the bile is passing freely into the gall-bladder and that the anastomosis is likely to produce a good result.

When the gall-bladder has been completely emptied and lies collapsed and flaccid, the common ducts and the head of the pancreas must be carefully examined to ascertain as far as possible the nature and the extent of the causative lesion.

The method of performing the short-circuit between the gall-bladder and stomach is somewhat similar to the operation of gastro-jejuno-stomy. The fundus of the gall-bladder is clamped so that about an inch and a half of it can be seen projecting above the blades. A fold of the anterior wall of the stomach, about two inches long and about two inches from the pylorus, is picked up, clamped, and brought alongside the gall-bladder (fig. 414). The clamps should lie in easy approximation to ensure freedom from tension on the suture line.

A sero-muscular continuous suture of fine silk or No. 00 20-day chromic catgut unites the adjacent portions of stomach and gall-bladder posteriorly for a distance of about an inch and a half. The stitches are placed very close to one another and should be carefully introduced so that the lumen of the stomach or gall-bladder is not entered.

An incision is now made through all the coats of the adjacent stomach and gall-bladder pouches for a distance of one inch, just anterior to the line of the first continuous suture.

The second through-and-through suture of No. 0 or No. 00 20-day chromic catgut is now introduced and embraces all the coats of the gall-bladder and the stomach around the whole circumference of the

opening. When it arrives at the point where it commenced, the ends of the sutures are tied and cut short.

The anterior portion of the anastomotic line is now invaginated by picking up the first suture and continuing it as a sero-muscular stitch. This may be introduced in the manner of a continuous Lembert or Cushing right-angled stitch. The ends of this suture are likewise tied



*Fig. 414.*—CHOLECYSTO GASTROSTOMY.

and cut short when it reaches the point where it started on the posterior aspect of the anastomosis.

A few interrupted sutures of fine catgut are placed here and there to strengthen the suture line and to relieve any tension. A wisp of omentum may be drawn over and fixed to the line of approximation for further protection.

It is important to close the abdominal wound very carefully and to introduce tension sutures to guard against the possibility of burst abdomen.

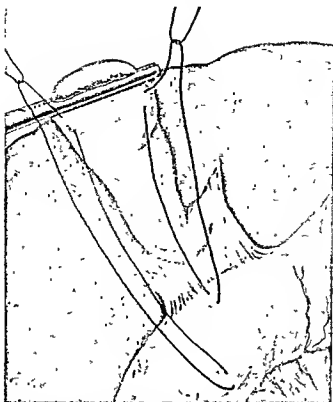


Fig. 415.—CHOLECYSTODUODENOSTOMY WITHOUT THE AID OF CLAMPS. THE DUODENUM HAS BEEN MOBILISED, LACRO KOCHER FORCEPS RAISE THE FUNDUS OF THE GALL-BLADDER. TWO TRACTOR SUTURES HAVE BEEN INTRODUCED AND ARE READY TO BE TIED. WHEN THESE SUTURES ARE TIED, THE DUODENUM IS DRAWN UPWARDS AND LIES AGAINST THE VENTRAL ASPECT OF THE GALL-BLADDER.

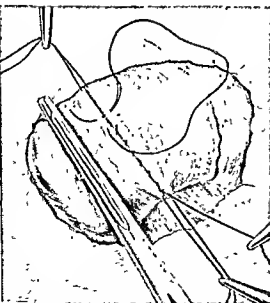


Fig. 416.—CHOLECYSTO-DUODENOSTOMY WITHOUT THE AID OF CLAMPS. THE POSTERIOR SERO-MUSCULAR SUTURE IS BEING INTRODUCED.

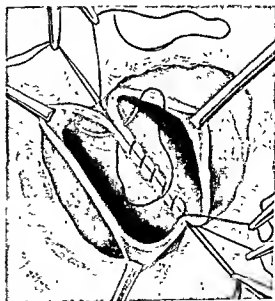


Fig. 417.—CHOLECYSTO-DUODENOSTOMY WITHOUT THE AID OF CLAMPS. THE CONTINUOUS POSTERIOR ALL-COATS HEMOSTATIC SUTURE IS BEING INTRODUCED AND IS ABOUT TO TURN THE CORNER.

(2) *Cholecysto-duodenostomy*. This operation is similar in many respects to the one just described. Here, however, the duodenum will often have to be mobilised before the intestinal clamp can be applied to it. The second part of the duodenum is selected for the anastomosis, and the incision into the gut is placed transversely. In a jaundiced patient the dangers of hæmorrhage from the raw surface are not inconsiderable, and all oozing points must therefore be carefully ligated before proceeding with the anastomosis. This operation is more easily performed without the aid of clamps. The essential steps in its performance are depicted in figures 415, 416, 417 and 418.

(3) *Cholecysto-jejunostomy*. In this operation a loop of jejunum some eighteen inches from the flexure is brought through an opening in the mesocolon and gastro-colic omentum and approximated to the gall-bladder (fig. 419).

Clamps are applied to the gall-bladder and to the apex of this loop, and the anastomosis is performed as in the operation of gastro-jejunostomy.

In certain cases, where the mesocolon is adherent posteriorly, or the right border of the gastro-colic omentum is matted with inflammation or growth, the jejunal loop should be brought over the transverse colon and anastomosed to the gall-bladder as in the operation of anterior gastro-jejunostomy. At the completion of this operation an entero-anastomosis between the afferent and efferent loops of jejunum should be performed at a point about two inches from the flexure.

#### OPERATIONS UPON THE BILE-DUCTS

##### (1) *Choledochotomy and Choledochostomy*

One of the following operations for *removal of calculi* from the common ducts may be required :

- (a) Moynihan's operation of rotation of the duct.
- (b) Supra-duodenal choledochotomy.
- (c) Retro-duodenal choledochotomy.
- (d) Trans-duodenal choledochotomy.
  - (i) McBurney's operation—duodeno-choledochotomy or ampullary choledochostomy.
  - (ii) Kocher's operation—trans-duodenal choledochostomy.

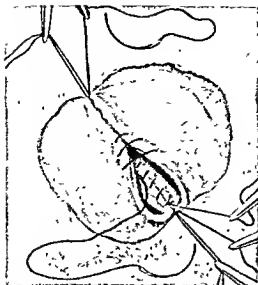


Fig. 418.—CHOLECYSTO-DUODENOSTOMY WITHOUT THE AID OF CLAMPS. THE ANASTOMOSIS IS NEATLY COMPLETE. THE FIGURE SHOWS THE METHOD OF INTRODUCING THE ANTERIOR THROUGH AND-THROUGH SUTURE.

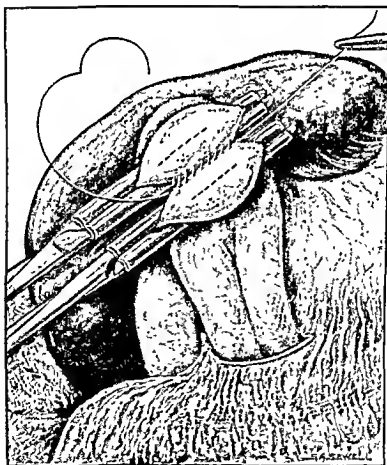


Fig. 419.—CHOLECYSTO-JEJUNOSTOMY.

*Rotation of the Duct*

This operation of Moynihan's is indicated in certain cases where there is obstruction of the common duct by an impacted calculus or calculi, where the minimum amount of operative interference is advisable owing to the jaundiced and poor condition of the patient, and where the gall-bladder is small and shrunken and numerous adhesions obscure the cystic and common ducts. In such cases the necessary freeing of adhesions and time-consuming dissection that is usually necessary to expose the common bile-duct may be too great a strain on the patient's resources.

In this operation the adhesions are ignored and yet the duct is made accessible and can be opened and drained with the minimum amount of interference.

"After isolating the operative field the surgeon's left hand is passed transversely inwards in front of the pylorus and above the stomach along the gastro-hepatic omentum. When the hand is well placed the fingers are flexed and the wrist and hand are bent over to the patient's left, with the result that the common duct is twisted up into the wound and readily incised." (Moynihan, *Abdominal Operations*, Vol. ii, p. 360, Saunders, 1926.)

When the surgeon's thumb is placed over the duodenum, as shown in figure 420, the stone or stones can be palpated in the duct between the fingers and the thumb. Two stay sutures are then introduced in the longitudinal axis of the duct, an incision is made between them and the stones are extracted from the ducts. The exploration and drainage of the duct does not differ in any essential detail from that already described, except that on cutting the loops of the stay sutures, four sutures are immediately available for infolding the duct around the trough end of a T-tube.

*Supra-duodenal Choledochotomy*

The majority of stones in the common bile-duct can be removed by the operation of supra-duodenal choledochotomy. Stones situated in the retro-duodenal portion of the duct can often be easily coaxed upwards; likewise, those lodged in the common hepatic duct can be worked downwards and removed by this operation.

In certain cases after cholecystectomy, exploration of the main ducts may be performed through the stump of the cystic duct if this is of sufficient width. If the exploration is negative, the stump is tied

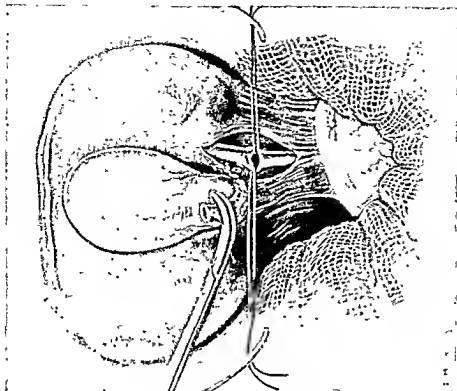


Fig. 121.—SUPRA-DUODENAL CHOLECYSTOTOMY. TWO STAY SUTURES ARE INTRODUCED TO ACT AS RETRACTORS WHILE THE DUCTS ARE EXPLORED.

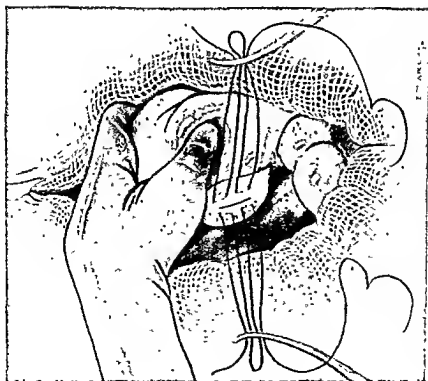


Fig. 430.—MOYNIHAN'S OPERATION OF ROTATION OF THE DUCT. NOTE PRECISELY THE LOCATION OF THE DUODUM AND THE METHOD IN WHICH THE STAY SUTURES HAVE BEEN INTRODUCED.

off; otherwise, a tube is inserted into the duct to provide the necessary drainage.

In the operation of supra-duodenal choledochotomy, the common duct is freely exposed by gauze dissection or by gently tearing through the overlying peritoneum with long dissecting forceps. If any doubt exists as to the identity of the duct, it should be punctured with a small hypodermic needle and a little of its contents aspirated and examined. If bile is withdrawn the duct should be picked up on each side with Allis forceps and an incision, one-third to half an inch long, should be made through its anterior wall just below the junction of the ducts. The edges of the wound in the duct are then held apart with two stay sutures (fig. 421).

In all operations upon the common duct the field of operation must be carefully packed off with abdominal swabs and mackintosh squares, the placing of a gauze pack under the duct so as to block the foramen of Winslow being an important point in the technique, as it must be assumed that the bile which escapes after the ducts are incised is highly infective. Escaping bile should be mopped up at once or removed by a suction tube. The forefinger and thumb of the left hand will feel for the stone in the common duct and will work it upwards to the incision in the duct through which it can be easily extracted (fig. 422).

If the duct is greatly dilated and is large enough to admit a finger, a digital exploration of the ducts should be made; otherwise the ducts should be probed and a search made for remaining calculi with the aid of special scoops, probes, and Desjardins forceps (fig. 423). If biliary mud or inflammatory debris is present the ducts should be irrigated with normal saline through a small rubber catheter, and the irrigation be continued until the fluid returns quite clear.

Graduated Liston sounds should then be passed very carefully and gently through the papilla into the duodenum, and the sphincter of Oddi stretched (fig. 424). If any doubt exists as to these sounds passing freely into the duodenum, a small gum elastic or rubber catheter should be introduced downwards as far as possible into the duct, and a quantity of saline injected through the catheter. If no fluid seeps back into the wound it may be rightly inferred that the end of the catheter lies free in the intestine.

When the surgeon is satisfied that no obstruction is present, the papilla should be freely dilated, either by graduated Liston sounds or by means of Desjardins forceps. When it is ascertained that the points of these forceps have passed through the ampulla into the duodenum,



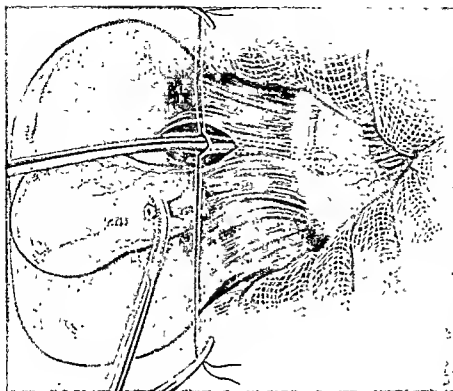


Fig. 423.—SUPRA-DUODENAL CHOLECYSTOTOMY. EXTRACTION OF A CALCULUS FROM THE COMMON DUCT BY MEANS OF DEBARDIS'S FORCEPS.

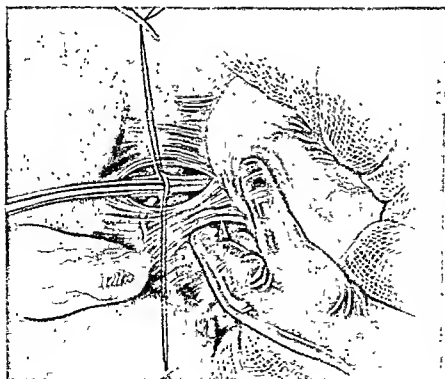
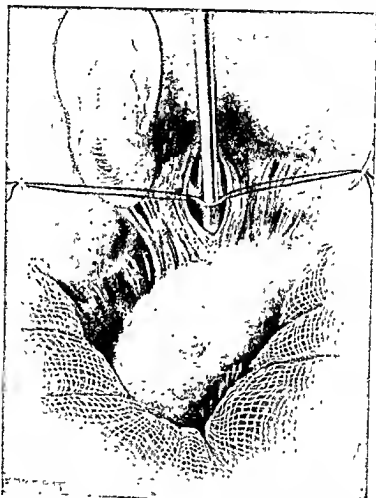


Fig. 422.—SUPRA-DUODENAL CHOLECYSTOTOMY. A SCOOP HAS BEEN PASSED DOWN THE COMMON DUCT, AND THE FINGER AND THUMB OF THE LEFT HAND ARE COINING A CALCULUS TOWARDS THE SCOOP.

the blades should be gently opened and closed so that the lower reaches of the common duct, and especially the papilla, may receive a thorough dilatation.

In doubtful cases, and particularly in those where it is thought that stones may be lodged in the dilated ampulla of Vater or in a pouch of



*Fig. 424.*—SUPRA DUODENAL CHOLEDOCHOTOMY PROBING THE AMPULLA OF VATER. A LISTON SOUND HAS BEEN PASSED THROUGH THE COMMON DUCT, THROUGH THE PAPILLA, AND IS BULGING THE ANTERIOR WALL OF THE DUODENUM OUTWARDS, PROVING THAT THE DUCT IS PATENT.

the common duct, an open-mouthed metal tube attached to an electrical suction apparatus may be passed downwards towards the ampulla, to remove any grit or calculi from this region by suction. This suction tube is also very useful for extracting stones from the hepatic ducts.

A No. 8 or No. 10 Jaques catheter, or a small rubber drainage-tube of similar size, is introduced into the duct and passed upwards towards the liver for half an inch or so and anchored in position by a fine catgut

stitch. The incision in the duct is then closed round the tube with a few interrupted plain catgut sutures.

If preferred, a T-tube, as depicted in fig. 425, may be used instead of the Jaques catheter or small rubber tube. The choledochostomy tube is fixed to the skin by a silkworm-gut stitch and carried through the dressings into a medicine bottle or a baby's feeding-bottle which is attached to the abdominal binder.

In certain instances, however, where on exploration the ducts appear to be healthy and normal in every respect, the small incision in the duct

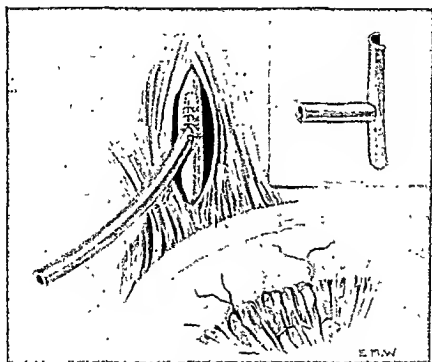


Fig. 425.—SUPRA-DUODENAL CHOLEDOCHOSTOMY. DRAINAGE BY MEANS OF A T-TUBE.

is carefully sutured with a series of interrupted Lemmert sutures or a continuous stitch of fine plain catgut, and a drainage-tube is placed near the line of suture in case any subsequent leakage occurs. This constitutes internal biliary drainage.

Exploration of the ducts is very rarely omitted in cases of cholelithiasis, as soft pigment stones, collections of biliary sand, inspissated pus, or inflammatory debris cannot always be palpated, even when present in large amounts.

In cases of severe and prolonged infections of the ducts drainage should be afforded by means of the author's T-tube which should be kept *in situ* for many weeks or even months (fig. 426). For a few hours

every day hile is allowed to drain out through the external limb into a hottle ; in the intervals this tube is clamped so that the hile passes into the duodenum.

### *Retro-duodenal Choledochotomy*

This is an operation which is very rarely performed. It is only indicated when a calculus impacted in the second part of the duct cannot be manipulated upwards into the first part of the duct or into

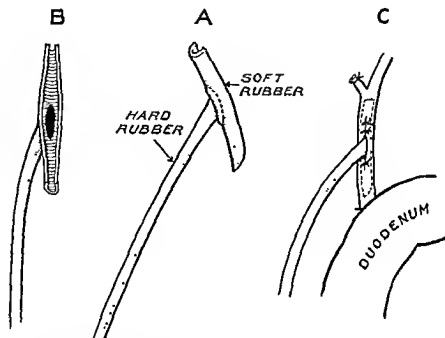


Fig. 426.—T-TUBE (AUTHOR'S PATTERN).

A—SIDE VIEW OF TUBE.

B—VIEW OF TROUGH PORTION (1½ IN. LONG) SHOWING THE LARGE OPENING INTO THE LONG LIMB WHICH IS 24 INCHES IN LENGTH.

C—TUBE IN POSITION IN THE COMMON DUCT.

the duodenum, or be extracted through a supra-duodenal incision by means of Desjardins forceps.

In order to approach this portion of the duct the duodenum will have to be adequately mobilised by Kocher's method.

A vertical incision, 1½–2 inches long, is made through the peritoneum on the outer side of the duodenum (fig. 427). The fingers are introduced through this opening and the lax areolar tissue and peritoneum on the outer side of the gut are stripped from the posterior abdominal wall inwards towards the duodenum, and this stripping is continued until the head of the pancreas is raised from its posterior attachment and is capable of being turned partially over towards the

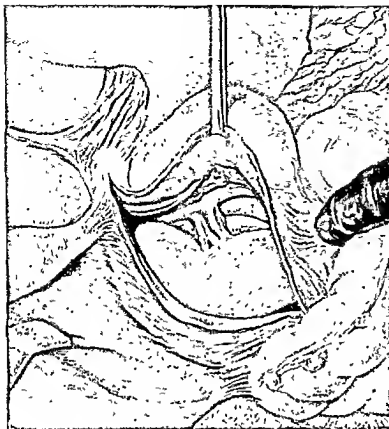


Fig. 423.—Kocher's Method of Mobilizing the Duodenum. The Mobilisation is Completed and the Retro Duodenal Portion of the Common Duct is Shown. (Adapted from "The Duodenum," by Dr. Edward L. Kellogg, New York, Paul B. Hoeber, Inc., 1933.)

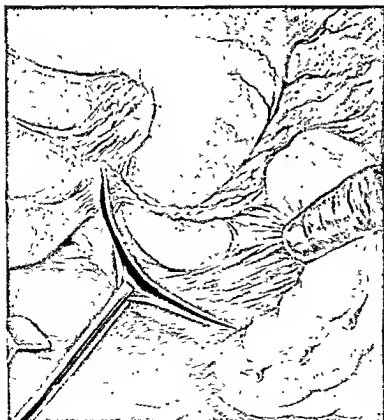


Fig. 427.—Kocher's Method of Mobilizing the Duodenum. The Peritoneal Incision is Shown. (Adapted from "The Duodenum," by Dr. Edward L. Kellogg, New York, Paul B. Hoeber, Inc., 1933.)

middle line (fig. 428). The impacted stone may now be felt as a small hard tumour, covered by or lying within a groove in the pancreatic tissue.

An incision is made over the tumour, and the impacted calculus is shelled out. The wound in the duct is then closed by a series of interrupted sutures.

Leakage is likely to occur as here the duct is not covered by peritoneum. At the completion of the operation in such cases, therefore, I would advise opening the supra-duodenal portion of the common bile-duct, exploring the ducts through this fresh incision, and providing external biliary drainage. Another drainage-tube should be passed down to the site of the incision in the retro-duodenal portion of the duct in case there is any contamination in this region through leakage of infected bile.

### *Trans-duodenal Choledochotomy*

(i) *McBurney's operation of duodeno-choledochotomy or ampullary choledochostomy* will be required when a stone is impacted at the termination of the common bile-duct. The duodenum is mobilised by Kocher's method and the operative field is carefully packed off. By invaginating the anterior wall of the second portion of the duodenum the stone will often be felt as a hard nodule in the posterior wall of the gut.

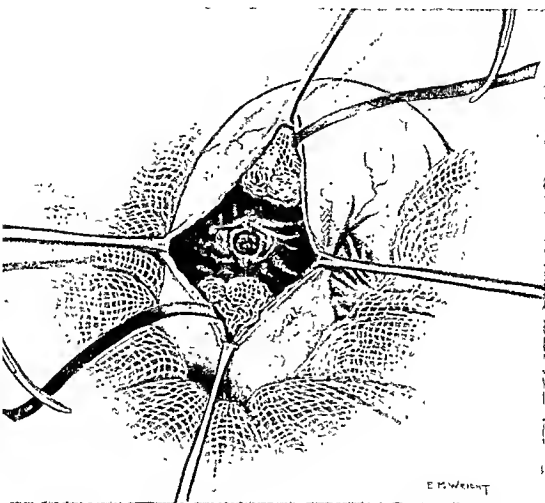
A longitudinal incision about one inch long is then made through the anterior wall of the duodenum opposite the impacted calculus, and its edges are drawn apart with Allis forceps. Any fluid which is present in the duodenum is either mopped up or removed by suction, and further contamination by duodenal juice is prevented by packing the lumen of the gut above and below the opening with swabs, by the application of intestinal clamps, or by the employment of gauze slings.

The stone may be visible and may be seen partially projecting through the papilla (fig. 429). On the other hand, it may be firmly impacted in the ampulla of Vater where it can be readily felt.

No useful purpose will be served by attempting to dilate the papilla in order to extract the stone. It is better to incise the edge of the papilla for a short distance, after which the stone can easily be removed. Again, this incision greatly enlarges the opening in the duct, and prevents the subsequent formation of a stricture at this site.

Scoops should now be passed upwards into the common duct to ascertain the presence or absence of any further calculi. If a supra-duodenal choledochotomy preceded the performance of this operation, a large Liston sound should be passed through this opening.

downwards into the duodenum, to ensure that no further calculus is present. By means of Desjardins forceps fine strips of gauze are drawn through from one opening to the other to gather up any biliary gravel in their meshes, and finally the ducts may be irrigated with warm normal saline solution to wash away any inflammatory



*Fig. 423.—McKENNEY'S OPERATION OF ANFOLLARY CHOLEDOCHOSTOMY. THE SELECTED SITE FOR INCISION THROUGH THE EDGE OF THE PAPILLA IS INDICATED BY A DOTTED LINE.*

debris. It is not necessary to suture the edges of the small wound in the papilla.

By traction on the Allis forceps the wound is rendered transverse, and is very carefully closed in this direction, first with a Connell stitch, and then by a sero-muscular continuous stitch which invaginates the suture line. A few interrupted sutures are inserted to relieve any tension, and the operation is completed by fixing an omental graft over the sutured gut.

(ii) *Kocher's operation of trans-duodenal choledochostomy* is indicated when a stone becomes impacted in the second or third part of the common duct. A stone impacted in the second part may often be forced upwards into the first part and be removed by supra-duodenal choledochotomy. If, however, it is firmly impacted in this position, the surgeon has a choice of removing the stone by retro-duodenal choledochotomy or by trans-duodenal choledochostomy; in the majority of cases the latter, i.e. Kocher's operation, is to be preferred.

Here, again, the duodenum is mobilised by Kocher's method and the field of operation is packed off. A longitudinal incision of 1-1½ inches is made through the anterior wall of the second portion of the duodenum, with its central point opposite the papilla. The wound is then retracted and contamination of the operative field is prevented in the manner described. The papilla and the posterior duodenal wall just above it are palpated, as any localised induration will indicate the site of the impacted stone.

Although the stone is often difficult to locate, its position is sometimes indicated by a small mound just above the ampulla. The folds of mucous membrane over this little projection are put on the stretch with the fingers and thumb and a small longitudinal incision is made with a scalpel through the posterior duodenal wall, through the sclerosed pancreatic tissue, and through the anterior wall of the duct, before the calculus can be seen.

Stitches are then taken on each side of the incision through all the tissues between the stone and the posterior duodenal wall. These stitches will therefore embrace a portion of the duct, the compressed pancreatic tissues which lie between the duct and the posterior duodenal wall, and the duodenal wall itself.

As the incision is lengthened to permit of easy extraction of the stone, additional sutures are introduced in the newly-cut tissues, the ends not being tied, but being held by hæmostats and used as retractors (fig. 430).

As soon as the stone is removed these sutures are carefully tied, and the new opening in the duct is inspected to make sure that it is ample and patent. The placing of a stitch at the upper and lower ends of this wound is never omitted. The upper reaches of the duct are then explored for any remaining calculi, and if the exploration is negative the incision in the anterior wall of the duodenum is closed in a transverse manner as in McBurney's operation.



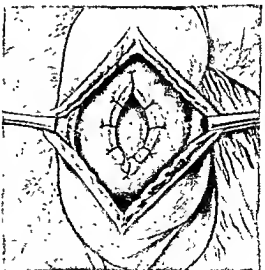
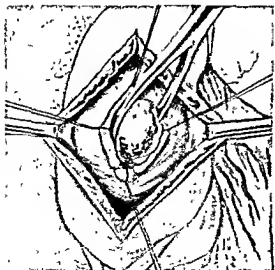
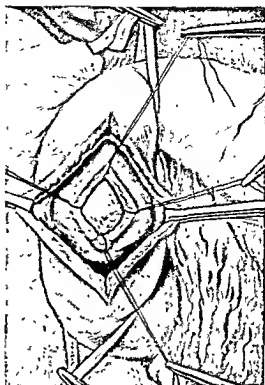


Fig. 431.—Kocher's Operation of Trans duodenal Cholecystostomy.

(Adapted from "The Duodenum," by Dr. Edward L. Kellogg, New York, Paul B. Hoeber, Inc., 1922.)

(2) *Choledcho-Choledochostomy*

Direct suture, or end-to-end anastomosis between the divided ends of the ducts, can, as a rule, only be performed immediately after the duct has been divided. When the duct is divided the lower end at once contracts and retracts. If a long-standing external biliary fistula is present, it is quite futile at operation to search for the retracted lower portion of the duct, as it becomes collapsed, cord-like, and welded to the back of the duodenum by adhesions.

Direct suture can therefore only be undertaken and is only to be recommended :

- (1) Immediately after accidental division of the supra-duodenal portion of the duct.
- (2) Following an economic segmental or annular resection of the duct for primary growth or simple stricture.
- (3) If operation is performed within a few hours of a violent injury which has resulted in rupture of an accessible portion of the duct.

There are many methods devised for anastomosing the ends of the duct, but only three will here be described :

(1) The ends of the duct are mobilised for one-third of an inch or so, but the free edge of the gastro-hepatic omentum and any adhesions which are present *behind* the ducts are not divided, as these structures act as a prop and help to relieve tension on the suture line when the anastomosis is completed.

A few cross-stitches, if accurately introduced in the longitudinal axis of the tissues immediately behind the ducts, afford additional support and draw the ends of the ducts more closely together. The finest catgut sutures and the smallest atraumatic or eyeless intestinal needles are used for suturing. Three or four interrupted sutures are required to re-establish the continuity of the duct posteriorly (fig. 431).

A small short rubber tube is now placed into the duct to facilitate the introduction of the anterior sutures (fig. 432) and is withdrawn by a hæmostat immediately before the sutures are tied. A drainage-tube is placed just below the anastomosis and kept *in situ* for four days in case there is any leakage of bile.

Two common complications of this operation may be mentioned :

- (a) *Immediate.* The suture line breaks down with the formation of a persistent external biliary fistula.
- (b) *Late.* A stricture forms at the site of the anastomosis.

In view of these unpleasant possibilities, direct suture should not be attempted unless it can be carried out with the greatest ease and precision. Where the operation has presented no undue difficulties the late results are satisfactory in a large proportion of cases.



Fig. 431.—DIRECT SUTURE OF A SEVERED COMMON DUCT. A FEW SUTURES HAVE FIRST BEEN INTRODUCED POSTERIORLY.

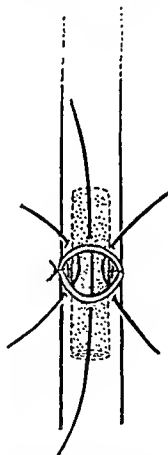


Fig. 432.—DIRECT SUTURE OF A SEVERED COMMON DUCT OVER A RUBBER TUBE. INTRODUCTION OF THE ANTERIOR SUTURES. THE TUBE IS WITHDRAWN JUST BEFORE THE ANTERIOR SUTURES ARE TIED.

(2) The ends of the duct are freed in the manner described ; if they do not come together easily the duodenum should be mobilised to facilitate the approximation. A few interrupted sutures bridge the posterior gap in the divided duct, and the tissues behind this and in the

right border of the gastro-hepatic omentum are drawn together longitudinally with a few cross-stitches to relieve tension. The small trough-like end or cross-piece of a rubber T-tube is inserted into the partially constructed duct so that one limb lies above and the other below the line of suture. The long limb of the T-tube is drawn through

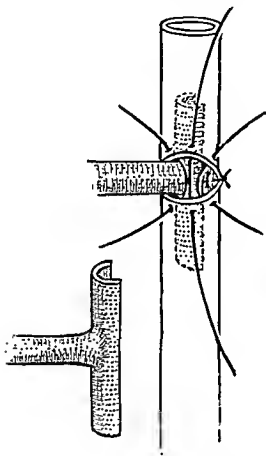


Fig. 433.—DIRECT SUTURE OF SEVERED COMMON DUCT. T-TUBE IN POSITION. INSET SHOWS THE SOFT FLEXIBLE, TROUGH LIKE CROSS PIECE OF THE T-TUBE.

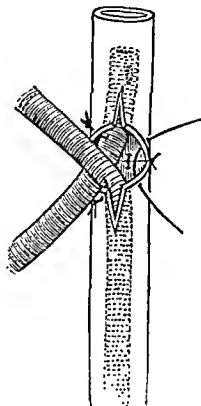


Fig. 434.—RECONSTRUCTION OF A SEVERED COMMON DUCT. TWO TUBES ARE INTRODUCED INTO THE DUCT, ONE UPWARDS TOWARDS THE LIVER, AND THE OTHER DOWNWARDS INTO THE LUMEN OF THE DUODENUM.

a small stab wound in the abdominal wall to the surface where it is securely anchored (fig. 433).

All that now remains is to close the ends of the duct snugly and in a watertight manner round the tube. Another drainage-tube should be inserted below the sutured duct and kept in place for three or four days. For the first four days after operation bile drains through the long limb of the T-tube into a small receptacle, such as a baby's feeding-bottle, which is fixed to the bed or to the patient's abdominal

## ABDOMEN

hinder, but after this time the tube is intermittently clamp the bile to pass into the intestines.

After twenty-one days the T-tube is cautiously withdrawn. The cross-piece is very short and flexible it is unlikely to tear the line of suture or otherwise lacerate the duct.

This is a very satisfactory method, and the employment of it in addition to being useful in cases of direct suture of a severed duct, can also be recommended where *prolonged drainage* is called for in cases of severe cholangitis.

(3) Here the gap in the posterior wall of the duct is unbridgeable. A few interrupted sutures of the finest catgut. Each end of the duct anteriorly is now incised longitudinally for about a quarter of an inch. Before inserting two small rubber catheters, one of which is directed upwards towards the liver and the other downwards through the common bile-duct into the duodenum (fig. 434). A few sutures are introduced through the lateral and anterior edges of the wound in the duct to reduce the aperture through which the tubes have been inserted. These tubes are stitched to the edges of the duct to anchor them securely in position, and the free ends are drawn through each of the abdominal wound and fixed to the skin.

The duodenal tube is used for feeding purposes, and a solution of six to eight pints of 5-10 per cent glucose may be introduced through this tube during the first few post-operative days. The biliary tube drains bile into a small receptacle which is fastened to the patient's abdominal binder.

*This bile is returned through the duodenal tube from time to time. The ends of the two tubes may be joined together by a glass connector so that the bile flows continuously into the duodenum.*

Both these tubes are removed at the end of three weeks. For a few days following their extraction an external biliary fistula may result, but, provided that the passages are clear, this discharge soon ceases.

### (3) *Lateral Cholelocho-Duodenostomy*

In this operation a side-to-side anastomosis is performed between the dilated common bile-duct and the first part of the duodenum. The main indication for this operation is where the duct is obstructed down in the retro-duodenal portion and is inextricably wedged by the pancreas.

when the obstruction is situated in the retro-duodenal portion of the duct, and when there is no biliary fistula.

The operation is performed as follows : The duodenum is mobilised by Kocher's method, and the anastomosis is made at a point where the common duct and duodenum can most easily be approximated. Where possible, the parts to be anastomosed should be held by traction sutures. The incision should be made transverse in the duct and vertical in the duodenum. The parts are approximated and a continuous sero-muscular suture of No. 00 chromic catgut or fine linen thread is applied. A posterior through-and-through all-coats hæmostatic suture of No. 00 20-day chromic catgut draws together the posterior margins of the wounds in the duodenum and duct, and this is carried anteriorly to the starting-point. The sero-muscular stitch is then taken up again and completed anteriorly. A few Halsted stitches are inserted anteriorly to relieve tension, and the suture line is protected with an omental graft.

#### (4) *Reconstruction Operations—End-to-Side Choledcho- or Hepatico-Duodenostomy*

These operations of re-formation of the common duct are amongst the most difficult tasks in surgery.

"To obtain the best results all the highest qualities of the surgeon may be called into play: courage, resource, patience, accuracy and rapidity of judgment, the finest craftsmanship, and a tranquillity of mind and action that nothing can disturb." (Moynihan.)

(a) *Anastomosis between the Coned-out Fistula and the Duodenum.* In cases where an external biliary fistula has followed upon a very high division or a complete ablation of the common hepatic duct, making anastomosis between the hepatic duct and the duodenum impossible, the fistula should be freed from the skin and from its surroundings as far as the hilum of the liver, and implanted directly into the duodenum or jejunum.

If the fistula is lined with mucous membrane and an anastomosis with the duodenum is performed, the final result should be satisfactory; but if, on the other hand, the tract is not lined with mucosa, it will probably contract, leading to the formation of a stricture.

Where the common ducts have been accidentally removed at operation and the patient has survived, either an external biliary fistula or profound jaundice will develop. In the latter case an external

hiliary fistula should be established, which can later be coned out and transplanted into the duodenum or stomach with satisfactory results.

Waltman Walters reported five such cases, in three of which excellent results had followed. When transplantation of an external hiliary fistula is performed, it must adequately drain the intra-hepatic ducts, and these ducts must be free of stones; otherwise the obstruction will recur.

(b) *Mayo's Operation of Direct Implantation.* W. J. Mayo (*Ann. Surg.*, xlii, 90, 1905) was the first to perform this operation of direct implantation of the hepatic duct into the duodenum. The result in his first case was eminently successful, as the patient survived and enjoyed good health for over twenty years. But in some of his subsequent cases stricture at the site of the anastomosis frequently followed. This complication led Balfour (*Ann. Surg.*, lxxiii, 346, 1921) to devise the following method, the results of which have proved to be very satisfactory. Balfour describes this operation as follows: "The stump of the hepatic duct is freed as much as possible from its adhesions, but it is rarely possible to secure a projection beyond the liver fissure for more than 0.3 cm. to 0.5 cm. The duodenum, as has been noted, is usually drawn into the same mass of adhesions and it is always wise to avoid separating it posteriorly. If it is separated, a few catgut stitches will draw it up again to the stump of the duct. A slightly curved flap is then dissected out of the entire thickness of the duodenal wall over an area which will leave an opening into the duodenum about 2 cm. in diameter. The duodenal flap is then approximated to the posterior and lateral aspects of the stump of the hepatic duct in such a manner as to permit of a muco-mucous union of the posterior half of the circumference of the duct, with the edge of the flap sutured as shown in the figures. The opening in the duodenum is, of course, much larger than the hepatic duct. The remaining free margins of the opening in the duodenum are sutured to the capsule of the liver just above the hepatic duct end by a continuous catgut suture so that the under-surface of the right lobe of the liver, or more correctly Glisson's capsule and the scar tissue adherent to it, effectually closes the opening in the duodenum not occupied by the end of the hepatic duct. A considerably wider area of the duodenum is then drawn up towards the liver and fixed with catgut sutures. The omentum is caught by the tip and divided if necessary so that it may be used effectually to surround the anastomosed area. Drainage is seldom necessary; if needed, two small strips of rubber tubing are introduced, one above and one below the anastomosis. In

some cases a moderate amount of bile may escape for a few days. This has always ceased within a week, however, and healing finally has been complete in each case. In no case has there been any evidence of subsequent obstruction to the duodenum, and in no case a failure to deliver bile into the duodenum.

"It will be noted that this technic provides a large opening in the duodenum and a muco-mucous union for two-thirds or, at least, one-half the circumference of the hepatic duct stump. These provisions, together with the method of suturing the opening in the duodenum to the liver, allow for contraction and obviate the danger of secondary stricture, so that obstruction does not take place" (figs. 435 and 436).

Grey Turner speaks very favourably of this operation and has performed it successfully in a number of cases.

(c) *Coffey's Modification of Sullivan's Operation of End-to-Side Choledcho-Duodenostomy.* Kellogg (*The Duodenum*, p. 667, Paul B. Hoeber, Inc., New York, 1933) describes the essential steps of this operation as follows: "The distal portion of the common duct is ligated and peritonealized. The proximal portion may be united to the duodenum by the method suggested by Coffey.

"A vertical incision is made in the duodenum down to, but not through the mucosa. The duct is divided obliquely so that the apex will be on the anterior surface.

"The apex is transfixed with a suture of chromic gut, both ends threaded.

"The lower angle of the duodenal mucosa, exposed by the incision, is incised and the needles are introduced into the lumen of the duodenum and made to transfix the wall 1 inch (1.25 cm.) below. The end of the duct is drawn down into the lumen of the gut and held in contact with the mucosa by tying the suture.

"The peritoneal and muscular coats exposed by the incision are sutured over the duct and the stitch is carried down to cover the transfixion suture.

"This procedure may be facilitated by suturing over a rubber catheter which extends from the lower third of the duct into the duodenum. The catheter ultimately is discharged through the bowels" (fig. 437).

(d) *Walton's Operation of Indirect Implantation.* Walton (*Surgical Dyspepsias*, 2nd ed., p. 573, Arnold, 1930) who has performed a large



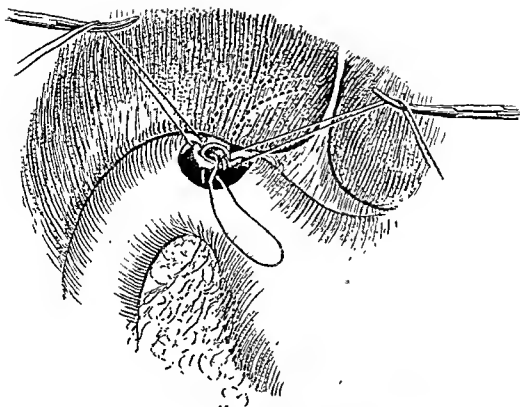


Fig. 433.—BALFOUR'S MODIFICATION OF MAYO'S OPERATION OF DIRECT IMPLANTATION OF THE HEPATIC DUCT INTO THE DUODENUM. THE THROUGH AND THROUGH SUTURE IS BEING INSERTED. (After Mayo)

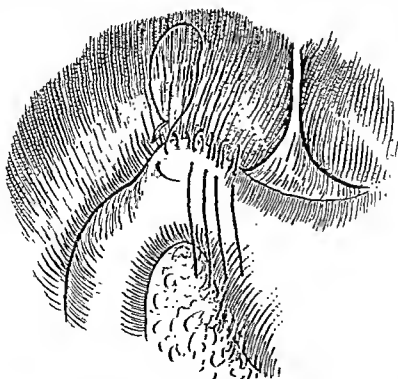


Fig. 436.—BALFOUR'S MODIFICATION OF MAYO'S OPERATION NEARLY COMPLETED. THE DUODENUM IS BEING STITCHED TO THE LIVER OR RATHER GIBSON'S CAPSULE. (After Mayo.)

number of these operations with great skill and success, describes his method as follows :

"Most of the modern operations are based upon the method advocated by Sullivan, who inserted a tube into the proximal end of the duct and then implanted the distal end of the tube into the

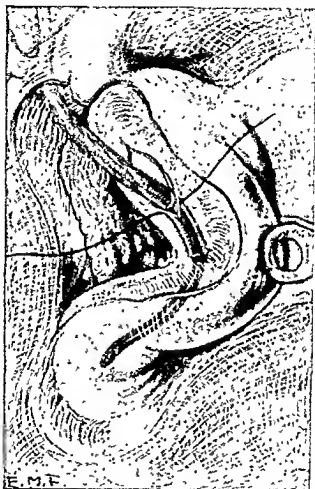


Fig. 437.—END-TO-SIDE CHOLEDOCHODUODENOSTOMY. (LUFFY'S METHOD) THE CATHETER EXTENDS FROM THE DUCT INTO THE DUODENUM, AND THE INCISION IN THE DUODENAL WALL IS SUTURED OVER THE DUCT.

(Reproduced from "The Duodenum," by Dr. Edward L. Kellogg, New York, Paul B. Hoeber, Inc., 1933)

duodenum. The free portion of the tube was wrapped round with omentum in the hope that a fistulous tract would thereby be formed and would persist after the tube was passed. A valvular opening into the duodenum was ensured by suture of the tube into the duodenum after the manner of the Witzel method of gastrostomy.

Mayo lays stress upon the fact that a stricture is likely ultimately

to occur, but if the operation can be combined with direct union of some portion of the duct so that there is a partial lining of mucosa, this tissue may grow around and ultimately give satisfactory results.

On theoretical grounds the operation would certainly appear to be faulty. The tube, being held in non-contractile tissue, is unlikely to be passed. The wall of the duct is formed of omentum alone and thus at best is a fistulous tract; stenosis is therefore very likely to occur and lead to a recurrence of the condition.



Fig. 438.—WALTON'S OPERATION. A FLAP IS CUT FROM THE ANTERIOR SURFACE OF THE DUODENUM AND IS TURNED DOWNWARDS.

(Adapted from "Surg., Gynec., and Obstet.," Oct., 1929.)

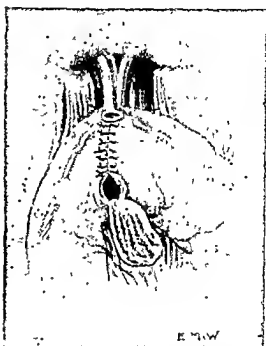


Fig. 439.—WALTON'S OPERATION. THE UPPER PART OF THE OPENING IN THE DUODENUM IS SUTURED.

(Adapted from "Surg., Gynec., and Obstet.," Oct., 1929.)

The difficulties and drawbacks which are associated with all of the above methods led me to devise the following operation, which I first published in 1915, and which I have used in eighteen cases with satisfaction.

Exposure is gained by an upper right paramedian incision. The common bile-duct is now laid bare. If there has been a prolonged biliary fistula the lower end will probably not be discovered. If there is a stricture or carcinoma this is, if possible, removed, so that there now remains a condition in which the upper end of the duct is patent but is separated by a wide gap from the duodenum so that a direct implantation is impossible. The upper border of the duodenum is drawn upwards

and sutured so that the gap is as far as possible reduced. The largest-sized tube that will enter the cut end of the duct is inserted and sutured in position with plain catgut. A flap is then cut from the anterior surface of the duodenum and is turned downwards (fig. 438). The upper part of the resulting opening is sutured until it is only sufficiently large to admit the tube (fig. 439). The tube is then inserted and the flap turned upwards over it (fig. 440). In the upper portion the edges of the flap are sutured around the tube and to the edges of the cut duct (fig. 441). Below they are sutured to the wall of the duodenum, which forms the structure adjacent to the posterior surface of the tube. For safety a small drainage-tube is inserted down to the junction.

The operation in practice is very simple to perform. A new duct can readily be formed of practically any length, it is lined with mucous membrane which is impervious to the action of the bile, and being lined by such a membrane will show no contraction; the tube passes obliquely over the duodenal surface and hence there will be a well-defined valvular action. Owing to the presence of the mucous membrane lining it is not necessary for the tube to remain long in position. It can be sutured in place with plain catgut, which is dissolved in a few days, and thus there is little or no danger of the tube being retained.

I have found the method so easy to perform and so generally satisfactory that I have made a slight modification of it for use in those cases in which there is an obstruction low down in the duct. In the cases already mentioned, in which there is an obstruction due to carcinoma or chronic pancreatitis, and in which the duct has been opened for exploratory purposes, so that cholecyst-enterostomy becomes a risky procedure, it is a perfectly simple matter to insert the tube into the lateral opening of the common duct instead of into the cut extremity, and then to reconstruct the new duct from the duodenal flap around the tube so that there is, in fact, a new duct entering the lower part of the original one at a slight angle.

My own series of twenty-six cases include eight of terminal and ten of lateral reconstruction. Of the terminal, two died as the result of operation, one had a return of severe jaundice and died three years later, two have had occasional slight attacks of jaundice but are otherwise well, and three have remained in perfect health for one, ten, and fifteen years respectively. Of the ten cases of lateral reconstruction five died as the result of the operation, a very high immediate mortality, but three of these had advanced carcinoma of the ducts and pancreas, and two showed the ducts full of 'white bile,' the presence of which is an indication of severe hepatic insufficiency."

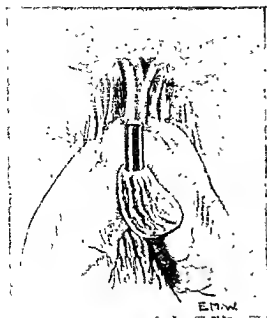


Fig. 440.—WALTON'S OPERATION. THE TUBE HAS BEEN INSERTED INTO THE DUCT AND INTO THE DUODENAL OPENING.

(Adapted from "Surg., Gynec., and Obstet.," Oct., 1929.)

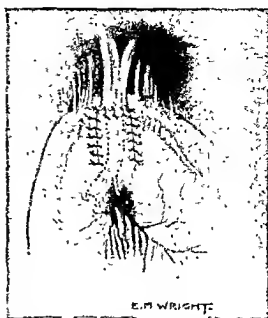


Fig. 441.—THE DUODENAL FLAP HAS BEEN SUTURED AROUND THE TUBE.

(Adapted from "Surg., Gynec., and Obstet.," Oct., 1929.)

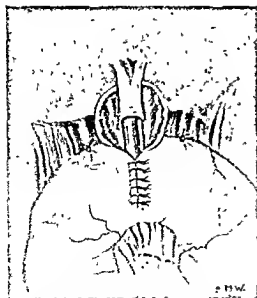


Fig. 442.—RECONSTRUCTION OF THE COMMON DUCT.  
(After Moynihan, modified.)



Fig. 443.—RECONSTRUCTION OF THE COMMON DUCT.  
(After Moynihan, modified.)

(e) *Another Method of Reconstructing the Common Duct* is depicted in figures 442 and 443. In this operation, as the opening into the duodenum is direct and not valvular, an ascending infection of the ducts is more liable to occur.

When the duodenum is firmly bound to the underlying structures, or is friable from inflammation, it may be impossible to mobilise it or to utilise it for purposes of anastomosis. In such cases the duct should be implanted into the jejunum. A loop of jejunum some fourteen inches from the flexure is either drawn through an opening in the mesocolon and gastro-colic omentum, or is taken in front of the transverse colon and anchored in the region of the hilum of the liver in such a manner that no kinking of the jejunum itself or of the colon results. A small rubber tube is then stitched to the stump of the hepatic duct, and led through a small opening in the jejunum which lies in close proximity to it. The opening in the jejunum is then stitched around the stump of the hepatic duct. I have performed this operation of hepatico-jejunostomy in one case with complete success.

#### (D) POST-OPERATIVE TREATMENT

The following measures are advised :

(1) *CO<sub>2</sub> Inhalations.* Owing to the frequency of post-operative chest complications following operations upon the gall-bladder and bile-ducts, 5-10 per cent CO<sub>2</sub> in oxygen is given during the final stages of the operation, and this is repeated for ten minutes in each hour for several hours after the patient has returned to the ward. Inhalations of CO<sub>2</sub> are beneficial in three ways :

- (a) The lungs are more fully inflated by the deeper breathing.
- (b) Better oxygenation of the blood is provided.
- (c) The likelihood of atelectasis is greatly diminished.

(2) *Stomach Tube.* Shortly after the operation a small stomach tube should be passed and the stomach be freely irrigated with normal saline. Gastric lavage is likewise indicated where vomiting is persistent or distension marked.

(3) *Continuous Intravenous Drip Method.* Ringer's solution and glucose 5-10 per cent are administered by this method during the first 48 hours after operation.

(4) *Heat.* Hot fomentations are applied to the lower half of the right side of the chest three or four times a day, or the heat may be applied by diathermy apparatus through the lower thoracic region. As stated above, this will sometimes stimulate a flagging liver and increase its activity.

(5) *Tubes.* Cholecystostomy and choledochostomy tubes are left *in situ* until they work loose, i.e. until about the tenth day. When they are extracted the flow of bile through the external wound soon ceases. Other drainage-tubes which are used to drain Morrison's pouch or the liver bed are usually removed on the fourth day.

(6) *Medicines.* The morning dose of Epsom salts and the hexamine mixture should be continued for two or three weeks after operation. One to two drachms of dilute hydrochloric acid, in 5-10 oz. of water to which is added orange juice and sugar, is given as a beverage three times a day. Belladonna is an indispensable drug after operations upon the gall-bladder and bile passages. In the immediate post-operative period it seems to relieve the pylorospasm which is presumably caused by the irritation of the tubes, whilst during convalescence, by relaxing the sphincter of Oddi, a free flow of bile is encouraged and colic is assuaged.

No restriction of cholesterol is required after operation, and the only instances where special diet is necessary is in cases of obesity and chronic gastritis.

## (E) RESULTS OF OPERATIVE TREATMENT

### (1) *For Chronic Cholecystitis.*

The mortality of cholecystectomy in cases of chronic cholecystitis (with or without gall-stones) varies from 0.5-3 per cent. Walton (*Surgical Dyspepsias*, p. 508, 1930) operated upon 242 cases with two deaths—a death-rate of 0.8 per cent; and Wilkie (*B.M.J.*, p. 767, Oct. 28, 1933; and *Lancet*, p. 751, Ap. 7, 1934), in 341 cases, had only five deaths—1.4 per cent. Judd and Priestley (*Coll. P. Mayo Clin.*, p. 92, 1932), state that during 1931 cholecystectomy was performed at the Mayo Clinic on 579 cases for chronic cholecystitis with a mortality of 1.7 per cent.

The late results of cholecystectomy for chronic cholecystitis show at least 75 per cent of cases cured and another 20 per cent improved.

The unsatisfactory results are due, in many instances, to imperfect primary operations in which stones remain undetected in the common duct; to faults in operative technique such as not peritonising the raw surfaces of the ducts and of the gall-bladder fossa; to the advent of chronic sclerosing pancreatitis; and to the presence of other chronic visceral lesions which were overlooked at the original operation. Some 20 per cent of these cases have post-operative flatulence, distaste for fats, chronic dyspepsia, and an occasional attack of biliary colic. Colic arising after gall-bladder operations may be due to stones in the common bile-duct which have been overlooked or to spasm of the sphincter of Oddi. Cases which develop colic after operation should be treated by dieting and ample doses of belladonna. If, however, jaundice supervenes, further operative interference will be necessary as this symptom suggests the presence of stones ("recurrent" calculi), or the development of a stricture of the common duct. As a general rule it may be stated that the grosser the gall-bladder pathology the better the results of the operation. The late results are, on the whole, very encouraging. There is an increase in physical energy, freedom from "rheumatism," an improved appetite although the distaste for fats continues, and any cardiac disability which was present before operation rapidly decreases and may even disappear entirely.

Cholecystostomy is rarely performed for chronic cholecystitis as there is proof that the results of this operation are greatly inferior to those of cholecystectomy. Following cholecystostomy there will be unsatisfactory results in from 40-50 per cent of cases, whilst some 20 per cent of the failures will require another operation—usually cholecystectomy and choledochostomy. Judd and Priestley have shown that there is a greater possibility of late recurrence of stones in the common bile-duct if cholecystostomy rather than cholecystectomy is performed at the original operation. Recurrence of stones after cholecystectomy and choledochostomy is extremely rare, whilst after cholecystostomy and choledochostomy the recurrence-rate at the Mayo Clinic was 7 per cent.

The death-rate of cholecystostomy for chronic cholecystitis should not exceed 0.5-1 per cent.

Cholecystostomy is not advised for uncomplicated cases as:

- (a) Gall-stones recur in 10 per cent of cases.
- (b) Biliary dyspepsia usually persists in spite of the drainage.



- (c) Carcinoma of the gall-bladder may develop at some future date.
- (d) A diseased organ is left behind to exert toxic influences upon the heart, kidneys, joints, fascial planes, etc., as emphasised by Wilkie (*Med. Soc. Trans.*, p. 105, 1930).

(2) *For Acute Cholecystitis.*

Heuer (*Ann. Surg.*, p. 773, Oct., 1933), in a summary of 1066 cases of acute cholecystitis, found a general mortality of 8 per cent. The individual death-rate varied from 4.7-22.5 per cent in the hands of different surgeons. Judd and Phillips (*Ann. Surg.*, p. 773, Oct., 1933), with the success characteristic of the work performed at the Mayo Clinic, showed that of 508 cases operated upon for acute cholecystitis there were 24 deaths, a mortality of only 4.7 per cent. Heuer, in another summary of 502 cases of gangrene with perforation of the gall-bladder subjected to operation, showed a general mortality of 46 per cent, and an individual mortality varying from 15-65 per cent. This author states that some 20 per cent of cases of acute cholecystitis will have complications such as gangrene, perforation, or peritonitis, if a policy of inactivity towards the disease is adopted, and considers that the removal of the acute non-gangrenous, non-perforated gall-bladder is usually not difficult, and is attended by a mortality which, in the hands of various surgeons, has ranged from 2-6 per cent.

It is difficult to arrive at an average mortality for cholecystostomy for this disease. If it is performed as a routine procedure it is low; if, on the other hand, it is reserved only for desperate cases the operative mortality is high, i.e. about 20 per cent.

(3) *For Stones in the Common Duct.*

- (a) Jaundice absent: Cholecystectomy with choledochostomy—mortality about 2-3 per cent.
- (b) Jaundice slight: Cholecystectomy with choledochostomy—mortality about 5 per cent.
- (c) Jaundice severe, but dark bile aspirated from the common duct: Cholecystostomy with or without choledochostomy, or choledochostomy alone—mortality about 15 per cent.
- (d) Jaundice severe with back pressure on the liver with white bile: mortality about 30 per cent.

*Causes of Death after Operations upon the Biliary System :*

The death-rate of operations upon the biliary tract depends largely upon the presence of complicating factors and errors in operative technique. Amongst these would be :

- (1) Intense jaundice.
- (2) Sub-acute pancreatitis.
- (3) Gangrene and perforation of the gall-bladder.
- (4) Cholangitis.
- (5) Lack of care in the selection and preparation of the patient for operation.
- (6) Rough handling of and injury to the liver, hepatic ducts, and blood-vessels, and exposure and chilling of the liver.

Nearly 40 per cent of deaths following operations upon the biliary tract are caused by peritonitis, hæmorrhage, and shock, 20-25 per cent by pulmonary complications, and about 12 per cent by cardio-renal disease. So-called "liver death" is responsible for about 4 per cent of cases, whilst gangrene and perforation would account for about 10 per cent.

## CHAPTER II

### GALL-STONES AND CHOLECYSTITIS

by

A. M. A. MOORE

*Types.* Gall-stones are of several varieties :

(1) The most common is a soft, fragile, *faceted calculus* composed principally of cholesterol with a small amount of calcium and pigment. It forms exclusively in the gall-bladder. The stones are deposited in large numbers and are moulded into their characteristic shape while still soft. The colour of these stones varies from almost white to dark brown. There may be hundreds of stones of this variety, and when they are sectioned they present a characteristic laminated structure (figs. 444, 446 and 447).

Each stone has a nucleus of organic material consisting of bacteria or desquamated epithelium. These stones are probably the result of infection of the gall-bladder, and the infecting organism may sometimes be recovered from the crushed stones so that evidence of typhoid, for instance, may be obtained many years after the disease was contracted. If faceted gall-stones are found together with an oval cholesterol stone it may be concluded that infection of the gall-bladder followed the formation of the single stone.

(2) The *single stone* composed purely of cholesterol is of fairly common occurrence. It is usually rounded or oval and may be practically colourless. It consists of almost pure cholesterol and is believed to be formed by precipitation of cholesterol from the contents of the gall-bladder, independently of any infection. The stone may become very large (fig. 448). Frequently it gives rise to no symptoms, but in some cases it may become impacted in the ampulla of the gall-bladder, which then becomes greatly distended with clear mucous secretion, constituting a mucocele. If such a gall-bladder becomes infected a condition known as empyema develops.

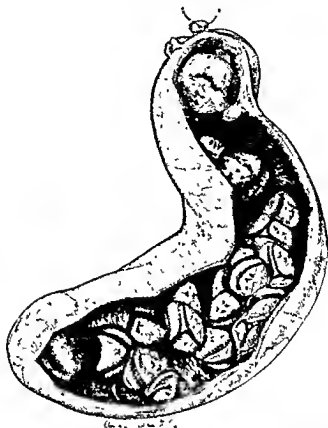


Fig. 444.—GALL-STONES. SPECIMEN OF A VERY MUCH ENLARGED GALL-BLADDER CONTAINING MULTIPLE FACETED STONES, ONE STONE BEING IMPACTED IN THE NECK OF THE GALL-BLADDER.  
(Museum, London Hospital.)



Fig. 445.—NORMAL CHOLECYSTOGRAM. (H. Cecil Bull.)

(3) A less common type of stone is the so-called *pigment stone*, composed of almost pure calcium bilirubinate (fig. 449). It is black and has an irregular surface. There may be one or multiple stones of this variety, or sometimes the gall-bladder may be filled with a fine sand or mud-like material. Stones of this type are found in those diseases in which there is constant destruction of red blood cells, as, for example, familial acholuric jaundice.

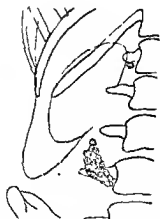


Fig. 446.—Multiple (alphen containing gall-stones in a gall-bladder which is low in position, its fundus pointing towards the mid line. There is also a single gall stone in the cystic duct. Note the low position of the right lobe of the liver and its relation to the kidney shadow. (H. Cecil Bull.)

(4) A very rare type of stone is one composed of *calcium carbonate*. Any of the other stones already mentioned may themselves contain some calcium carbonate, but stones composed solely of this substance are extremely rare. It must be remembered that the majority of gall-stones are formed in the gall-bladder. Sometimes, however, stones may be formed in the different biliary ducts themselves.



Fig. 447.—MULTIPLE GALL-STONES SHOWING THE RING SHADOWS OF CALCIUM AND THE GALL-BLADDER OUTLINED BY DYE. (H. Cecil Bull.)



Fig. 448.—A LARGE SINGLE CHOLESTEROL GALL-STONE.  
(Museum, London Hospital)



Fig. 449.—"SIEVE RING" GALL-STONE. (H. Cecil Bull.)

*The Causation of Gall-stones and Cholecystitis.* A considerable amount of experimental work has been done on this important subject, and there is no doubt that gall-stones frequently follow some degree of inflammation of the gall-bladder. When gall-stones cause symptoms, organisms, especially those of the colon group, are found not only in the bile but in the calculi themselves. Their presence leads to a mild catarrh of the mucous membranes and so to an increased production of cholesterol.

Another method by which gall-stones can be formed has been described by Aschoff. Mere stagnation of bile in the gall-bladder without any symptoms of infection may be followed by a deposition of cholesterol stones in the gall-bladder. When infection occurs as well there is the increased production of cholesterol and also of calcium salts which are deposited in connection with the cholesterol. The addition of bile-pigment produces the characteristic pale and dark lamination so often seen when a gall-stone is divided.

Infection of the wall of the gall-bladder by bacteria most commonly results in fibrous thickening. Sometimes it is associated with a condition called "strawberry gall-bladder," in which many pale yellowish-white specks are scattered over a reddish-coloured mucous membrane. This is the result of the deposition of small amounts of cholesterol in the mucous membrane.

To summarise the position, therefore, cholesterol production as determined by stagnation in the presence of other constituents of gall-stones implies infection as an additional factor.

Gall-stones certainly occur very much more frequently in women than in men, and particularly in stout women of about the age of 40 years. The reason why gall-stones occur more frequently in women is believed to be associated with those infections which occur at the time of pregnancy. The bile of pregnant women contains four times as much cholesterol as that of ordinary women, and, because of this, precipitation can easily occur. Probably the colon bacillus circulating in the blood passes to the gall-bladder and produces some degree of cholecystitis, which may lead to gall-stone formation.

*Typhoid Fever as Related to Gall-stones.* Infections of the gall-bladder frequently occur in typhoid and paratyphoid fever, and it is believed that gall-stones following typhoid may be responsible for the fact that some unhappy people are typhoid carriers. It has been demonstrated clearly that a culture taken from gall-stones sometimes reveals the presence of the typhoid bacillus very many years after an attack of typhoid fever had subsided.

*Pathological Complications of Gall-stones.* In considering this very important matter, it must be borne in mind that post-mortem examinations on people who have died from other conditions frequently reveal the presence of gall-stones in cases where no history of these stones has ever been obtained. It is possible that the patient may have suffered from very mild symptoms which passed unrecognised.

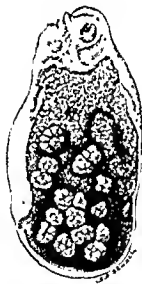


Fig. 450.—STRAWBERRY GALL-BLADDER.

(Museum, Royal College of Surgeons.)

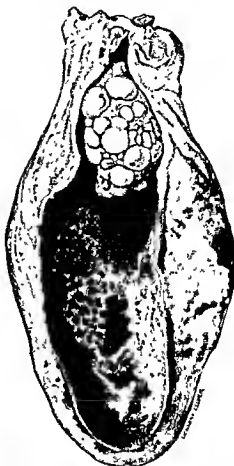


Fig. 451.—ACUTE OBSTRUCTIVE CHOLECYSTITIS DUE TO A "CONGLOMERATE" STONE IMPACTED IN THE NECK OF THE GALL-BLADDER.

(Museum, St. Bartholomew's Hospital.)

*Inflammation.* Inflammation invariably follows directly on the presence of gall-stones in the gall-bladder. It may be of a low grade chronic type which results in considerable fibrous thickening of the wall of the viscus. Acute inflammation of the gall-bladder following on the presence of stones is probably not so common as the simple chronic variety, but is of extreme importance owing to the pathological conditions which can themselves follow this complication (fig. 451).



*Complications of Acute Cholecystitis*

(1) *Local Peritonitis.* There must invariably be some degree of local peritonitis in the majority of cases of acute cholecystitis, and, depending on the virulence of the infecting organism, the local peritoneal inflammation may, or may not, spread with great rapidity. Protective adhesions may form, especially in the lower grades of inflammation, and the gall-bladder may rupture into the subhepatic area leading to an intra-peritoneal subphrenic abscess.

(2) *General Peritonitis.* In those cases of extreme rapidity of onset of the disease where local adhesions have had no opportunity to form, the gall-bladder may rupture into the general peritoneal cavity, followed by severe general peritonitis.

(3) *Subphrenic Abscess.* A subphrenic abscess may follow acute cholecystitis associated either with local or general peritonitis, and is often known to follow a so-called leaking gall-bladder, where numbers of surrounding adhesions have formed, preventing the infection from spreading to the general peritoneal cavity.

(4) *Empyema of the Gall-bladder.* Where there has been a pre-existing mucocele with a stone impacted in the neck of the gall-bladder the supervention of acute cholecystitis leads to the formation of an empyema. The gall-bladder then becomes nothing more or less than a bag of pus, and the complications mentioned above may follow.

(5) *Suppurative Pylephlebitis.* Acute inflammation of the gall-bladder has been known to result in phlebitis of the portal vein or one of its tributaries, followed by multiple abscess formation in the liver itself.

*Complications of Chronic Cholecystitis.* As has already been emphasised, some degree of chronic cholecystitis is extremely common in nearly all cases of gall-stones. The most interesting condition which may follow chronic inflammation of the gall-bladder is the formation of a fistula between the fundus of the gall-bladder and the duodenum. These structures are frequently closely related to each other anatomically, so that an inflammatory process of the gall-bladder may lead to adhesions forming between the gall-bladder and the duodenum. The pressure of a large single stone may gradually produce erosion of the

wall of the gall-bladder and then of the duodenum, and result in the passage of the stone into the intestinal canal. Such stones are the cause of a certain percentage of cases of acute intestinal obstruction.

*Obstructive Jaundice.* Gall-stones are certainly the commonest cause of obstructive jaundice in adult patients, the other, but less common, cause being carcinoma of the head of the pancreas. In the majority of cases of obstructive jaundice, the patient has had a preliminary attack of gall-stone colic followed by jaundice some hours later.

One important factor to be borne in mind in this connection is the explanation of the law enunciated by Courvoisier, which inferred that obstructive jaundice associated with palpable enlargement of the gall-bladder was invariably due to carcinoma of the head of the pancreas and not to gall-stones. The reason for this is that in gall-stones there is always chronic cholecystitis, the wall of the gall-bladder is fibrotic, and obstruction to the outflow of bile in the common bile-duct produces a certain increased pressure in the gall-bladder above this, not sufficient, however, to distend the gall-bladder. The normal thin-walled gall-bladder, which is to be expected in a case of carcinoma of the head of the pancreas, easily distends because of a similar bile pressure.

An exception to the rule is where a stone has become impacted in the cystic duct producing a mucocele of the gall-bladder, and where some oedema of the tissue has spread away from the cystic duct, causing pressure on the common hepatic duct together with some catarrhal inflammation of this structure. This could conceivably lead to obstructive jaundice resulting indirectly from a gall-stone and associated with an enlarged, palpable gall-bladder.

*Gall-stone Colic.* This clinical condition is not in itself associated with any special pathological changes.

*Carcinoma of the Gall-bladder.* Carcinoma of the gall-bladder is frequently associated with pre-existing gall-stones (Erdmann, *Ann. Surg.*, p. 1139, May, 1935). The factor of chronic irritation, which appears to be associated with carcinomatous change in many parts of the body, would seem to be responsible for cases of carcinoma of the gall-bladder. The growth is usually a columnar-celled carcinoma, although occasionally spheroidal-celled growths are seen. It tends to

spread down the ducts and to involve the liver by direct extension. It may spread to the colon, duodenum, and other surrounding structures.

*Acute Intestinal Obstruction.* As has already been mentioned, acute intestinal obstruction may follow the impaction of a large single stone in the small intestine. This can only occur where a cholecysto-duodenal fistula has formed. The stone is liable to become impacted at the duodeno-jejunal flexure, or at a point some two or three feet from the lower end of the small intestine. As the stone passes along the small intestine, it becomes progressively larger through faecal matter being deposited on it, while the calibre of the intestine itself becomes progressively smaller.

*Clinical Features of Gall-stones.* In considering the question of the symptomatology of gall-stones, it must be remembered that some cases do not appear to have any obvious symptoms.

*Flatulent Dyspepsia.* The symptoms of gall-stones are frequently those of flatulent dyspepsia, i.e. a feeling of fulness or discomfort in the upper part of the abdomen, particularly after meals, associated with much flatulence. There is frequently some nausea and occasional vomiting, and the patient may feel tenderness when pressing the upper part of the abdomen, or as a result of pressure from a tight garment.

*Gall-stone Colic.* Colicky pain starts abruptly in the epigastrium or right hypochondrium, frequently immediately after a meal. The pain is agonising, doubling the patient up and making him roll about on the floor. It is associated with marked coldness, vomiting, and much perspiration. The pain ceases as suddenly as it has started, and the description which the patient gives after an attack is frequently that of someone tying a cord round the upper part of his abdomen and rapidly tightening it until the pain can no longer be borne. Examination usually reveals considerable tenderness at the right costal margin.

The only treatment for patients suffering from gall-stone colic is the immediate administration of morphia to relieve the pain.

*Obstructive Jaundice.* Obstructive jaundice which follows an attack of gall-stone colic does not come on for some hours after the pain has subsided. In such cases the patient shows generalised jaundice of varying grades of severity. Hæmorrhages are present and itching of the skin is a very distressing symptom. Slight fever may

occur and occasionally, where there is a stone in the common bile-duct together with infection of the bile, repeated rigors may occur, thus producing the clinical picture of Charcot's intermittent hepatic fever.

*Other Complications.* Other complications which may accompany gall-stones are localised peritonitis, general peritonitis, subphrenic abscess, and acute intestinal obstruction.

## CHAPTER III

### CONGENITAL CYSTIC DILATATION OF THE COMMON BILE-DUCT

by

A. DICKSON WRIGHT

THIS condition affects the common bile-duct between the junction of the cystic duct and a point in the duct level with the upper border of the duodenum, i.e. about 2 inches above the ampulla of Vater. It is probably at first one of the achalastic conditions, but when the cyst reaches a certain size a valvular flap appears at the outflow from the dilated portion of the duct so that the condition passes from a passive dilatation to an obstructive condition. The cyst varies greatly in size; the largest case recorded contained one gallon of bile (Neugebauer). Females are almost exclusively affected, and cases have been reported from the still-horn foetus to the age of 56, the average being 15.

The symptoms are sufficiently characteristic to make the disease easily diagnosable if the condition is known and understood. A young girl has a smooth spherical tumour on the right side of the abdomen continuous with the liver dullness, and sometimes having a band of colonic resonance over it, the tumour being movable sideways but not up and down. She may be symptomless, or show signs of biliary and duodenal obstruction such as intermittent jaundice, colic, and cholangitic fever and rigors if the former, and indigestion and bloating if the latter.

X-rays can be of great value in diagnosis.

(1) Excretion pyelography may show right-side hydronephrosis of a minor degree from the pressure of the cyst on the ureter.

(2) Barium meal will show the first part of the duodenum and the pylorus depressed and pushed over to the left, and the hepatic flexure of the colon also at a much lower level than usual.

(3) Cholecystography gives a very characteristic picture of a high gall-bladder compressed into a comma-shape by the cyst, but still able to receive bile and concentrate it.

(4) Calcification in the cyst wall and hepatic ducts can sometimes be seen.

Complications are mainly those due to biliary obstruction, such as suppurative cholangitis and liver abscesses, and biliary cirrhosis of either hypertrophic or atrophic types. In one case of the writer's the right lobe (physiological) was atrophic and the left lobe was greatly hypertrophied and mistaken for the spleen. The diagnosis in this case had been for two years hepatomegaly and splenomegaly, the cyst being regarded as enlarged liver and the huge left lobe as enlarged spleen. Following on cirrhosis, gastric hæmorrhages and ascites may develop, the diagnosis of Banti's disease being made.

Rupture of the cyst is a possibility, as in the following case :

A girl of 13½ with no significant family history had been under treatment at hospital for two and a half years with attacks of indigestion, pain, jaundice, acholic stools and fever, and recently had developed ascites and large abdominal veins. The upper abdomen showed the two large tumours mentioned above. After a blow on the abdomen the girl developed severe abdominal pain and vomiting, and later the ascites and icterus became more marked and the abdomen very tender. Paracentesis yielded two pints of bile-stained fluid. Four days after the injury coma developed, and on the seventh day she died. Post-mortem showed the characteristic condition, a tremendously dilated common duct which had ruptured, causing a large retro-peritoneal biliary extravasation and bile-stained ascites. The mucous membrane of the common bile-duct was ulcerated and gangrenous in patches, and the liver showed the peculiar distribution of cirrhosis already mentioned.

The treatment of congenital cystic dilatation should only be undertaken when biliary obstruction develops ; the mere discovery of the cyst should not be the signal for operation because it is not uncommon for the cyst to have been present for as long as 30 years without much detriment to health, whereas no cases seem to live longer than three years after operation. The operation most commonly recommended is anastomosis of the cyst with the duodenum or stomach. This is a bad operation because gastric and duodenal contents enter the cyst and cholangitis

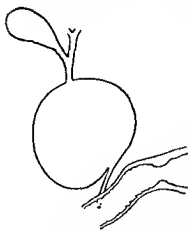


Fig 452.—CONGENITAL CYSTIC DILATATION OF THE COMMON BILE-DUCT.

Only a portion of the duct is affected as a rule. When the cyst enlarges further the gall bladder will become flattened out and lie on its upper surface.

ensues with fatal results after a length of time. The gall-bladder may be anastomosed to the stomach, which is a safer operation than the first, but is not often possible because of the size of the cyst.

The best operation is excision of the cyst with the exception of the portion between the hepatic ducts and the lower end of the common bile-duct. This gutter is reconstructed round a T-tube to form a new duct and the T-tube is then left in for three months or more; by this means the affected bile tree is drained, and when the T-tube is removed the bile enters the duodenum through the valve of Oddi, which prevents ascending infection.

The operative mortality is very high, 80-90 per cent, this being due to allowing the cases to go on too long without operating after biliary obstruction has once developed, and to the ignorance of the operating surgeon of the existence of the condition and how to tackle the surgical problem involved.

SECTION 7

LIVER

CHAPTER I

Liver

by

A. DICKSON WRIGHT

CHAPTER II

Jaundice

by

R. SLEIGH JOHNSON



## SECTION 7

### LIVER

#### CHAPTER I

##### LIVER

by

A. DICKSON WRIGHT

**INJURY** of the liver is the result of either penetrating wounds or contusions. The liver is sometimes injured also in falls from a height or in motor accidents through the sharp costal margin being driven into it by acute flexion of the trunk.

In penetrating wounds of the liver it is possible to arrest hæmorrhage by large stitches of catgut passed through the liver with a blunt liver needle, or by using an ordinary curved needle, eye end first. The stitches are tied over small omental pads to prevent cutting of the stitches. Drainage should be employed on account of the risk of bile leakage from wounding the ducts.

Tears of the liver, the result of contusion, are dealt with in the same way. Sometimes in these cases a seven-day iodoform gauze pack has to be used on account of the difficulty of sewing up, and in some cases living fascial sutures taken from the rectus sheath may be used. If a piece of the liver is observed to be torn off and is free in the peritoneal cavity it should be looked for carefully and removed on account of the risk of autolytic peritonitis (when a piece of the liver of the dog is detached and left free in the peritoneal cavity the animal always dies in 24 hours from an overwhelming foaming peritonitis due to the *B. Welchii*—autolytic peritonitis).

Recoveries from extensive liver injuries are sometimes amazing, provided the loss of blood is rectified by transfusion of the blood found in the peritoneal cavity after filtration through gauze, or if this blood is too clotted or contaminated the services of a compatible blood donor should be obtained.

ABSCESSES of the liver are of four kinds :

- (1) Tropical or amœbic abscesses.
- (2) Septicæmia abscesses.
- (3) Portal pyæmic abscesses.
- (4) Cholangitic abscesses.

Tropical abscess is due to the amœba histolytica, and is the final stage of a condition of amœbic hepatitis which with care can be diagnosed before the formation of the abscess. If at this stage thorough emetine treatment is carried out the hepatitis can be cleared up before necrosis and abscess formation have occurred. This amœbic hepatitis manifests itself as a pyrexial illness with hepatic tenderness and enlargement occurring in a person with a dysenteric history and amœbæ in the stools. The blood count also resembles that found in amœbic abscess, viz. a marked leucocytosis without the polymorphonuclear preponderance found in the usual suppurative processes.

*The Symptoms* of tropical abscess are sometimes so classical that the diagnosis is of the easiest, in other cases the picture is by no means clear, and diagnoses of cholecystitis, duodenal ulcer, appendicitis, pleurisy, tuberculosis, etc., are made. The general symptoms are those of a continuous remittent fever with drenching night sweats, anorexia, and exhaustion; emaciation and slight icterus give the patient a characteristic cachectic appearance. Sometimes rigors occur when the abscesses are multiple or the infective process very severe. The history generally discloses dysenteric attacks, although as a rule at the time of the abscess development dysenteric symptoms are absent, and it is an interesting point that the more common solitary abscess of the right lobe of the liver is usually associated with a small ulcerative process in the cæcum not enough to cause any symptoms, whereas the much less frequent multiple liver abscesses are found in association with extensive colonic disease and marked symptoms.

The local signs of the disease are hepatic enlargement and tenderness; rarely the abscess may form a fluctuant swelling appearing below the ribs; sometimes œdema of the thoracic or abdominal wall can be seen. Tenderness on pressure over the thoracic wall may indicate the situation of the abscess, and in some cases deep breathing and coughing may cause pain. Especially characteristic is the pain on sudden movements such as on turning in bed, and to prevent this to some extent the sufferer keeps his arm pressed to his ribs ("carries his abscess under his arm"). Pain may also be referred to the shoulders, scapular regions, and right iliac fossa. The right base of the lung often shows changes such as impaired air entry, crepitation, and even consolidation from

inhibition of diaphragmatic movements, and sympathetic congestion of the lung due to the contiguous process in the liver. X-ray will reveal diminished diaphragm movements and possibly localised hulging of the diaphragm produced by the abscess.

The trend in the treatment of liver abscess is towards conservatism; the older methods of precipitate exploration and drainage carried a very high death-rate, which is now greatly reduced by dealing with the cases more gently.

Firstly, on the appearance of liver signs the amœba is carefully looked for in the stools and the blood count is done, and then emetine is given daily in 1 grain doses, and improvement in general condition and blood count watched for. If this does not occur, then X-rays are carried out, and if these and local signs do not give the position of the abscess, exploratory aspiration is resorted to, using a  $3\frac{1}{2}$ -inch needle of wide bore. A preliminary  $\frac{1}{4}$  grain of morphia is given, and a line is drawn from the middle of the 12th rib horizontally forward to mark the pleural reflection, the surface marking of the liver is then drawn on the chest wall, and the needle inserted 1 inch above the pleural reflection in the mid-axillary line. It is first inserted the whole of its length towards the most usual place for liver abscess, viz. the upper and back part of the right lobe. The needle is then attached to a Potain's vacuum bottle or aspirating syringe and slowly withdrawn. If no pus is found the liver is needled in other directions until pus is found. Once found all the pus is aspirated and measured, and one half its volume of 1/1000 quinine or 1/1000 yatren is injected, allowed to stay five minutes and aspirated again. This instillation may be repeated until the fluid comes out clear. Before removing the needle its direction is well noted, and may be remembered best by painting a little silver nitrate solution on the point on the opposite side of the chest where the needle would emerge if the thorax were transfixed.

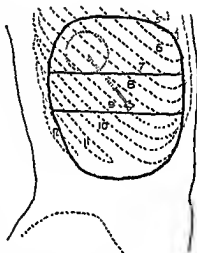


FIG 453.—ASPIRATION OF LIVER. THE SHAPE OF THE LIVER IS MARKED OUT ON THE SKIN WITH A FLESH PENCIL. A HORIZONTAL LINE DRAWN FROM THE MIDDLE OF THE 12TH RIB MARKS THE PLEURAL REFLECTION AND A PARALLEL LINE 2½ INCHES ABOVE THIS, THE LOWER LIMIT OF THE LUNG. THE NEEDLE IS PASSED AS SHOWN.

After aspiration a very firm binder is put on and the patient continues to receive emetine daily. If no improvement occurs aspiration

is continued up to six times, and then operation is decided upon. If examination of the pus shows secondary infection then aspiration is not persisted in, but drainage is carried out. A large number of cases are cured by simple aspiration plus emetine, and this is a most satisfactory outcome to any case.

Drainage is first done, if feasible, through a tube inserted through a cannula passed along the line of the aspirating needle. If the tube is passed through the cannula on the stretch, and the cannula removed, the tube will when released fill the cannula track very tightly, stopping leakage and bleeding.

Open operation is not very often indicated nowadays, cases which resist aspiration or cannula drainage being generally very severe cases of multiple abscess which are doomed from the start. However, in certain situations open drainage is probably better, as in abscesses in the left lobe or anterior part of the right lobe. Drainage is carried out through an appropriate abdominal incision, a large tube packed round with iodoform gauze being used.

Abscesses in the right lobe can be drained laterally after resection of a portion of the 9th rib, and brushing back the pleura with gauze or securing adhesions between parietal and diaphragmatic pleura by leaving an iodine pack in the wound for seven days. The suturing of the two layers of the pleura together is not a safe way of preventing empyema.

The posterior part of the right lobe can be drained by resecting the 12th rib, pushing the pleura upwards and then inserting a drainage-tube into the liver, giving the vena cava a wide enough berth.

*Complications of Liver Abscess.* Rupture of the abscess into the lower bronchus is the commonest accident, and terminates in the large majority of cases quite favourably, this rather inadequate drainage being sufficient to cure the abscess provided emetine is given.

Rupture into the peritoneum provokes an abdominal catastrophe much resembling perforated duodenal ulcer. The prognosis is not as bad as would be expected, amœbic pus not being as dangerous as true pus. Drainage of the liver abscess and the peritoneal cavity, if combined with active medical measures, results in a large proportion of recoveries.

Rupture into pleura and pericardium are rare and are serious emergencies. Rupture may rarely occur into stomach, duodenum, colon, bile-ducts, renal pelvis, and even the vena cava. Metastasis to other parts of the body such as spleen or brain is extremely rare. Secondary infection

of the abscess with bacillus coli or streptococcus is fairly common and rather a serious occurrence, as it excludes the safer and simpler methods of treatment and makes open drainage essential.

The prognosis of hepatic amœbiasis is steadily improving with careful surgical treatment, and nowadays mortality is in the neighbourhood of 20 per cent. Multiplicity of the abscesses and secondary infection all make for bad prognosis.

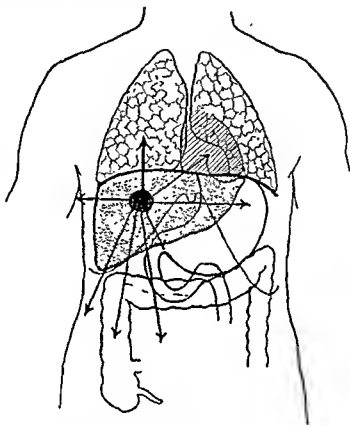


Fig. 154.—DIAGRAM TO SHOW SOME OF THE ROUTES OF BURSTING OF LIVER ABSCESS (after Cope). THE THICK ARROW INDICATES THE USUAL ROUTE.

The other abscesses of the liver have little surgical application because they are generally terminal complications in hopeless septic conditions. There are, however, two types of abscess in which there is some hope.

(1) The solitary staphylococcal abscess occurring in association with boils, especially in adolescents.

(2) The solitary abscess of the right lobe, which develops with a rigor and all signs of a subphrenic abscess two months after suture of a perforated duodenal ulcer. If in such a case no X-ray signs of subphrenic abscess can be found, then the liver should be needled for the abscess.

In cases of portal pyæmia the portal vein has been occasionally ligatured to stop the occurrence of further abscesses.

*Portal Cirrhosis* has only surgical aspects in the earlier stages of ascites or hæmatemesis. The cases are usually passed on to the surgeon when death is near at hand, although there is good reason to believe that operation done in good time lengthens life in this disease and prevents ascites. Death from hepatic insufficiency is much more pleasant than death from ascites. The operation performed is known as the Talma-Morison operation (Narath's modification), and consists

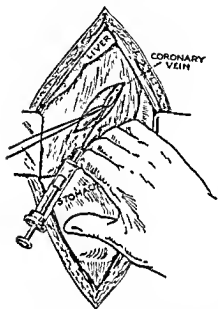


Fig 455.—LIGATION OF THE CORONARY VEIN. INJECTION OF 10 CC. OF SODIUM MORRHUATE DISTAL TO THE LIGATION IS A VALUABLE PROCEDURE IN CASES OF HÆMATHEMESIS FROM PORTAL CIRRHOSIS.

in mid-line epigastric laparotomy and withdrawal of a good-sized piece of omentum. The wound is closed in part, leaving a non-constricting gap for the omentum, and to the edges of this gap the omentum is fixed by fine sutures. The external mass of omentum is then thrust into a pocket hurried out in the subcutaneous fat. The object of this operation is to provide more anastomotic channels between portal and systemic circulations than are provided naturally at the umbilicus, rectum, and œsophagus. If ascites is very troublesome, permanent drainage may be provided by glass tubes pushed through the peritoneum and the muscles when the abdomen is open and conducting the

fluid into the subcutaneous fat. Leashes of silk threads may also be used, and the saphenous vein may also be transplanted into the peritoneal cavity and drain the ascitic fluid into the femoral vein.

Surgical treatment of hæmatemesis is often of value as many of these patients die of œsophageal hæmorrhage when there is still enough liver function to maintain life for some years. Ligature of the coronary vein is the best procedure, and may be combined with sclerosing injections of sodium morrhuate into the prominent varicose veins at the œsophago-cardiac junction.

*New Growths of the Liver* have little surgical application. The majority of growths are secondary malignant growths and inoperable.

Occasionally a solitary malignant nodule has been excised at the same time as a carcinomatous stomach or colon.

Primary carcinoma of the left lobe of the liver has been removed on a few occasions, bleeding being arrested by ligature of the left hepatic and portal vessels in the portal fissure, and by interlocking sutures in the liver, and by use of the endothermy knife when cutting the liver.

*Hydatid Disease of the Liver.* The hydatid cyst is the intermediate cystic stage of a  $\frac{1}{8}$  inch long tapeworm of the dog (*tænia echinococcus* or *echinococcus granulosus*). The eggs of this worm are passed in the faeces of the dog and are swallowed by the human being (generally a child). Before being swallowed, or shortly after, the ovum develops into a small six-hooked (hexacanth) embryo, which perforates the stomach wall and via the portal vein arrives in the liver; here it is arrested by the liver filter except in 30 per cent of cases, when it passes on to the lung filter, where another 15 per cent are held up, while the remaining 15 per cent which pass the lung filter are deposited in the skin, muscles and other parts of the body.

In the liver the hexacanth embryo develops into a cyst which consists of three layers, the outer one of fibrous tissue derived from the host and the inner two provided by the parasite. The inner of these two, the granular layer, is cellular, and the outer a protective layer of hyaline, known as the laminated membrane because of its structure. The cyst cavity contains a clear fluid containing salt and a little protein. Small buds form on the granular layer, and these become vesicles known as brood capsules, from the lining of which small buds of cells form which quickly develop into the heads of the future tapeworms, and these are known as scolices. Should the development of the cyst be disturbed by trauma portions of the wall become detached and form into two-layered daughter cysts floating in the interior of the main cyst (endogenous daughter cysts). Sometimes the daughter cysts extrude from the main cyst (exogenous daughter cysts).

The diagnosis of these cysts arises when there is a hepatic tumour together with a local prevalence of the disease, and is confirmed by two tests—the complement fixation test, using hydatid fluid obtained from the sheep as antigen, and the production of local allergy on injecting the same hydatid fluid under the patient's skin (Casoni test). The position of the cyst is determined by clinical examination (fortunately 70 per cent of cysts are present on the under-surface of the liver) and by X-ray (30 per cent being present on the upper surface). (These

figures are the exact reverse to those of liver abscess.) The treatment consists in operation through an incision placed directly over the cyst, and in order to do this there should be no hesitation in resecting a rib, obtaining pleural adhesions by a 7-day gauze pack, and then carrying on through the obliterated phrenico-costal angle and diaphragm. Should an abdominal incision not give easy access to a posterior cyst there should be no hesitation in closing the incision and making a fresh one at the back.

When the cyst is simple, as in children, the cyst is punctured, a little fluid drawn out, a syringeful (2 to 10 cc.) of commercial formalin injected, and barbotage done to mix the fluid thoroughly. After five minutes the contents of the cyst are dead, and guide stitches are inserted, a small opening made in the cyst, and a sucker inserted to evacuate the fluid. The cyst is then opened widely and the lining wiped out with gauze. If the size of the cavity can be reduced by excising some of the adventitia this should be done. The cavity is then completely closed after filling it with saline and the abdomen closed. Drainage is to be avoided if possible, and is only used when bile or pus is present in the cyst, and when used the tube must be judiciously placed to give easy drainage with gravity. Marsupialisation is an unwise procedure leading to a chronic sinus which may be a great handicap.

When granddaughter cysts are present neither aspiration nor formalin sterilisation can be carried out, so special steps are taken to counteract implantation of brood capsules into the peritoneum. Black or dark green towels are carefully placed to prevent this and to visualise any spilt hydatid structures which show up white on the towels. The cyst is then holdly opened between guide sutures with a diathermy needle, and a powerful electric sucker with a special large bore tube is used to suck out the contents. The clearing of the cavity is continued with gauze swabs dipped in 5 per cent formalin, and when the cavity is empty it is closed by suture, the packs carefully removed, and the abdomen closed without drainage unless pus or bile was present in the cyst.

Pedunculated cysts can be removed completely and calcified dead cysts are best left alone.

Complications of hydatid cysts resemble those of tropical abscess; rupture into adjacent cavities can occur, suppuration and pointing on the abdominal wall. One complication of hydatid does not occur with tropical abscess, viz. rupture into the main bile-ducts. This event produces biliary colic, fever and jaundice, and is treated by evacuating



the cyst thoroughly and exploring the common duct and removing hydatid material from it.

*Granulomata of Liver.* The most important is the gumma, which forms a smooth liver tumour, unlike the irregular umbilicated carcinoma of the liver. Gumma of the liver may reach huge dimensions, as in the following case :

A woman of 40 with abdominal pain and vomiting was found to have a large epigastric mass. On exploratory laparotomy a giant spherical tumour was found protruding from the under-surface of the liver. It was the size of the largest grape fruit. A biopsy was made and reported on as cirrhotic liver tissue. The diagnosis was not made until other characteristic gummatous lesions appeared on the face and neck six months later, and then appropriate therapeutic measures caused the liver tumour to disappear.

*Tuberculosis of the Liver* is just a pathological curiosity, and actinomycosis is secondary to lesions of the alimentary canal, producing a very characteristic "honeycomb liver," which is only of pathological interest because once the liver has become involved surgical removal of infected intestine is a waste of time.

## CHAPTER II

### JAUNDICE

by

R. SLEIGH JOHNSON

JAUNDICE may be defined as a staining of the body tissues with bilirubin. Its causes are manifold, covering a wide field of medical and surgical interest, so that the problem of elucidation in a given case never fails to provide opportunity for clinical judgment and diagnostic skill. The recent work of McNee has thrown much light on the intricacies of its pathology and helped towards a clearer understanding of a difficult and complicated subject.

Before considering the different varieties of jaundice, it is necessary in the first instance to review in the light of these observations the main features in the physiology of bile-pigment formation. The ultimate derivation of bile-pigment is from the hæmoglobin of red blood cells, liberated on their death and disintegrated into the circulating blood-plasma. During its passage through certain organs the liberated hæmoglobin becomes picked out and removed from the blood stream, to undergo a sequence of changes which separate off its iron-containing derivative, and to be finally excreted in the bile as bilirubin. The opinion was formerly held that the site of conversion into bile-pigment was actually within the substance of the polygonal liver cells, but recent work has shown that this function is apparently confined to a highly specialised group of phagocytic tissue cells known as the reticulo-endothelial system. These cells are spread widely throughout a number of organs, being most numerous in the pulp of the spleen and in the liver, where they are known as Kupffer cells. They are also contributed in smaller numbers by the endothelial cells of lymphatic glands, suprarenal glands and bone-marrow, the reticular cells of the thymus and the interstitial cells of the testis, all of which possess certain staining reactions in common, characteristic of their common function of converting hæmoglobin into bilirubin or bile-pigment.

The greater portion of this process is certainly concentrated in the Kupffer cells of the liver, which lie as large endothelial cells scattered on the walls of the portal capillaries. These in their turn run among and surround the polygonal glandular cells of the liver lobule and separate them from the bile-capillaries (fig. 450). It is therefore evident that between the circulating blood in the portal capillaries and the capillaries of the biliary system there is interposed a layer of polygonal cells, through which the converted bile-pigment must normally pass

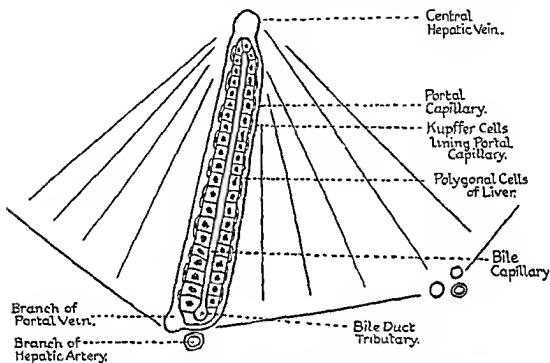


Fig. 450.—STRUCTURE OF LIVER LOBULE (DIAGRAMMATIC), TO ILLUSTRATE NORMAL PROCESS OF BILE-PIGMENT FORMATION.

before it is collected in the bile-ducts proper and excreted into the small intestine. The polygonal cells do not share with the Kupffer cells the power of forming bilirubin, their function being rather excretory in collecting the altered pigment and passing it through into the lumen of the bile-capillaries. There nevertheless occurs in its transit through their substance a change in the composition of the bilirubin, detectable by a variation in chemical reaction. In this way it is possible to determine the origin and type of an excess of bile-pigment in the circulating blood, and some help is so afforded in the differential diagnosis of the nature of jaundice present.

For clarity this process may be summarised as under :

- (1) Liberation of hæmoglobin into the blood-plasma by degeneration of red blood cells.

- (2) Conversion of hæmoglobin into bilirubin by the Kupffer cells of the reticulo-endothelial system.
- (3) Transmission of bilirubin through the polygonal cells of the liver into the bile-capillaries, some modification in its composition taking place in its passage, responsible for a change in chemical reaction.

The biochemical test which allows of distinction between these two varieties of bilirubin is the *Van den Bergh reaction*. In brief, it is found that bilirubin which has passed through the liver cells, as in bile from the gall-bladder, gives an immediate positive direct reaction, while the bilirubin normally circulating in the blood-plasma, and not therefore having passed through the liver cells, gives a delayed or negative reaction to the same test. By a modification of the test known as the indirect Van den Bergh reaction the latter type of bilirubin gives a positive result and can thereby be detected and measured. Details of the test and interpretations of its results are for convenience described later (see page 752).

Following an exhaustive study of the subject from its clinical and biochemical aspects, McNee (1) describes a clinical classification of jaundice into three main groups :

- (I) *Obstructive hepatic jaundice.*
- (II) *Hæmolytic jaundice.*
- (III) *Toxic and infective hepatic jaundice.*

The underlying pathology based on this classification and theory may be briefly reviewed.

(I) *Obstructive hepatic jaundice.* In the first group the bile after a normal formation is prevented from escaping by the natural route into the intestine by a mechanical obstruction of the biliary channels. The possible causes of obstruction, which are many, will be considered in detail at a later stage—a concrete example for purposes of illustration being afforded by the complete blockage of the common bile-duct by a stone. For a time at least the normal process of bile-formation continues, distension of the larger channels being followed by a rising pressure and progressive dilatation of the minute bile-canalculi in the liver lobules, which become dammed up with dark concentrated bile. In long-standing cases histology shows the presence of plugs or casts of inspissated bile with branching processes extending into the substance of the liver cells. The bile-pigment which cannot be passed on into the

hiliary passages can at first be stored within the substance of the polygonal cells. When, however, these cells have become filled to their maximum capacity, the bilirubin can no longer be retained and therefore diffuses out into the surrounding hepatic blood-capillaries and so enters into the general circulation, together with the associated bile-salts and cholesterol. The excess of bile-pigment found, therefore, in the blood-serum in cases of obstructive jaundice is of the type which has passed through the polygonal cells of the liver, and gives a prompt direct Van den Bergh reaction. The threshold of the kidney for this form of bilirubin being usually exceeded, bile-pigment appears in the urine.

(II) *Hæmolytic jaundice.* This term, known also as hæmatogenous jaundice, is applied to a group of conditions all of which are characterised by a simple increase in the blood-serum of bilirubin of the type normally present. Such an excess is found to accompany increased blood destruction from whatever cause, a frequent example being pernicious anæmia. The conversion of hæmoglobin into bilirubin by the Kupffer cells being thus excessive, the whole amount cannot be passed out by the polygonal cells into the biliary channels sufficiently rapidly to prevent some escape of pigment directly into the main hepatic veins. If the rise in bilirubin content of the serum is marked, it will be shown in a yellowish discoloration of the skin, but with lesser degrees this may not be evident and the jaundice is then termed *latent*, the excess being revealed only by estimation in the indirect Van den Bergh test. There being no obstruction of the polygonal cells, the results of the immediate direct test are negative. Moreover, unless hæmolysis is marked bile-pigment seldom appears in the urine, the kidney threshold for this normal form of serum-bilirubin being considerably higher than for the other variety. The urine, however, is frequently dark in colour, not from bile-pigment, but from the presence of excess of urobilin (stercobilin) which is normally re-absorbed by the liver from the intestine. It is thought that owing to alteration of function this conservative recovery and re-excretion of broken-down bile-pigment is upset, the excess of urobilin being passed in the urine. It is in the diagnosis and measurement of this type of jaundice that the Van den Bergh reaction proves of greatest value and precision.

(III) *Toxic and infective jaundice.* This third clinical variety of jaundice comprises a large and diverse group of affections of the liver and bile-passages, in which there is presupposed a combination of the two elements of obstruction to the outflow of bile and damage by

- (2) Conversion of hæmoglobin into bilirubin by the Kupffer cells of the reticulo-endothelial system.
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biliary passages can at first be stored within the substance of the polygonal cells. When, however, these cells have become filled to their maximum capacity, the bilirubin can no longer be retained and therefore diffuses out into the surrounding hepatic blood-capillaries and so enters into the general circulation, together with the associated bile-salts and cholesterol. The excess of bile-pigment found, therefore, in the blood-serum in cases of obstructive jaundice is of the type which has passed through the polygonal cells of the liver, and gives a prompt direct Van den Bergh reaction. The threshold of the kidney for this form of bilirubin being usually exceeded, bile-pigment appears in the urine.

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infection or poisoning of the glandular substance of the liver. An example may be given in the common "catarrhal jaundice" of adolescents and young adults. It is thought that here, owing to a toxic degeneration of the polygonal cells, some bilirubin fails to be taken up from the plasma and to be passed on by the damaged cells into the bile-capillaries, being absorbed unaltered into the circulation; an excess of the normal serum-bilirubin is therefore found, giving a raised indirect Van den Bergh reaction. In addition, there is considered to be some degree of organic obstruction to the outflow of bile by inflammatory swelling of the minute bile-capillaries, leading to retention and re-absorption into the blood stream of some bilirubin already excreted by polygonal cells that have escaped toxic degeneration and are still functionally active. In such cases both varieties of bilirubin are therefore found in the serum, and the response to Van den Bergh's test usually shows a corresponding modification, combining the features characteristic of each and known as a *biphasic reaction*. The results are, however, inconstant and subject to rapid variation. For convenience the principles of the reaction may be described at this stage:

#### VAN DEN BERGH REACTION

The reaction is a very delicate test for bilirubin by the use of Ehrlich's diazo-reagent. This is freshly prepared immediately before the test by the mixture in standard proportions of a solution of sulphanilic acid in HCl and a solution of sodium nitrite, details of technique being fully described in the appropriate text-books. As first introduced the test was carried out on the blood-serum obtained after allowing the clotting of blood removed from a vein. With a revised technique, McNee and Keefer (2) have later recommended the use of the blood-plasma, separated by centrifuging about 10 cc. of blood freshly collected in an oxalated tube and removing the clear upper layer with a pipette; it is claimed that more delicate and reliable results are thereby obtained. The *direct reaction* is carried out by the addition of the diazo-reagent to the separated plasma with suitable control tubes. It is found that the test is rendered more sensitive by the addition of a small flake of caffeine sodium salicylate. In the normal subject no colour change is seen and the direct reaction is therefore negative. A positive direct result may take one of three forms, as under:

(a) *An immediate direct reaction.* Here a bluish-violet colour develops at once, increasing and reaching its maximum intensity in ten to thirty seconds, the depth of colour being proportional to the content of bilirubin.

(b) *A delayed direct reaction.* There is no immediate change, but a reddish colour begins to appear within about 15 minutes, gradually deepening to violet.

(c) *A biphasic direct reaction.* In this case a reddish colour appears immediately, but the change of blue to violet is considered prolonged, e.g. for half an hour.

*The Indirect Reaction.* The indirect reaction affords a means of detecting and estimating the bilirubin resulting from hæmolysis. The test is extremely delicate, detecting one



part in a million of bilirubin, and a positive qualitative result is therefore obtained with normal blood. If the direct reaction is negative, the indirect test is then made by the further addition to the mixture of plasma and diazo-reagent of a standard quantity of 96 per cent alcohol and ammonium sulphate solution. A positive result is shown by the immediate development of a violet colour. By colorimetric estimation against a standard solution a quantitative measurement can be obtained of the concentration of bilirubin in the plasma, expressed in units, one unit of bilirubin representing a concentration of 1 in 200,000. The standard employed for colour matching is a solution of ferric thiocyanate in ether, of a tint exactly representing one unit of bilirubin, which remains stable over several months. A standard "unit" of anhydrous cobaltous sulphate solution may alternatively be used.

#### INTERPRETATION OF RESULTS

The test is of definite value, provided that its limitations are clearly recognised and no attempt made to exceed them. A prompt direct reaction confirms the presence of obstructive jaundice, which is, however, in most cases equally evident from clinical examination. In the differential diagnosis, however, of the cause of the obstruction, e.g. of stone from growth, the test fails to give any help. It is to be remembered that in any case where a direct positive result is obtained, the indirect result after addition of alcohol is also automatically positive, although the converse does not hold good. In obstructive jaundice the concentration of bilirubin in the plasma and in the urine is greatly raised and may reach a figure of 50 units or more.

It is in the distinction of mild or early forms of obstructive from hæmolytic jaundice that the Van den Bergh test does give decisive information. In the hæmolytic form, as in pernicious anæmia and acholuric jaundice, delayed response may be obtained to the direct reaction, but the immediate response is always negative. The indirect test, which is positive, shows a quantitative increase in the number of units of bilirubin. Where the red cell destruction is markedly excessive, this increase may be as high as 10 or 12 units. The normal bilirubin content of the blood-plasma lies within limits of 1 in 1,000,000 and 1 in 400,000, i.e. 0.2 and 0.5 units.

As already mentioned, a biphasic reaction indicates a combined type of jaundice and is characteristic of the wide group of toxic and infective cases.

#### RENAL THRESHOLD FOR BILIRUBIN—LATENT JAUNDICE

The presence or absence of bile-pigment in the urine will depend upon the level of bilirubin in the plasma above which the kidney allows

of its passage. The renal threshold for bilirubin is found to vary according to the type of pigment. In obstructive jaundice any increase above 4 units of bilirubin in the blood is accompanied by excretion of the pigment in the urine. In hæmolytic jaundice, however, as already noted, the threshold is much higher, and 10 or 15 units of bilirubin may be retained in the blood without leakage into the urine, which remains therefore free from bile.

Where the increase of bilirubin in the blood-plasma is too small to bring about either bile in the urine or a detectable discoloration of the skin, the term "latent jaundice" is applied. In the majority of such cases its origin is hæmolytic and associated with anæmia, but an early obstructive type also may be first, or indeed only, revealed by blood examination, e.g. in cirrhosis or secondary malignant disease of the liver, where a prompt direct Van den Bergh reaction may be obtained before clinical jaundice is evident.

A further reaction used to detect bile-pigment in the blood is *Fouchet's test*, depending on the development of a greenish-blue colour on adding the reagent to the blood-serum on a porcelain surface, the colour gradually deepening for about twenty minutes. The reagent used is a solution of ferric chloride and triehloroacetic acid. The reaction is somewhat less sensitive than the Van den Bergh test, giving a positive result only in concentrations of bilirubin greater than 1 in 60,000, a negative reading being therefore obtained with normal blood.

A useful rough guide to the progress of a case of jaundice is afforded by estimation of the *Icterus Index*, which is a simple colorimetric measure of the depth of bile-staining of the serum or plasma against a standard solution of potassium bichromate. Fallacies due to hæmolytic or opacity of the serum must be avoided, but apart from such errors the index in a given case will render evident an increase or decrease in the intensity of the jaundice.

This review of the current theories of the pathology of jaundice would be incomplete without reference to the classification set forth by Arnold Rich (3). His two main groups of *Retention Jaundice* and *Regurgitation Jaundice* correspond in general to the *Hæmolytic* and *Obstructive* types respectively of McNee. With regard to the former group, he holds, however, that excessive production of bilirubin alone, as in hæmolytic conditions, is insufficient to produce jaundice without the added factor of some derangement of liver function and interference with excretion. The classification recognises also a third form of *Combined jaundice* in which there is both excessive production of

bile-pigments by the reticulo-endothelial system and a regurgitation into the blood stream of bile which has been excreted by the polygonal cells into the bile-canaliculi. The different chemical reactions of bilirubin in the two main types are explained as being due, not to an alteration in its composition, but rather to the presence or absence of the other constituents of bile. In the group of retention (hæmolytic) jaundice, bile-pigment alone is in excess and unaccompanied by retention of bile-salts and cholesterol. The pigment is then held to be adsorbed to the proteins of the blood-plasma and gives a positive indirect reaction only, the direct reaction being negative. In regurgitation (obstructive) jaundice, not only the pigment, but all the constituents of the bile are normally retained, under which circumstances it is claimed that the bilirubin is prevented from becoming adsorbed and gives an immediate direct Van den Bergh reaction.

Retention of pigment alone without accompanying accumulation of bile-salts is spoken of as *Dissociated jaundice*, and is characteristic in the main of the retention or hæmolytic group. In the later stages of catarrhal jaundice, however, while pigments are still retained the excretion of bile-salts may return to normal and a dissociated jaundice thus be present.

The clinical features may now be reviewed in greater detail, based upon McNee's classification.

## I. OBSTRUCTIVE JAUNDICE

The general signs and symptoms may be first considered before progressing to individual causes.

The most obvious sign of obstructive jaundice is the staining of the tissues by bile-pigment, which appears first in the ocular conjunctiva or sclerotic as a light yellow discoloration. Its presence becomes rapidly evident in succession in the mucous membranes of the lips and palate, the finger-nails, the skin of the face, neck, trunk and limbs. The ease with which even a considerable degree of jaundice may be overlooked in artificial light must be remembered. It is also more readily observed in the fair-complexioned than in dark-skinned subjects. The involvement of the sclerotics enables this form of jaundice to be distinguished from the lemon-yellow colour of the skin in Addisonian anaemia or the special form of pigmentation seen in diabetics (carotin), in whom the sclerotics escape pigmentation or, on the contrary, are unusually transparent and bluish in hue. In obstructive jaundice

almost all the tissues of the body become bile-stained, the only exceptions being the structures of the central nervous system and cerebro-spinal fluid, with the tears, saliva and milk. Pathological secretions and exudates occurring from intercurrent inflammatory lesions in a jaundiced patient are also bile-stained, e.g. pleural fluid or pneumonic sputum. In the great majority of cases bile-pigment and bile-salts are present in the urine. If there is complete obstruction to the common bile-duct, after the intestine is once emptied the stools cease to contain bile-pigment and have special features described below.

According to its duration and the completeness or otherwise of the obstruction, the degree of staining of the tissues varies, progressing through a deep orange bronzing to olive-green or sometimes almost black, in the most chronic cases of some years' standing, the latter change being due to a conversion of the bile-pigment in the skin from bilirubin to biliverdin. In these cases other skin changes also are seen, including multiple xanthomata or xanthelasmata due to the local deposit of cholesterol. These usually form flat yellowish plaques situated most commonly on or around the eyelids. Telangiectases (spider naevi) are also apt to develop, especially on the face, with liability to spontaneous purpuric hæmorrhages. In this connection is related the increased tendency to severe bleeding at operation common to all conditions of jaundice.

The urine varies in colour from a deep amber to an intense orange-red or dark reddish-brown. In very chronic cases it may, like the skin, be dark olive-green in colour. The simplest test for bile-pigment is the ready frothing on shaking in a half-filled test-tube, the froth being coloured bright yellow. Tincture of iodine run on to the surface of bile-stained urine causes the appearance of a green ring at the junction of the two fluids, through oxidation to biliverdin. Gmelin's test employs the same principle, a play of colours, of which green is the essential band, being produced by the action of fuming nitric acid run down the side of a test-tube containing the bile-stained urine. The test can more delicately be carried out on a filter paper through which the urine has first been filtered to concentrate the bile-pigment, or alternatively upon a porcelain tile.

Bile-acids, usually less abundantly present than bile-pigment, or sometimes even absent, are examined for by Hay's test. The lowering of the surface tension of the urine through the presence of dissolved bile-salts causes powdered sulphur sprinkled upon its surface to sink, whereas upon the surface of normal urine it floats.

During recovery from an obstructive jaundice, bile-pigments may

disappear from the urine despite the persistence of staining of the skin, the bilirubin in the blood having fallen below the renal threshold of four units, and being therefore no longer passed through the kidney substance.

Bile-salts, present abundantly in the urine in the early stages of an obstructive jaundice, then usually diminish rapidly in amount and may even disappear, giving rise to one form of dissociated jaundice. Whereas the production of bile-pigment seems to continue unabated, that of bile-salts soon becomes lessened in degree, the disproportion being reflected in the urine.

Bile-stained hyaline casts are often found in a jaundiced urine and do not necessarily signify independent renal damage. Where failure of liver function occurs, however, i.e. cholæmia, albumen and blood cells also occur as evidence of trauma to the kidney through elimination of toxic substances. The stools also show characteristic changes from the deprivation by the intestine of bile. This being the normal stimulant to peristalsis, constipation is the rule, with passage of pale, offensive, bulky motions, containing 50 per cent or more of undigested and unabsorbed fat, present largely as fatty acids. Fermentation is excessive, with flatulent distension, loss of appetite, nausea or vomiting. The interference with absorption of fat, as well as, indirectly, of other articles of food, results usually in rapid and progressive loss of weight. When the obstruction to the entrance of bile into the intestine is complete, the motions are of a pallor and appearance described as "clay-coloured." With an incomplete or intermittent obstruction they contain a varying amount of bile-pigment.

Other well-known symptoms most marked in the early stages of an obstructive jaundice include headache, irritability, mental depression or drowsiness, a slowing of the pulse-rate with coincident fall of systolic pressure and intense itching of the skin, all of which are considered to be caused by retention of bile-salts in the circulating blood. Scratch-marks are abundant, while the pruritus if persistent may become the most serious aspect of the case, to the point of inducing insomnia and mental changes, or even suicidal tendencies. Yellow vision or xanthopsia rarely occurs. More serious nervous symptoms progressing to delirium, convulsions and coma may uncommonly follow, indicating a severe derangement of liver function, with an ultimate fatal issue.

The retention in the blood of bile-salts and bile-pigments has already been noted, the concentration of the latter being often extremely high, with invariably a prompt direct Van den Bergh reaction. Other blood changes are slight except for some retention of cholesterol.

In obstructive, as contrasted with hæmolytic, jaundice the resistance of red cells to hypotonic saline solutions is increased rather than lowered. The greater liability to hæmorrhage, spontaneous or operative, has been already mentioned and is confirmed pathologically by estimation of the coagulation-time, which may be prolonged up to as much as twenty minutes; its surgical significance is obvious, and constitutes one of the most serious risks.

Enlargement of the liver is the rule, from a damming-up of the biliary channels throughout its substance, while in many cases other factors, according to the ætiological cause of the jaundice, may contribute to the enlargement. The presence of a detectable swelling of the gall-bladder will also depend upon the causal nature of the jaundice, as described below.

*Causes.* The possible causes of obstructive jaundice are very numerous, but fortunately many are comparative rarities, and clinical diagnosis is commonly narrowed down to a few conditions of outstanding frequency and importance. The jaundice may be brought about by an obstruction inside the lumen of the bile-ducts, intrinsic changes in their walls, or external pressure upon the main ducts mechanically obstructing the outflow of bile.

(1) *Obstruction within the lumen.* There is only one condition of practical importance in this group, namely blocking of the common bile-duct by a gall-stone. This possibility has to be reviewed in every patient over middle age before any other cause need be considered, its incidence being of all such lesions by far the most common. Rare causes of obstruction from within include blockage of the common bile-duct by a round-worm or swallowed foreign body, or occasionally the direct spread along the lumen of a malignant tumour.

(2) *Obstruction originating within the walls of the ducts.* In this group are included the various types of cholangitis, wherein the inflammatory swelling and surrounding œdema of the walls of the bile-channels suffice to bring about an obstruction, either partial or complete. Such a cholangitis may be acute or chronic and of any grade of severity, either catarrhal or suppurative. Within this group is usually included the condition of "catarrhal jaundice," in the ætiology of which a catarrhal cholangitis, possibly ascending from the duodenum, is considered to play a contributory part, together with toxic changes in the liver cells. An uncommon cause of obstruction under this heading is cicatricial stenosis of the common bile-duct, which may

rarely follow the passage of a gall-stone, but is more often the result of a primary contracting scirrhus carcinoma of the duct, if the rare congenital stricture be excluded.

(3) *Jaundice resulting from external pressure upon the bile-ducts.* Compression of the bile-ducts from external pressure is a very common cause of obstructive jaundice, the site of pressure being either within the liver structure or at any point along their external course to the intestine. The compressing agent may thus be one of a large variety of conditions, which are considered in detail below.

If it is decided, on clinical and pathological grounds that have already been enumerated, that the jaundice in a given case is obstructive in origin, the possible causes of the obstruction are reviewed in their order of probability.

The two outstanding causes are *gall-stones* and *malignant disease*.

*Gall-stones.* The commonest cause of chronic obstructive jaundice in middle or later life is blocking of the common bile-duct by an impacted calculus. Jaundice which results merely from the passage of a stone through the bile-passages into the duodenum, although of the obstructive variety, is usually but slight in degree and of transient duration. Its occurrence indeed is not invariable, being noted in from 50 to 75 per cent of cases only, while the slightest degrees may be evident solely by an increase in the blood-bilirubin. It appears in from twelve hours to two or three days after the attack of biliary colic, and persists for a similar time; it is thought to result from a temporary traumatic or inflammatory oedema of the lining walls of the bile-ducts from abrasion by the calculus in its passage.

These temporary attacks of jaundice associated with cholelithiasis without other complications therefore present little difficulty in diagnosis. It is only when one is faced with a case of long-standing and persistent jaundice that the possibility of an impacted calculus has to be considered. In these circumstances the guiding points in diagnosis may be reviewed.

In most cases a history is forthcoming of recurrent attacks of biliary colic associated with vomiting, perhaps over a number of years, with or without subsequent transient attacks of jaundice. A history may be obtained of prolonged dyspepsia, either of the atonic flatulent type, with nausea and poor appetite, or suggestive of peptic ulcer. There may alternatively have been bouts of pain and tenderness in the right hypochondrium short of severe biliary colic.

Rarely there may be an interval up to several years between the

phase of biliary colic and the onset of obstruction by a calculus; jaundice may then develop insidiously and without pain, and closely simulate the onset of malignant disease.

In the typical picture of impaction, however, a characteristic syndrome occurs to which the term of "*intermittent hepatic fever*" is aptly applied. Variations in the clinical state coincide pathologically with a ball-valve action of the calculus, which from lying loosely in the distal portion of the dilated common bile-duct periodically descends and firmly plugs its lumen. A chronic cholangitis is thus set up with intermittent damming-up of the liver radicles with inspissated and infected bile. This pathological state of affairs is reflected clinically in bouts of pain, fever and jaundice. During an attack, pain is felt in the right hypochondrium and epigastrium, or referred to the back, of any grade up to the severest colic. Its onset is frequently accompanied by shivering or an actual rigor, with a rapid rise of temperature to 103° F. or more. Nausea and vomiting are usual, with marked prostration, while during and after an attack the liver may be enlarged and tender. Most cases show a slight permanent degree of jaundice, but soon after an attack its degree is usually intensified in proportion to the grade of obstruction, with appearance of bile in the urine and pale stools. In the interval between attacks, bile is not necessarily found in the urine. Deterioration of health is invariable, with rapid loss of weight as a result of digestive interference.

As a rule, enlargement of the gall-bladder cannot be made out, its walls being thickened and shrunken and adherent to surrounding structures, in contra-distinction to obstruction by malignant disease where, provided that its walls are healthy and it has not previously been the seat of gall-stones, it is commonly palpable as a tense elastic swelling (Courvoisier's law). The distinction is thus usual, but not absolute. If the condition remains unrelieved, a variety of sequels may occur.

(a) *Complete and permanent biliary obstruction*, shown by steadily deepening jaundice and progressive enlargement of the liver, while pain and fever usually become less marked.

(b) A superadded acute infection of the bile-passages or *acute suppurative cholangitis* extending into the liver substance, ending in empyema or gangrene of the gall-bladder and multiple abscess formation in the liver. The general state becomes one of marked gravity and toxæmia, with continued fever, leucocytosis, deep jaundice, a rapid decline in strength and fatal termination in a state of cholæmia.

(c) *Spread of acute inflammation to adjacent structures*. Infection does not necessarily remain confined to the biliary channels, but may



extend to the adjacent portal vein, producing a *suppurative pylephlebitis* which may similarly ascend to its finer intra-hepatic radicles. Symptoms are usually of more abrupt onset than with a suppurative cholangitis and are shown by severe abdominal pain and vomiting, with repeated rigors and sweats and development of a septicæmic or pyæmic state. Some increase of jaundice occurs, often slight, from obstruction and toxic damage. The liver is enlarged and tender and the abdomen rapidly becomes distended, while there is repeated vomiting and diarrhœa, often with hæmatemesis and the passage of bloody stools. Progressive weakness and prostration precede a final state of stupor and coma.

(d) Gradual development of *cicatricial stenosis of the bile-ducts*, a rare sequel, with slowly but steadily increasing jaundice and a final state of practically complete biliary stasis.

(e) Impaction of a gall-stone in the ampulla of Vater may bring about a simultaneous obstruction and dilatation of the main pancreatic duct, with an *acute or chronic pancreatitis*, having its characteristic train of clinical symptoms. The cutting-off of pancreatic juice leads to creatorrhœa and steatorrhœa with passage in the stools of undigested muscle-fibres and fat respectively. The islets of Langerhans do not usually become involved in the ensuing gland atrophy and diabetes therefore does not follow.

In the elucidation of any case where cholelithiasis or one of its complications is suspected, the greatest help is obtained from *X-ray examination*, discussed fully in a further section (see page 1535). Only in about half the total cases will a straight X-ray picture of the abdomen yield conclusive evidence of calculi, but the percentage is increased to the region of 80-90 per cent by the preliminary administration, intravenously or preferably by mouth, of an opaque substance such as sodium tetra-iodo-phenolphthalein or the corresponding bromine compound, which in health is excreted and concentrated in the bile of the gall-bladder. Where jaundice is intermittent, examination by cholecystography between attacks is of great value. Evidence of gall-bladder abnormality will be based upon a failure of the gall-bladder through defective concentration of dye to cast an X-ray shadow of the normal degree of density or within the normal time, upon shrinkage in size or irregularities in outline, defective power of contraction or the presence of gall-stones shown as negative shadows against a background of dye.

In the presence, however, of a persistent obstructive jaundice, cholecystography usually fails to give additional evidence, since further

bile, and with it the dye, can no longer be excreted through blocked channels into the gall-bladder. For the same reason the giving of dye may in such cases be unsafe and inadvisable, and reliance has therefore to be placed on a simple X-ray picture.

Apart from gall-stones, the only other cause of obstructive jaundice at all common is *malignant disease*. In its more distal course the common bile-duct is intimately related to the pancreas, through whose substance it has to pass before entering the duodenum. Compression within a *scirrhous carcinoma of the head of the pancreas* is the commonest cause of a painless, ingravescent jaundice in middle and later life. This intractable disease is commoner in the male subject and usually of later onset than calculus obstruction. Its characteristic features are the gradual onset of jaundice of obstructive type which progresses relentlessly and without intermission to an extreme degree of intensity. There may be a sense of weight or discomfort in the epigastrium, but colic or definite pain is in most cases absent. Rapid and progressive wasting occurs. Together with the deep jaundice, the liver shows a moderate smooth firm enlargement, the gall-bladder being distended with bile and usually readily palpable as a tense, insensitive, globular cystic swelling below the liver edge. Sometimes pancreatic carcinoma may be suspected from a firm fixed resistance or swelling lying transversely deep in the abdomen across the epigastrium. More often the growth cannot be felt. Changes are found in the stools indicating pancreatic as well as biliary obstruction. In chronic pancreatic lesions the urinary diastase content, normally 10 to 30 units, may be much reduced. Loewe's adrenalin mydriasis test may also be positive. Rarely a *primary carcinoma of the duodenum or of the common bile-duct itself*, usually originating at or near the ampulla of Vater, may be the cause of obstruction, but no exact diagnosis can be made on clinical grounds since the condition exactly resembles a pancreatic carcinoma and is distinguishable only at operation or post-mortem. Occasionally a *cyst or gumma of the pancreas* may act in the same way—here again the distinction is difficult apart from recognition of the swelling, if large enough, from its special characteristics. Sometimes an innocent fibrosis resulting from a *chronic pancreatitis* rather than growth may be the cause of pressure, with equal difficulty of diagnosis.

Chronic obstructive jaundice occurs not infrequently from *pressure upon the hepatic or common bile ducts in the portal fissure by enlarged lymphatic glands* in this region, usually malignant, especially secondary to a gastric neoplasm, very rarely lymphadenomatous, leukæmic or tuberculous. Such an event occurs commonly as a late manifestation

of the illness, while the age, sex, history and physical examination will reveal evidence of the primary abdominal lesion, e.g. a carcinoma of the stomach. Moreover, pressure on the hiliary passages in this region is almost invariably accompanied by ascites due to the simultaneous compression of the portal vein. An organ other than the stomach may be the site of the primary lesion, although the latter association is outstandingly common. Again, compression may be brought about by involvement of the ducts in a generalised secondary *carcinomatosis of the peritoneum*. The ducts may also uncommonly be involved at this point in an innocent *chronic perihepatitis*, with gross fibrinous thickening of the capsule, which with organisation and subsequent contraction of fibrous tissue grips the ducts in a ring of cicatricial stenosis or kinks them in their course. Local peritoneal *adhesions* around the gall-bladder or duodenum may rarely produce a similar obstruction. Any cause in this region, however, other than malignancy is unusual. Occasionally the pressure of a tumour of the right kidney or suprarenal may be responsible.

*Secondary malignant deposits* within the liver substance, again usually carcinomatous in nature, are common causes of obstructive jaundice by the local pressure they exert during growth upon adjacent intra-hepatic tributaries of the bile-ducts. Jaundice, however, is not constant, and may be of only moderate degree, since bile still enters the intestine from unobstructed ducts. Where there is pressure upon the main hepatic ducts in the region of the liver hilum, the outflow of bile is, however, completely shut off and clay-coloured stools result. Obstruction, moreover, is not the sole factor in the production of jaundice, which depends also upon the coincident damage to liver tissue and excessive hæmolysis. The Van den Bergh response in such cases is therefore often hiphasic in type, while the stools may be normally coloured with bile-pigment.

Diagnosis will depend upon careful attention to the history, the detection by the usual methods of the primary growth, and a correct interpretation from the physical signs of the nature of the hepatic enlargement. When due to secondary deposits the liver usually shows considerable enlargement, with a firm margin upon which masses of neoplasm may perhaps be detected. Ascites is frequent, and preliminary tapping may be needed before diagnostic features are evident.

*Gummatous disease* of the liver may similarly produce jaundice of obstructive or mixed type, and may be shown by a markedly coarse nodular irregularity of the liver surface, or by the detection of a large single rounded tumour, usually upon the exposed anterior surface.

The organ is seldom much enlarged and more often shrunken. History, concurrent signs of syphilis, blood Wassermann reaction, and frequently the response to specific treatment by iodides, will aid the diagnosis.

*Hydatid cystic disease* of the liver may also simulate malignant disease; jaundice is unusual, but may occur. Here, in addition to physical signs, X-ray examination of the abdomen, the presence of eosinophilia, a positive skin reaction and complement fixation test are all helpful in diagnosis. Amœbic abscess may similarly be occasionally accompanied by jaundice.

*Cirrhosis* of the liver is not characteristically accompanied by jaundice except in the rare type of Hanot's hypertrophic biliary cirrhosis, which is made evident by a deep obstructive jaundice in a young subject and a marked firm uniform enlargement of the liver and spleen without ascites or evidence of portal vein obstruction. The condition is characterised by febrile exacerbations accompanied by an increase of jaundice, abdominal pain and swelling of the liver and a slowly progressive downward course towards a fatal termination.

In the *common portal, multilobular, or alcoholic cirrhosis*, jaundice is not a general feature, being usually confined to the late stage of hepatic failure, when it is an expression mainly of toxic degeneration of liver cells, the blood-serum showing a biphasic or indirect Van den Bergh reaction. In the earlier stages the sallow muddy discoloration of the skin and conjunctiva can with care be distinguished from a true simple jaundice, while the harshness and dryness of the skin and general bloated appearance reveal the diagnosis, confirmed by evidence of commencing portal obstruction. Slight transient jaundice may, however, occur in attacks during the progress of the disease, which are usually unaccompanied by bile-pigment in the urine, although again urobilin may be in excess.

In *hæmochromatosis* there is a deep bronzing of the skin, together with glycosuria and cirrhosis of the liver. The staining in this disease is due not to bile-pigment but to a deposit in the tissues of hæmosiderin. The diagnosis is made conclusive only by removal of a portion of skin for microscopic section and examination by special staining methods.

## II. HÆMOLYTIC JAUNDICE

Jaundice of this variety is symptomatic of a clear-cut clinical group of cases in which, as the result of excessive blood destruction, there is

an accumulation in the blood stream of bilirubin without a corresponding retention of bile-salts, the jaundice being thus dissociated in type and usually mild in degree. As already noted, the excess of bilirubin present is of a chemical form, the renal threshold of which is high, and none therefore leaks through into the urine, while the blood-serum gives an indirect Van der Bergh reaction only.

This form is characteristically found in *acholuric family jaundice*, both of the congenital and acquired types, which are described fully in the section on surgical diseases of the spleen. Here the underlying pathological feature of the disease is an increased fragility of the red blood-corpuscles, hæmolysis of which occurs with greater readiness than normal in hypotonic saline solutions. Associated with this increase of fragility is the appearance of an abnormal proportion of reticulocytes or immature red cells in the circulating blood, representing an attempt on the part of the bone-marrow to compensate for the excessive destruction. The primary causal factor of the disease is not clearly defined, but probably lies in an excessive phagocytic activity for red blood cells on the part of the cells of the reticulo-endothelial system, centred in the spleen, which shows a corresponding and frequently marked enlargement. In the more common congenital or familial form jaundice is evident from birth or in early infancy. The acquired variety usually begins in early adult life. In both types when fully developed a mild degree of jaundice with some degree of secondary anaemia is commonly present throughout life. The general health may not be greatly disturbed. Enlargement of the spleen is constant, and, as noted, may be very marked. Bile-salts and bile-pigment are absent from the urine, which is, however, frequently of a deep amber colour from excess of urobilin. The stools are normally or deeply pigmented with bile. As already described the Van der Bergh reaction shows a negative direct and a well-marked positive indirect reaction.

In each form of the disease characteristic exacerbations occur, shown by an increase of jaundice and anaemia, with intensification of blood destruction and rapid enlargement of the spleen, which may be painful and tender.

The disease is of surgical significance from two aspects, firstly its cure by splenectomy, although the red cells remain unduly fragile, and secondly the great liability to subsequent cholelithiasis with pigment stones and their usual complications.

Other diseases in which a hæmolytic jaundice is seen include some cases of *pernicious anaemia*, *icterus neonatorum* (a temporary physiological breaking-down of unwanted red cells), and clinical states of

*excessive blood destruction*, for example in conditions of septicæmia, after the transfusion of an erroneous blood-group, and in blood destruction of parasitic or chemical origin.

### III. TOXIC AND INFECTIVE HEPATIC JAUNDICE

Within the confines of this group are included a large number of conditions in many of which the exact ætiology and pathology are obscure, but in which as a basis for discussion jaundice is considered to result largely from a toxic damage and degeneration of the polygonal liver cells, combined with blockage of interjacent bile-capillaries and obstruction of the outflow of bile from consequent œdema and swelling of their walls. Jaundice then follows from retention in the blood-plasma of both types of bilirubin, and a biphasic response is obtained to the Van den Bergh test. It is not surprising, therefore, that in this mixed group the greatest confusion and difficulty may arise in differentiation and diagnosis, seeing that the clinical features also combine the phenomena of obstruction and infection or hepatitis. Jaundice, usually slight or moderate, but of any possible grade of severity, is present, while according to the degree of obstruction a variable amount of bile-pigment is present in the urine and stools.

The form most commonly met with is the clinical syndrome known as *Catarrhal Jaundice*. This is seen most often in adolescence or early adult life, most cases occurring under the age of thirty, in itself a helpful although inconclusive point of diagnosis from gall-stones. The majority of cases of jaundice in young people in this country are of this nature. Owing to its usual mild course, with spontaneous recovery, the exact morbid histological features are elusive, but whereas an inflammatory duodenitis was formerly held responsible, with ascending catarrh of the common bile-duct, producing swelling of the mucosa and obstruction of the lumen, it is now considered to be due rather to a primary infective hepatitis and cholangitis, of doubtful ætiology, running usually a benign course with complete resolution. To some extent it is certainly infective, seeing that small groups of cases often occur together without other obvious association in common. Although the normal course is benign, a very small proportion of cases suddenly develop the severe features of acute necrosis or icterus gravis and terminate fatally. Caution in prognosis is therefore always wise. In the early stages the Van den Bergh test may in some cases give a direct positive result indicating biliary obstruction, but after the first few days the result is

commonly biphasic signifying liver damage also. In any given case of jaundice, therefore, undue reliance must not be placed upon a single blood examination, but to avoid erroneous conclusions this should be repeated at intervals. Quantitative estimation of bilirubin as already described, together with estimation of the icterus index, affords a valuable measure of progress.

Onset is usually with vague gastro-intestinal symptoms, epigastric discomfort, anorexia, furred tongue, nausea and perhaps vomiting. Constipation and flatulent distension of the abdomen are the rule, but definite abdominal pain is absent. There is much mental depression and malaise with mild pyrexia. After an interval up to a few days in duration, a light yellow jaundice appears involving the conjunctiva, and rapidly deepening to a canary yellow. As the jaundice becomes more definite, so gastric symptoms usually subside in intensity. Itching of the skin may be marked, and precede the observation of jaundice. The urine is deeply bile-stained, and in the early stages contains abundant bile-salts, although these tend within a few days to disappear with a relief of pruritus and bradycardia. Rarely glycosuria may occur. The stools are pale and offensive, and, although to the naked eye free from bile at the onset, later it is clear that there is a reduction in the amount only and not complete absence. Chemical tests will show the presence of at least a small amount of bile-pigment throughout. The offensive character of the stools is due largely to bacterial decomposition and to the presence of an excess of fat, a high percentage of which has been split by the pancreatic enzymes into fatty acids, but remains unabsorbed owing to the lack of bile.

On physical examination a slight uniform enlargement of the liver is commonly evident, with tenderness on pressure in the epigastrium; the gall-bladder cannot usually be identified, while the spleen is not enlarged. It is a point of note that although there may be diffuse tenderness over the upper abdomen it shows no special predilection for the gall-bladder area.

After rapidly reaching a maximum intensity, the jaundice and its associated symptoms usually persist for two to four weeks and then gradually lessen and subside. Permanent liver damage is rare. As mentioned, very rarely a case of apparent catarrhal jaundice will proceed to a widespread acute toxic degeneration of the liver cells and death, but generally speaking the prognosis is good. The usually accepted upper limit for the duration of catarrhal jaundice is six weeks—if after this period the jaundice persists unabated in intensity, it is highly probable that the diagnosis is in error and a further cause should be sought.

The differential diagnosis constitutes one of the most important and often difficult features. The helpfulness of the patient's age in considering probabilities has been mentioned. Differential diagnosis has to be made from :

(a) Other causes of infective and toxic hepatitis (given below).

(b) A purely obstructive jaundice, which at the beginning it closely resembles, especially from calculus and growth, e.g. in the liver, pancreas or lymphatic glands. Considering this group first, the main points of help are the limited duration and signs of early abatement of the jaundice, and absence of biliary colic or of previous dyspeptic history. A waxing and waning in the intensity of the jaundice, or its disappearance and return, suggests gall-stones and not catarrhal jaundice. The hue, moreover, in catarrhal jaundice is always yellow, never green, which in an elderly patient suggests growth. Although a considerable loss of weight may take place during the course of catarrhal jaundice, the previous nutrition is normal. No evidence of a primary neoplasm can be found. At the onset the grade of obstruction may appear to be complete, but later it rapidly becomes evident that a partial blockage of bile only is present. Duodenal intubation confirms this point, showing that biliary obstruction is only marked in the early stage, and that later the outflow of bile may even be above normal. The value of X-rays and of blood examination has already been mentioned. The levulose or galactose tolerance tests, described later, will show by a marked rise of blood-sugar a temporary liver insufficiency in catarrhal jaundice, not met with in obstructive jaundice due to calculus or growth.

Considering the remaining group, a large number of poisons, bacterial or chemical, will produce jaundice of toxic type by their action on the liver. Sporadic cases are met of *infective jaundice* of unknown aetiology, yet differing from catarrhal jaundice as described above in their relative absence of abdominal symptoms. After two or three days of fever and malaise, jaundice suddenly appears and within a like period has again cleared. Known specific diseases in which severe infective jaundice is the main feature include "epidemic" or spirochaetal jaundice and yellow fever, while a milder degree is a frequent complication of typhoid fever, relapsing fever, pneumonia, septicæmia, pyæmia, or indeed of any infective illness of sufficient severity. *Eclampsia* of pregnancy comes within the same category. Jaundice may occur in *syphilis* as a symptom of the disease, or as a result of treatment, and this has nowadays become an important cause. In any case of jaundice of obscure aetiology, a blood Wassermann reaction



should always be done. If necessary, leading enquiry should be made of infection or treatment, and an inspection of the antecubital regions—and, if possible, of the buttocks—made for puncture marks. In the acquired disease, an infective hepatitis may rarely be met in the secondary stage, but a much commoner occurrence is a toxic damage to the liver from drugs of the arseno-benzol group being used in treatment. This is usually found to come on from two to three months after the end of a course of injections. Symptoms closely resemble those of catarrhal jaundice except that preceding gastric symptoms are less in evidence, while enlargement and tenderness of the liver may be more marked. Albuminuria usually precedes the onset of jaundice and should be tested for as a routine; of greater help is the presence in the urine of an excess of urobilinogen in cases liable to be followed by jaundice. A Van den Bergh reaction may similarly reveal latent jaundice. Precautions against the development of jaundice in a patient undergoing treatment for syphilis include attention to general health, administration of cane sugar or glucose before injections, the avoidance of too high doses (above 0.6 gramine N.A.B. for an adult male), or of too prolonged a course without interval for rest (i.e. more than eight consecutive weekly injections). Delay in injection of N.A.B. after preparation of its solution, thus allowing time for development of toxic products by oxidation, is by some considered an important factor. When dissolved it should therefore be given at once.

The course of salvarsan jaundice is in the majority of cases satisfactory, with recovery. Subsequently, arsenic should either be avoided in treatment, or given with the greatest caution. Rarely the condition may progress to one of acute yellow atrophy and even prove fatal.

Jaundice is similarly met with in the congenital form of syphilis, where it is of serious omen, associated with widespread spirochætal invasion and degeneration of the liver, and usually ends fatally.

The giving of other *heavy metals* may produce jaundice, e.g. bismuth, and rarely, organic gold salts; inorganic as well as organic arsenic may be a cause. Phosphorus poisoning is now only of historic interest. *Chloroform inhalation* in the susceptible subject may produce jaundice as a symptom of "delayed chloroform poisoning." Other *chemical poisons* causing a similar liver damage and jaundice include arseniureted hydrogen, tetrachlorethane, trinitrotolnol, atophan, snake venom and mushroom toxin, among many others. The general features are similar throughout, and in any instance the jaundice may be accompanied by a variable grade of constitutional disturbance up to the picture of complete liver failure. In any case of jaundice where the

ætiology remains in doubt after ordinary examination, enquiry should be made as to drugs taken, having in mind the possibility of such poisoning. In some instances the jaundice results from undue susceptibility or idiosyncrasy on the part of the patient rather than from any faulty technique in the administration of the drug.

### DIFFERENTIAL DIAGNOSIS

In a case presenting as its main clinical feature the symptom of jaundice, an accurate diagnosis of its cause is clearly an essential preliminary to treatment. The important point to decide from the surgical point of view is whether the jaundice results from a lesion capable of relief by operative measures, or whether it is due to a "medical" cause, where surgery is not only of no avail, but possibly harmful. Differential diagnosis has already been largely considered at appropriate points in the discussion of the several causes of jaundice, but for convenience a summary of the main points may here be given.

Considerable help as to the probable nature of the jaundice in a given case will be obtained in the first place by a careful and complete *history taking*. In every instance of jaundice, great importance is to be attached to an exhaustive analysis of *gastro-intestinal symptoms*. Enquiry is made as to appetite, the presence or previous occurrence of nausea, vomiting, constipation or diarrhoea, hæmatemesis or melena, or of any naked-eye abnormality that may have been noted in the appearance of the stools or urine. Special attention is paid to the occurrence of *abdominal pain*, noting its situation, character, time of onset and relation to jaundice. As regards the *jaundice* itself, points of importance are its duration, distribution and intensity. Any variability or fluctuation in its depth, or an actual intermittency of the jaundice, is of the greatest possible significance. The occurrence of *fever* or rigors and their relation to other symptoms should be enquired for. Other points of importance in the history are the age of the patient, occupation, possibility of pregnancy or exposure to known toxic substances, and complaint of wasting or general illness. A family history of jaundice may be significant.

*Physical examination* will often confirm a tentative opinion based upon the history. Special attention is, of course, paid to abdominal examination, for possible enlargement of liver or gall-bladder, noting the characters of each, and any local tenderness or rigidity. The presence of splenomegaly, ascites or a tumour elsewhere in the abdomen

or pelvis will be searched for. Rectal examination will, of course, never be omitted.

The general indications, results, and limitations of *X-ray investigations* have been discussed under their appropriate subjects. In addition to X-ray of the gall-bladder, a barium meal and "follow-through" examination may reveal a primary lesion in the stomach or colon, or may show a deformity of the duodenum from the outward pressure of an enlarged or displaced gall-bladder.

On the *pathological* side, a routine chemical and microscopical examination of the *stools* is essential, including examination for and estimation of unabsorbed fat, split and unsplit, undigested muscle-fibres, bile-pigment, and occult blood, as appears indicated by the merits of the case and the naked-eye appearance of the stools. A similar examination is made of the *urine* for bile-pigment and bile-salts and for excess of urobilin. As a means of direct examination of the duodenal contents as a test for the patency and contractile power of the gall-bladder and biliary passages, *duodenal intubation* may be employed and the bile, if any, withdrawn and examined for the presence of pus cells, cholesterol and micro-organisms, as described in detail elsewhere. Description of the information to be obtained from *blood analysis* has been fully considered at the beginning of this article.

### HEPATIC FAILURE—"CHOLÆMIA"

Whenever the physiological efficiency of the liver becomes so seriously impaired, whether through stagnation by chronic obstruction of its ducts or from severity of damage by disease or chemical poisoning, that it finally becomes inadequate to sustain the metabolic needs of life, a train of symptoms ensues collectively known as "*Cholæmia*," analogous to the uræmia of renal failure. It is thought to be due to a final loss of the detoxifying function of the liver cells for amino-acids. It is a danger which is always present if chronic obstructive jaundice remains unrelieved, there being then usually a sudden acute febrile termination. Surgical interference may, therefore, be directly indicated, where feasible, if only to relieve the obstruction and obviate the risk of cholæmia.

The onset may, according to the precipitating cause, be either acute or insidious, but when established the condition is rapidly terminal. It is usually heralded by malaise, headache, restlessness, delirium, muscular pains and twitching or convulsions, and drowsiness deepening

into coma. There is intense prostration, with fever and general illness, frequent pulse, dry tongue, and the rapid development of a "typhoid state" with subsultus tendinum and paralyses, ending fatally within a few days or less. Jaundice appears or, if already present, increases in intensity. In very acute cases it may be but slight, there being insufficient time before death for its full development. Hæmorrhages occur in the skin and from mucous membranes. Hyperpyrexia may be present before death. Intractable vomiting with hæmatemesis is a prominent feature. The bowels are constipated or melæna may be present. The urinary output is diminished; it is bile-stained, with bile-acids, albumen in large quantities and casts. The percentage of ammonia-nitrogen is greatly increased and the urea excretion low. Leucine and tyrosine crystals may be found in the deposit, but are not invariable, and are in no way diagnostic, undue importance having been attached to their presence.

As regards direct post-operative association, liver failure is apt to ensue in a susceptible subject from the culminating effects of anaesthesia, trauma, shock, hæmorrhage, and absorption, all combining to overcome an inadequate reserve of function. Most so-called "liver-deaths" after operation are shown by the rapid development of nervous symptoms and coma, with steadily rising pyrexia, ending fatally in 24 to 48 hours. A delayed form may occur in which all hepatic symptoms remain latent for about four or five days, when a gradual transition ensues into the above state. In a third type, marked renal damage progressing to complete anuria may accompany the symptoms of cholæmia or even appear to dominate the clinical picture.

The liver in most cases terminating in chokemia becomes reduced in size, of soft flabby consistence with wrinkled capsule, shown clinically by a diminution or disappearance of liver dullness. The pathological change is one of widespread fatty degeneration with areas of necrosis and hæmorrhage, to which the term acute atrophy or acute necrosis is applied.

In young subjects, especially in children, the liver possesses to a remarkable degree the power of regeneration and repair, and where the infective or toxic process has not been of sufficient intensity to cause death, the liver may recover to a state of functional efficiency by a process of nodular hyperplasia, the condition then being known as a sub-acute necrosis.

*Diagnosis of Cholæmia.* In the more insidious types the early symptoms are indefinite, and the condition may be mistaken at first

for a catarrhal jaundice, from which there is little to identify it. The onset, however, of fever, persistent vomiting and nervous symptoms, with petechial and other hæmorrhages, is of the gravest significance and generally presages a fatal issue in a few days. Treatment is at this stage of no avail—reliance must be placed wherever possible on prophylactic measures as considered later.

### LIVER FUNCTION TESTS

An accurate measure of hepatic reserve would, if it were available, be of the greatest value before the contemplation of surgical procedures, as an index of urgency or safety of operation. Unfortunately, in spite of a multitude of tests, there is none which could with any accuracy be so described, although from a limited number a rough guide may be obtained. The position with regard to hepatic efficiency tests is, in fact, considerably less satisfactory than with corresponding tests of renal function. A liver may be riddled with carcinoma and yet show little or no defect in response to such tests.

The information to be obtained from examination of the blood, urine, and stools for bile-pigments, and of the urine for bile-salts, has already been detailed, including the *Van den Bergh reaction*, *Fouchet's test*, and estimation of the *Icterus Index*. It may be mentioned also that chemical tests for the presence of an excess of urobilinogen or urobilin in the urine are sometimes of value since these substances may be found for some days before skin pigmentation is recognisable, during the development, for example, of a catarrhal jaundice.

One of the chief metabolic functions of the liver concerned with nitrogen metabolism is to convert the ammonia resulting from the breakdown of amino-acids into the non-toxic form of urea. This function has been used as an estimation of liver efficiency as expressed by the relative proportion of urea-nitrogen to total nitrogen in the blood or urine respectively. This ratio in the urine, or *urinary nitrogen-coefficient* ( $\frac{\text{urea nitrogen}}{\text{total nitrogen}}$ ), is in health between 80 and 90 per cent, whereas in severe hepatic disease with metabolic failure it is stated to fall below 50 per cent. Similarly, in severe liver damage the *ammonia-coefficient* of the urine, or ratio in the urine of ammonia-nitrogen to total nitrogen, becomes correspondingly raised parallel with the decreased excretion of nitrogen as urea, the normal figure of 5 per cent being raised to 15 or 20 per cent, an increase responsible for the

production in chokæmic states of symptoms of acidosis and air-hunger. It must be made clear that only in the presence of the grossest insufficiency of the liver are positive results obtained from the above tests.

As an estimation of hepatic reserve more help is obtained from a test of the storage power of the liver for certain carbohydrates, namely, lævulose and galactose, under the terms respectively of *Lævulose Tolerance* and *Galactose Tolerance Tests*.

*The Lævulose Tolerance Test* is carried out by giving by mouth 100 grammes of lævulose (dissolved in water), and estimating in the ordinary way the total blood sugar (i.e. combined glucose and lævulose content) in specimens of blood collected before, and at  $\frac{1}{2}$ , 1,  $1\frac{1}{2}$  and 2 hours after, giving the lævulose. Specimens of urine are also collected at the same intervals and examined for sugar.

In the presence of liver damage above a certain threshold, the power of absorption and storage capacity of the liver for lævulose is diminished, and lævulose appears in the urine about one hour after ingestion, while there is a corresponding abnormal rise of blood sugar. It is stated by Strauss (4) that a rise of blood sugar above 140 mgms. per 100 cc., or an increase above the resting level of over 30 mgms., indicates a degree of hepatic insufficiency.

*The Galactose Tolerance Test* is carried out on similar lines. It is stated that in health the giving of 40 grammes of galactose should not be followed by its appearance in the urine, or by any appreciable rise in blood sugar. In states of damage or destruction of the actual liver parenchyma, as for example in catarrhal jaundice, toxic hepatitis or neoplasm, galactosuria and hyperglycæmia result. In pure obstructive or hæmolytic jaundice, on the other hand, a negative response is obtained. The test is of greater delicacy than the corresponding lævulose test, and is of definite diagnostic and prognostic value.

A large number of other tests have been described which are said to indicate derangement of liver function, depending upon the power of the liver to eliminate foreign substances, apart from those employed on account of their opacity to X-ray examination. One such test introduced by Rowntree, Hurwitz, and Bloomfield (5) depends upon the use of *phenoltetrachlorophthalein*, which when injected is excreted exclusively by the biliary system. The required amount of the drug is injected intravenously in solution, after a preliminary purging of the patient, and the feces collected for the ensuing 48 hours. By colorimetric methods the percentage of the total dye excreted via the bile

during this time is determined. It is considered that a minimum proportion of 30 per cent should be recovered from the stools, a lower percentage indicating pathological liver damage. Several modifications of technique have been devised, either by direct analysis of the excreted bile obtained by a duodenal tube, or by estimating the rate of disappearance of the dye from the blood in a standard time. In the normal subject almost the whole of the dye should be excreted from the blood within an hour, whereas in liver damage a considerable proportion remains. Bromsulphthalein is another dye similarly employed as a test for liver capacity.

*Cholecystography* as a method of investigation of liver and biliary function has already been noted, and is fully described under the appropriate section. Its use is limited to conditions of patency of the gall-bladder and bile-ducts, for if definite obstruction exists the dye cannot be excreted and its administration may be unsafe.

The value in the diagnosis of infective conditions of the biliary passages of *duodenal intubation* and direct examination of its contents is discussed in a separate section. By its means the presence and degree of biliary obstruction or infection may be confirmed, and the nature of the invading organism, if any, determined. As a means of drainage of infected bile, i.e. as a direct cholangogue, it affords also a helpful line of medical treatment in chronic catarrhal cholangitis or cholecystitis.

Of indirect but of great surgical importance in cases of jaundice is estimation of the *blood-coagulation time*, usually carried out by the lead-shot and capillary tube method. It has been mentioned that in the presence of jaundice this may be prolonged up to 20 minutes, the normal time being  $2\frac{1}{2}$ – $3\frac{1}{2}$  minutes. Methods of diminution of coagulation time are considered under the medical treatment of jaundice. The value of a complete blood count, and in some cases estimation of the fragility of the red cells, in differentiating certain forms of hæmolytic jaundice may again be mentioned.

### THE MEDICAL TREATMENT OF JAUNDICE

The indications for surgery and descriptions of operative technique in cases of jaundice of appropriate type are dealt with at length on page 627. It will suffice at this point to emphasise that, in view of the cumulative liver damage which ensues, chronic obstructive jaundice should be relieved wherever possible, by surgical means if

necessary, the precise measures to be taken depending upon the cause in question.

Here will be considered, therefore, only those non-operative measures applicable to cases of jaundice which are either from their nature not amenable to surgery, or in which the general state of illness is too advanced for operation to be safely contemplated. These patients may to some extent be helped by palliative measures directed at relief of symptoms. Precautions whereby operative risk in the presence of jaundice is lessened are also considered.

The jaundiced patient should remain at rest in bed, and attention in medical treatment given primarily to the condition of the gastrointestinal tract, aiming at the relief as far as possible of biliary obstruction, and the promotion and dilution of the flow of bile into the intestine.

Of fundamental importance is the question of *diet*. The absence of entry of bile into the intestine leads primarily to a defective absorption of fat, and secondarily to flatulent distension of the gut and putrefactive changes in its contents, owing to lack of the normal antiseptic action and peristaltic stimulus which the bile exerts. Interference results to a lesser degree with the digestion and absorption of other food substances. Carbohydrates, however, are the most easily absorbed and are also of great value in supporting the metabolic processes in the liver. A simple diet is therefore indicated, with abundant bland fluids and sugar. Where there is much nausea and retching, as in the early stages of catarrhal jaundice, solids are not tolerated and the diet must therefore be fluid in character. By some a milk-basis is advocated, but owing to its high fat-content it is not ideal and often causes nausea. Milk may, however, be given in small amount, e.g. not more than  $\frac{1}{2}$  to 1 pint in the 24 hours, in skimmed form (removing the upper third from natural milk which has been allowed to stand), and may if desired be citrated, peptonised or diluted with soda-water. Whey, barley-water, clear soups, meat extracts, fruit drinks and lemonade are all suitable, the latter with added sugar, together with abundant water to drink. Later, with improving appetite, dry, easily digested semi-solids may be given in frequent small meals, such as Benger's, Plasmon, milk puddings, white of egg, jellies, toast, rusks, steamed fish, minced meat. Fats above all are to be avoided, and foods rich in cholesterol, such as egg yolk, liver, kidney, sweetbread, tripe, and brains, for in cases of cholelithiasis these raise the cholesterol-content of the blood and tend to further deposition of calculi.



The following scheme may be taken as an example of a fat-free diet for jaundiced patients at this later stage :

## FAT-FREE DIET FOR JAUNDICE

*Breakfast.*

Bread and jam.	Weak tea or coffee (as much as desired).
Fruit.	Sugar (as much as desired).

*Lunch.*

Water biscuits or rusks.	Weak tea or coffee.
Lettuce, tomato, etc.	Sugar.

*Dinner.*

Lean meat or rabbit.	Water.
Chicken or white fish.	
Bread and vegetables.	
Jelly or stewed fruit.	

*Tea.*

Bread and jam.	Weak tea or coffee.
Fruit or lettuce, etc.	Sugar.

*Supper.*

Bread or water biscuits.	Bovril or clear soup or chicken or veal
Stewed fruit.	broth.
Jelly or boiled rice.	

*Hourly between Meals.* Lemonade, 150-200 cc., with glucose or sugar, 1-2 dr.

If permitted, just enough milk may be given to make the tea palatable, and porridge may be added to the breakfast, with not more than 2 oz. of milk, so that the day's intake does not exceed  $\frac{1}{4}$  pint.

As the jaundice subsides in intensity, and bile-pigment is again found in the fæces, the dietary restrictions may be cautiously relaxed, and the patient allowed to get up.

Attention to the bowels is of no less importance than diet. At the

onset of jaundice, calomel in small doses of  $\frac{1}{4}$ – $\frac{1}{2}$  gr., repeated hourly for 4 to 6 doses, has an action as a cholagogue and antiseptic without causing violent purgation, and should always be followed 12 hours later by a saline purge to prevent continued irritation. Magnesium sulphate 1–2 dr., given in concentrated solution on an empty stomach each morning, causes a reflex contraction of the gall-bladder where this is mechanically possible, the effect of which is prolonged by making the patient lie upon his right side. Enemata are given as necessary to ensure a free emptying of the bowel. An ice-cold enema is stated to be of value in relieving skin irritation, and in lessening the intensity of the jaundice, by inducing a reflex relaxation of the biliary sphincter. Direct transduodenal drainage of bile through an Einhorn tube by Lyon's technique is a method of treatment which should be used much more freely, and is of definite value in jaundice associated with chronic cholecystitis or cholangitis and in catarrhal jaundice.

In the stage of gastric irritation, bismuth and alkalis are helpful in allaying nausea and vomiting, given in a mixture such as bismuthi salicylatis 15 grs., sod. bicarb. 15 grs., tinct. belladonnæ 5 m., acid. hydrocyan. dil. 3 m., tinct. aurantii 20 m. and aq. chloroformi to 1 oz. 4-hourly. If the jaundice is associated with cholecystitis or cholangitis, atropine sulphate  $\frac{1}{100}$  gr. and adrenalin HCl (1 in 1000 solution) 5 m. subcutaneously, repeated as necessary, will promote relaxation of spasm of the sphincter, while for the relief of pain morphia (or a milder analgesic) may be necessary, together with the local application of antiphlogistine, hot fomentations, or poultices to the gall-bladder region.

At a later stage *cholagogues and biliary antiseptics* may be given with advantage. Of the possible drugs with this action which may be employed, hexamine and sodium salicylate 20–30 grs. are of the most certain value, given with alkalis to dissolve mucus and belladonna to dilate the biliary passages, while calomel, pulv. elaterinæ co. or podophyllin may also usefully be given in repeated small doses. A contra-indication to the use of hexamine in some subjects is its production of renal irritation or hæmaturia, a risk which is minimised by giving it well diluted with water. Helmitol 10–15 grs. is a hexamine product less liable to cause such irritation. A good preparation combining an antiseptic, cholagogue and aperient action is available in salol. 10 grs. and pil. hydrarg. 1 gr., given together t.d.s. in a cachet. Bile-acids, the natural cholagogue, may themselves be given, e.g. as felamine (combining cholic acid and hexamine). Where, from chemical examination of the stools, blockage of the pancreatic duct also

is suspected, the giving with meals of a pancreatic extract by mouth is indicated.

In deep or long-standing jaundice intense and universal *irritation of the skin* is often the most distressing symptom of illness. Its radical treatment, wherever possible, lies in surgical relief of the obstruction, e.g. by cholecysto-gastrostomy or cholecysto-enterostomy. Where, however, this cannot be done, the itching is partly alleviated by giving alkalis by mouth, by hot alkaline baths, and local applications to the skin containing calamine with acid. carbol., menthol, tar or ichthyol. Thyroid extract,  $\frac{1}{2}$  gr. by mouth, is said to assist in lessening pruritus. Sedatives such as bromide and luminal are often needed.

Before any operation upon a jaundiced patient is contemplated, he should be placed upon a detoxifying lacto-vegetarian diet and a preparation of calcium should be given for a week or longer, in an attempt to reduce the *prolonged coagulation time* of the blood. Oral treatment is of no value, the drug being best given in the form of the gluconate, say 10 cc. of a 10 per cent solution being injected intravenously or intramuscularly on alternate days. Sodium citrate 30–45 grs. t.d.s. by mouth for the few days before operation also has the temporary effect of increasing the coagulability of the blood. Intramuscular injection either of whole blood or of normal horse serum or hæmostatic serum may also be made, while a preliminary blood-transfusion of about 250 cc. on the previous day may be advantageous. At operation every measure must be taken by prompt hæmostasis to minimise blood-loss from hæmorrhage. The least toxic anæsthetic is ether in conjunction with local anæsthesia. Great care must be taken to avoid giving cardiac depressants, since in chronic jaundice the cardiac reserve is often lowered through a toxic myocardial degeneration. The mortality of a given operation upon a jaundiced subject rises in direct proportion to the depth of jaundice present.

Jaundice due to a toxic hepatitis is treated on largely similar lines. Similar dietary precautions are required, restricting not only fats, but also proteins, in view of their added metabolic strain upon a damaged organ, and giving abundant glucose, while the patient is induced to drink the largest possible quantity of fluid. In heavy metal poisoning, e.g. arseno-henzol jaundice, a solution of sodium thiosulphate should be injected intravenously three times a week, in doses of 0.6 gramme in 10 cc. of distilled water, the toxic effects of the N.A.B. thereby being lessened—the proprietary preparation known as “Amctox” is a safe and reliable form. The bowels must be freely opened throughout with calomel or cascara and morning salines.

When severe symptoms of general intoxication ensue and a state of cholæmia is threatened, more active measures are needed. Venesection  $\frac{1}{2}$ –1 pint is of value in lowering the concentration of toxins in the blood, together with free administration of 5–10 per cent glucose-saline by oral, rectal or preferably intravenous route. The rapid absorption and storage of glucose is aided by the simultaneous administration of insulin 10–20 units subcutaneously. Diuretics such as diuretin 10 grs., caffeine citrate 5 grs. or theocin sod. acetate 5 grs. t.d.s. aid simultaneous elimination by the kidney. Alkalis should be given freely with the fluids by mouth to combat acidosis.

If a condition of acute necrosis or cholæmia does develop in spite of prophylactic measures, it can only be treated symptomatically by careful nursing and sedatives with cardiac and general stimulants. The prognosis is, however, invariably grave in spite of all treatment, most cases ending fatally within a few days.

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SECTION 8  
PANCREAS

CHAPTER I  
Pancreas  
by  
A. DICKSON WRIGHT

CHAPTER II  
Diabetes in Relation to Abdominal Surgery  
by  
R. SLEIGH JOHNSON

## SECTION 8

### PANCREAS

#### CHAPTER I

##### PANCREAS

by

A. DICKSON WRIGHT

*Acute Pancreatitis.* Inflammation in the pancreas seems to be more the result of release of the pancreatic enzymes among the tissues than of any bacterial invasion of the pancreas. Secondary bacterial infection with *bacillus coli* occurs later among the sloughs and exudates produced by the action of the pancreatic ferments.

*Ætiology.* A large percentage of cases occur in association with calculous disease of the bile passages. A few cases have occurred in lithiasis of the pancreatic ducts and as a result of invasion of the duct of Wirsung by an ascaris. Another special group of cases follows operations upon adjoining viscera. Thus, in gastrectomy a portion of the head of the pancreas may be injured by clamps or imprisoned in a suture or ligature during the closure of the duodenal stump. Again, the body of the pancreas may be injured in removing an adherent ulcer or a carcinoma of the stomach, and the tail of the pancreas may be injured by being included in the pedicle ligature in splenectomy or nephrectomy. The pancreas may be injured also by penetrating wounds of the abdomen, and tears of the pancreas have been reported after abdominal contusions.

The effect of the release of the pancreatic ferments is to digest the adjoining tissue, thus, if the hunt falls upon the pancreas itself necrosis results, if upon the blood-vessels hæmorrhage, resulting in two special types of pancreatitis, viz. *gangrenous* and *hæmorrhagic* pancreatitis (pancreatic apoplexy). The lipolytic enzyme acts upon the fat, starting in the root of the transverse mesocolon and spreading to all the fatty tissues of the abdomen and even to the pericardial and subcutaneous fat. The effect upon the fat is to produce scattered areas of fat necrosis; these are whitish areas of saponified fat which are indurated and can

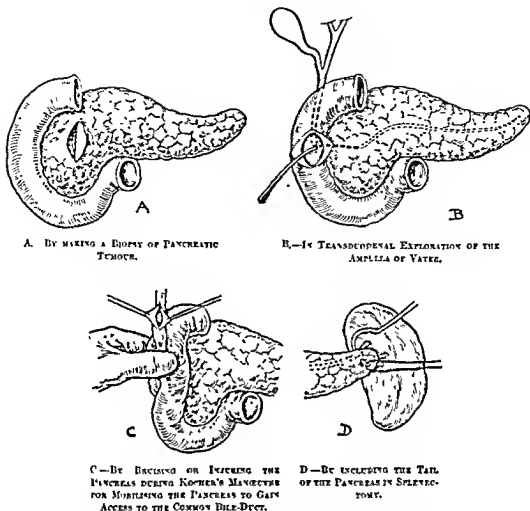


Fig 457.—METHODS BY WHICH PANCREATITIS MAY BE PRODUCED AT OPERATION.

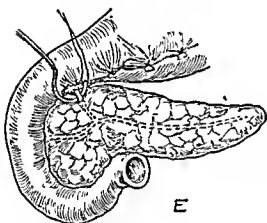
often be felt with the exploring hand in the abdomen before they are seen. The effect of the pancreatic inflammation produces a great outpouring of peritoneal fluid which is always blood-stained. If the pancreatitis is not dealt with at once and infection results in the damaged pancreatic tissues *suppurative pancreatitis* or *pancreatic abscess* is the result.

*Acute glassy edema of the pancreas* is an interesting condition described by Alexander of Montreal. It can be produced easily in the dog by injecting bile into the main pancreatic duct. Within a few minutes of injection the peripancreatic tissues are seen to undergo an amazing change, visibly filling with clear fluid until the pancreas appears to be imbedded in the most transparent of glass. The writer has operated upon one such case in the human.

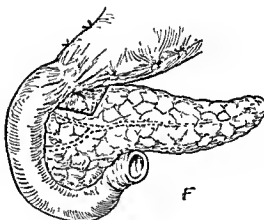
A woman of 41 who had a long history of gall-stone indigestion with

recurrent attacks of very severe pain was admitted to hospital in great epigastric pain of six hours' duration, and a diagnosis of acute pancreatitis was made. On opening the abdomen the root of the transverse mesocolon and the retroperitoneal tissues were infiltrated with crystal-clear œdema, and the pancreas appeared to lie at the bottom of a pool of fluid. A large number of small gall-stones were removed and the gall-bladder drained, while the pancreas was left untouched. A clue to the origin of the condition was discovered when it was noticed that on some days the drainage from the gall-bladder became clear and the surrounding skin became digested, indicating that the duct of Wirsung opened into the common bile-duct instead of into the duodenum. It was also noticed that on the days when pancreatic juice drained the patient was much more ill than when bile was draining. With the appropriate treatment for pancreatic fistula the patient soon got well.

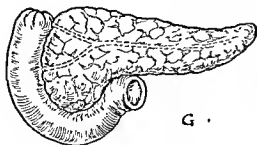
If the patient is not operated on in the glassy stage the œdematous fluid becomes cloudy from leucocytic infiltration, and enzymic action



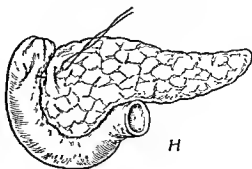
E.—INCLUSION OF THE DUCT OF SANTORINI IN GASTRECTOMY.



F.—BY REMOVING A PORTION OF THE PANCREAS FORMING THE BED OF AN ULCER.



G.—INCLUDING THE DUCT OF SANTORINI IN DUODENAL INVAGINATING STITCH.



H.—PICKING UP THE PANCREAS IN THE SAME MANNER.



and large masses of greyish jelly are seen; such cases have been described (Howard). Later on, the œdema may resolve or may pass on to the usual picture of pancreatitis. Probably in many cases several attacks of this œdema occur and resolve, and this may be an explanation of the exacerbations of pain which occur in cases of chronic pancreatitis.

The symptomatology of acute pancreatitis is fairly clear-cut. The onset is sudden, generally in a fat, florid patient, with a history suggestive of gall-stones. Alcoholism has been stated to be a predisposing influence. The pain is epigastric, and goes through to the back and between the shoulder blades, vomiting is a marked feature, and the expression "sounds like an acute pancreatitis" indicates the noisy type of retching produced. The upper abdomen is resistant, and full and tender rather than rigid. Signs of free peritoneal fluid may be present, and occasional bruising in the loin may be seen in late cases (Grey Turner's sign), and the writer has seen one case of bruising round the umbilicus (Cullen's sign). Cyanosis, general or confined to the abdomen, may be seen and is hard to explain. The temperature is not much raised, and the pulse-rate increases with the progress of the disease. The urine often contains sugar and much diastase, and the pupil of the eye is dilated by adrenalin (Loewi's test). Slight jaundice is often present, and the blood shows increased icterus index.

The diagnosis has to be made from gall-bladder colic, and especially from common duct stone. Perforated ulcer cases do not vomit more than once, and the X-ray shows the crescent of free gas under the diaphragm. Renal colic and acute pulmonary and cardiac complaints may also mimic pancreatitis.

The treatment generally recommended is to make a mid-line epigastric incision and to incise the peritoneum over the pancreas. The approach can be made through the gastro-hepatic omentum in visceroprotic individuals, between the stomach and colon in those of normal physique, and below the mesocolon in fat sthenic individuals. A drainage-tube is inserted down to the region of the most intense inflammation. At the same time the stones should be removed from the gall-bladder and the viscus drained. The drainage of the pancreas through the peritoneal cavity has some disadvantages. Drainage retroperitoneally by a tube inserted from the left loin in front of the kidney is an excellent method and should be used more often.

When the pancreatitis is not very severe the stones may be removed and the gall-bladder drained and pancreatic drainage omitted. If the pancreatitis is obviously resolving at the time of operation, cholecystectomy may be performed, and the common duct explored and

drained if necessary. Pancreatic abscesses are best drained through the loin, if possible, with a *large* drainage-tube, because these abscesses generally contain large pancreatic sloughs.

*Pancreatic Cysts* are of two types: (a) True cysts inside the pancreatic tissue, which are uncommon and are of the usual retention types, cystadenomas, and parasitic and lymphatic cysts; and (b) Pseudo-cysts, which are much more common, and originate as a result of injury or inflammation, lie outside the pancreatic capsule, and sometimes occupy the lesser sac, and present above the stomach, or between stomach and colon or below the colon. The contents of these

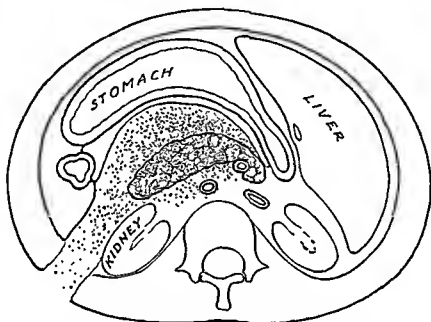


Fig. 459.—ILLUSTRATING LUSK'S APPROACH TO THE PANCREAS. IN INFLAMMATORY CONDITIONS THE SPACE AROUND THE PANCREAS IS FILLED WITH EXUDATE AND IS EASILY OPENED UP.

cysts is pancreatic juice, and as they usually cannot be removed, but have to be marsupialised or drained, the resulting fistulæ discharge digestive fluid. When possible, drainage should be made through the left loin, as in the following case of rather unusual interest:

A woman with a gall-stone history had been more ill in three recent attacks of pain. Noisy vomiting had been a marked feature, with epigastric rigidity and distension. I saw her when she was recovering from the last of these attacks, and she was slightly cyanosed, had a temperature and epigastric fulness, and the possibility of a subsiding pancreatitis was thought of. Two weeks later, when the patient was quite well, the abdomen was opened and the pancreas was found much swollen, while there was a great deal of fat necrosis throughout the abdomen, but

there was no sanguineous peritoneal fluid present, and it was felt that the attack was over, so the gall-bladder, packed with stones, was removed and the common duct explored with negative findings. She recovered well and went home twelve days later, and her doctor was told to watch for cyst formation. Five weeks later she was admitted with a large cystic swelling in the epigastrium. A three-inch incision in the left loin was made about one inch in front of the usual renal incision. The cyst was easily palpated in front of the kidney, and it was opened and two pints of straw-coloured fluid sucked out. The hand was then inserted into the cavity and a slough of the body of the pancreas removed from the posterior wall. The slough was black and about three inches long, and had calcareous patches of fat necrosis in it, and it was interesting to note that the only recognisable structure in the slough was the main duct. A pancreatic fistula formed after the drainage-tube was removed, but closed with the treatment outlined below.

*Pancreatic Fistulae* used to be regarded together with duodenal fistulae as of most serious prognosis, but now with appropriate management a large proportion get well. The digestion of the abdominal tissues and skin always became very serious, and infection resulted which, combined with the malnutrition, was generally too much for the patient.

The skin for six inches surrounding the fistula is treated with durofix (celluloid in amyl acetate) or mastisol so as to protect the skin. A small catheter with additional holes is inserted into the wound and connected with constant water suction or a McKesson noiseless suction motor. Round the catheter is packed gauze soaked in horse serum or meat juice (Potter), and then a liberal packing of kaolin powder spread over the dressing and the surrounding skin. With these precautions no digestion will occur, eczema of the skin will be prevented, and the fistula will close as any other fistula does.

*Pancreatic Calculi* are rare, but often cause much pain and suffering and require removal. This should be done as for any other calculus in a duct, and preparations made for dealing with post-operative pancreatic fistula.

*Pancreatic Neoplasms* are generally of malignant type, and when the head of the gland is involved are generally hopeless from an operative point of view. Complicated procedures involving removal of the duodenum and head of the pancreas, reimplantation of the common bile-duct and body of the pancreas into the intestine and gastro-enterostomy have been planned and attempted, but in all cases the patient has

failed to stay the course. In cases of carcinoma of the tail or the body removal may be quite possible, and numerous cases have been recorded, unique among which must be Waring's case, in which he removed the whole pancreas without metabolic or digestive disturbance ensuing! It is estimated that there is a margin of safety as regards insulin formation of a thousand times! Carcinoma of the islets of Langerhans has been described in the first famous case at the Mayo Clinic. Ten pounds of glucose a day could not prevent this patient from having hypoglycæmic fits, and secondary nodules of the growth in the liver contained large amounts of insulin as shown by rabbit tests.

*Benign Tumours* are of little interest except those adenomata of the islets of Langerhans, producing the state of hyperinsulism and hypoglycæmic state with its syndrome of ante-prandial fits, mental irritability, alkalosis, etc. Many of these tumours have now been removed, often with instantaneous cure: in other cases the adenoma has not been found, and at post-mortem a small adenoma, the size of a pea, has been found to be the cause of death. In cases where no adenoma can be found it is justifiable to resect the tail of the pancreas, but the results are as a rule unsatisfactory and the blood sugar remains low.

## CHAPTER II

### DIABETES IN RELATION TO ABDOMINAL SURGERY

by

R. SLEIGH JOHNSON

THE surgical considerations of diabetes mellitus may be said to fall into two distinct categories. On the one hand, the normal surgical misadventures of life equally bestrew the path of the diabetic as the non-diabetic, irrespective of his underlying malady. On the other hand is a group of special surgical conditions, including gangrene, cataract, and certain skin lesions, to which the diabetic is directly liable in virtue of his complaint. Each of these groups will call for appropriate modifications of surgical treatment. It is only, however, with the former class that it is intended to deal in the present review, with special reference to abdominal lesions in general and intercurrent abdominal emergencies in particular, as met with in the diabetic.

Before the days of insulin, a major surgical procedure, particularly if involving abdominal section, was to the diabetic patient a prospect of the gravest order; to such extent, indeed, that wherever possible, operation of any sort was sedulously avoided, so great was the risk of catastrophe. *With the modern benefits resulting from insulin, however,* not only has surgery lost its formidable outlook, but in the average case, given reasonable care and time for preparation, involves little or no greater risk than in the non-diabetic subject. The strength of such a statement, however, implies the supposition of facilities for close co-operation between surgeon, physician and pathologist, each contributing from his several aspect to the investigation and treatment of the case. It is an added advantage for the patient to be in an institution where accurate weighing and preparation of food can be relied upon, preferably supervised by an expert on dietetics.

In the consideration of abdominal surgery there is again a natural division into two groups according to the acuteness or otherwise of the case.

I. *The Chronic Case.* The relation with diabetes will be first discussed of non-urgent surgical conditions, where operation may be performed leisurely and at a time chosen to suit the convenience of patient and surgeon.

(a) *Pre-operative treatment.* With a straightforward "clean" operation as for a hernia or a quiescent appendicectomy, the object should be to get the diabetes completely under control as a preliminary measure so that the patient comes to operation in the best possible physical condition. The diabetes is therefore treated in the ordinary way, adopting whichever dietary scheme may be preferred by the surgeon, e.g. Graham's Ladder Diet or Lawrence's Line Ration Diet, in determining how much food and what doses of insulin, if any, are needed to keep the patient in a state of good nutrition, with fasting blood sugar maintained within normal limits and, above all, free in the immediate pre-operative period from ketosis.

The general regime of dietary and insulin treatment need not be considered here. It may, however, not be out of place to stress the importance of accurate diagnosis before beginning special diabetic treatment. In the first place, every glycosuria is not due to diabetes mellitus, and to such a case the administration of large doses of insulin may be not only unnecessary but dangerous. Suspicion should always be aroused by the discovery on routine examination of glycosuria unaccompanied by characteristic symptoms of diabetes such as thirst, wasting, and polyuria. Fallacies may arise from the technique of urinetesting, considered later. Granted, however, that a true glycosuria is found under such circumstances, the possibility should first be considered of other causes. The chief of these to be excluded is the so-called "renal glycosuria," where although there is no real disturbance of carbohydrate metabolism, the "leak-point" of the kidney for sugar lies at a lower level than normal and sugar is therefore allowed to leak through from the blood into the urine. The normal leak-point is between 170 and 180 milligrammes per 100 cc., below which level no sugar escapes from the blood. If, however, the leak-point is lower than this level, sugar will be passed in the urine although the blood sugar never rises above its normal upper limit of 170-180 milligrammes per 100 cc. Whether the resulting glycosuria is constant or only intermittent will depend upon the degree of lowering of the renal leak-point. If this is but slight, sugar will be found in the urine only after a carbohydrate meal. If markedly lowered, then glycosuria is constant.

An exact diagnosis of renal glycosuria can only be made from a sugar tolerance test, which wherever time will allow should always

be carried out. The diagnostic point is the presence of glycosuria in spite of a blood sugar curve which never exceeds the normal limit or remains persistently below. If this diagnosis is made, no special pre-operative treatment is called for, neither dietary restrictions nor insulin being indicated.

Other instances of non-diabetic glycosuria include nervousness from apprehension at the prospect of examination or operation, and usually confined to the immediate pre-operative period. Here stimulation of glycogen output from the liver by adrenalin is the causal factor. Other nervous conditions to be excluded are those producing glycosuria from increased intra-cranial pressure, as from a cerebral tumour, acromegaly, cerebral hæmorrhage or meningitis. The latter strictly affect the more acute group, but are mentioned here for convenience. The possibility of Graves' disease must also be remembered. Diabetes insipidus is, of course, unaccompanied by glycosuria and will not cause confusion.

In all these conditions the correct diagnosis will be confirmed from a glucose tolerance test, the abrupt abnormal rise and subsequent high plateau level of blood sugar of true diabetes mellitus being absent.

Fallacies may also arise, as mentioned above, from inaccurate urino-testing. Fehling's solution should not be the choice as a routine testing-agent, in view of the lack of delicacy of the test and the misleading results often obtained. It is, for example, reduced by such normal constituents of the urine as uric acid and creatinine. Benedict's solution is entirely satisfactory for clinical tests, which if made according to a fixed routine are roughly quantitative and afford a fair indication of the amount of sugar present. To 5 cc. of the reagent 8 drops of urine are added from a dropper, and the mixture boiled in a test-tube for 2 minutes over a flame, or in a boiling water-bath for 5 minutes, allowed to cool and the result read. According to the shade of colour change the presence and rough percentage of sugar is estimated, the alteration varying from a greenish opalescence only, without deposit on standing (approximately 0.1 per cent glucose), through shades of yellow, orange or bright red precipitate (2.0 per cent or higher); in the more complete stages of reduction there is, moreover, a proportionate disappearance of blue coloration of the supernatant fluid on standing.

Lactose in the urine of pregnant or lactating women also reduces Benedict's solution and has to be distinguished on other evidence.

In testing for acetone bodies and aceto-acetic acid as evidence of ketosis, the Rothera reaction is again the most satisfactory to employ,

depending upon the development of a deep permanganate colour in the presence of these substances in the urine. The test is made by saturating the urine with ammonium sulphate and adding a few drops of strong ammonia and of a freshly prepared solution of sodium nitro-prusside. The reaction is extremely delicate and far more reliable than Gerhardt's test, where a positive result is obtained only in the presence of a severe ketosis. The latter test depends upon the appearance of a light reddish-brown colour on the addition to the urine, drop by drop, of 10 per cent ferric chloride solution. It is also given in the presence of salicylates, aspirin or drugs of the phenol group.

To return to more direct considerations of treatment, there can be no rule of thumb as to the requirements of food and insulin in this preparatory period, as the needs will vary with the severity of the diabetes in the particular case. An initial blood sugar estimation is essential. Unless the hyperglycæmia be very marked the most convenient scheme of stabilisation is that now to be described. The older method of initial starvation has been largely superseded and the guiding principle is now to place the patient from the start upon a reasonable maintenance diet, for example, one "line" of Lawrence's diet per stone of body-weight or Diet X of Graham's scheme ("the top of the ladder"), irrespective of whether sugar is still being passed; if from the persistence of glycosuria it is then evident that dietary restrictions alone are inadequate, insulin is given without delay in gradually increasing doses until the blood sugar falls to within normal limits and the urine becomes sugar-free. The initial dose of insulin will be decided from the degree of hyperglycæmia; with a moderate rise a start may be made with 10 units daily, increasing by daily increments of from 5 to 10 units, or later less, until stabilisation is reached. The diet will then in most cases also need to be increased somewhat, modifying the final dosage of insulin accordingly. If more than 20 units a day are required the dose should be proportionately divided between a morning and an evening injection. The effects of a given dose of insulin in reducing the blood sugar vary considerably in different patients, and no exact rule can therefore be stated.

A diet liberal in carbohydrate should be the aim, building up a glycogen reserve by giving a minimum daily allowance of 100 grammes. It has been shown that with such a higher proportion of carbohydrate in the diet, the insulin requirements are proportionately reduced. In this regard it is of note that the Line Ration Scheme has been modified accordingly by Lawrence so that each "line" now contains 10 grammes of carbohydrate instead of the original 5 grammes. The



top stage (Diet X) of Graham's revised scheme contains 72 grammes of carbohydrate instead of 45 grammes as previously, while he also now recommends that the carbohydrate of the diet should be increased from this level by 10 grammes every two or three days until 100 grammes at least are being taken.

During the pre-operative period of stabilisation, it is an advantage for the patient to remain in bed, a more complete rest of the pancreas and earlier recovery of function being obtained. It is also important that any contributory cause such as minor foci of sepsis should be thoroughly treated before the major operation is undertaken. Not only does the eradication, for example, of septic teeth or skin lesions lessen the immediate operative and post-operative risk, but the effect on the severity of the diabetes and consequently on reduction of insulin requirements is often considerable. Where on account of the special nature of the abdominal lesion, e.g. peptic ulcer, the patient is unable to take the usual diabetic diet, an equivalent amount of carbohydrate should be given in a more easily digestible form, such as milk, bread-pap or sugar. Although it is possible to follow the progress of treatment roughly by routine urino examination alone, wherever laboratory facilities are available frequent blood sugar estimations, say at weekly intervals, should be made, since in the long-standing elderly type of diabetic the renal threshold is often considerably raised above the normal and a well-defined hyperglycaemia may be persistently present apart from any appearance of sugar in the urine. Such patients if inadequately treated run a serious risk of developing ketosis after operation. The time needed for satisfactory pre-operative control of a diabetic patient will vary greatly, from say three or four days up to a month, according to the individual circumstances.

To summarise, it is essential, therefore, in preparing a patient for operation that he should be fed generously and got into the best possible physical state, by a liberal use of insulin, so that no acidosis is present. The urine must be free both of sugar and of acetone bodies, the latter being a sign of inadequate carbohydrate supply and a call, therefore, to increase both carbohydrate and insulin accordingly.

Immediately before operation the body must have available an abundant reserve of sugar, stored as glycogen in the liver, to withstand the combined effects of anaesthesia, trauma, loss of fluid, and subsequent difficulties of feeding. This is ensured by giving say 50 grammes (2 ounces) of glucose 2 hours before operation, preceded by 25 units of insulin given 20 minutes earlier. The exact amount of each may be varied as considered advisable on the merits of the case; if,

for example, the diabetes is mild and well controlled, a smaller dose, say 25 grammes of glucose and 12 units of insulin, may be adequate. The probable length and severity of the operation is also to be borne in mind—the greater these factors the more sugar should be given. The risk of hypoglycæmia is obviated, provided that for every unit of insulin roughly 2 grammes of glucose are also administered. The glucose is least unpalatable if given flavoured with a little orange juice.

(b) *Operative Precautions—Anæsthesia.* The choice of anæsthetic is a matter of the first importance in view of the known effect of some upon the metabolism of carbohydrates and the production of acidosis. Where it is surgically adequate, e.g. for operations below the umbilicus, spinal anæsthesia is ideal, owing to the infrequency of subsequent vomiting and loss of fluid and the lack of liver disturbance. For many non-abdominal operations, of course, local anæsthesia alone will suffice and the difficulties inseparable from general anæsthesia avoided; local infiltration is similarly a valuable aid in suitable abdominal cases. The least disturbing of general anæsthetics is gas and oxygen, which, if cyanosis, its only drawback, be avoided by skilful administration, is free from objection. It may also be safely combined with spinal or local anæsthesia. Chloroform, at the other end of the scale, is the most dangerous because of its known toxic action on the liver, the protective glycogen reserves of which can never in a diabetic be up to the standards of health. Ether also is undesirable in view of its antagonistic action to insulin, which it inactivates in some obscure way, with liability to hyperglycæmia and the production without warning of acidosis. The loss of fluid from vasodilatation, sweating and post-operative vomiting also makes it undesirable, each of these factors tending to increase the liability to acidosis. If its use be kept down to a minimum, however, it may be given to supplement gas and oxygen without serious risk, and certainly with less danger than chloroform. Basal anæsthetics such as avertin are highly satisfactory and there does not appear to be any special contra-indication to their use in diabetics, carbohydrate metabolism showing no additional disturbance thereby.

(c) *Post-operative Care.* After operation on a chronic case, the general principles are to give abundant fluids to make good the body-loss sustained and to restore the glycogen reserve of the liver as rapidly as possible by further glucose and insulin. A temporary glycosuria is of no moment, the important factor being the avoidance of ketosis, formerly the great post-operative bugbear of diabetes. In the quiescent case it will suffice to give a quantity of each similar to that given before

operation, e.g. glucose 2 ounces with insulin 25 units. It is advisable, where convenient, to estimate the blood sugar before giving the post-operative dose of insulin and to modify it if necessary in accordance with the result. During the next 24 hours, whether vomiting be present or not, it is essential that the patient should be given, and retain, by some route a minimum of 100 grammes of carbohydrate, either by mouth, rectum or intravenously, together with enough insulin to cover it. It will generally be found that the insulin requirements of the patient after operation in this chronic group are at any rate temporarily increased.

It is seldom that the patient can return at once to the same details of diet as before operation, but it is, nevertheless, important that the necessary amount of carbohydrate should be given throughout, either as glucose or in some soft assimilable form. As early as convenient, the diet is gradually adjusted to what has previously been found to be the patient's individual requirement in the pre-operative stage of treatment. Details of control and transition are considered under the acute case, which they even more intimately concern.

**II. *The Acute Case.*** An outline has been given of the management of the diabetic surgical case where due time is available for investigation and preparation. Even greater care and watchfulness are called for in the event of a sudden abdominal emergency in a subject then found to be a diabetic and often inadequately controlled. It is, moreover, in the period immediately after operation that the chief anxiety will arise, supervision then being often a problem as complex and urgent as is the treatment of a diabetic coma.

The chief reason for these added difficulties lies in the profound effect of acute sepsis, so often associated with an abdominal lesion, in increasing the intensity of the diabetes and the liability to sudden intercurrent acidosis and coma. The insulin requirements are at the start enormously multiplied, while mechanical difficulties of absorption of carbohydrates from vomiting, diarrhoea or peritonitis add greatly to those of impaired oxidation from toxemia. It may be said dogmatically that an acute abdominal operation, if indicated immediately on other grounds, should never be postponed because of the presence of diabetes, even in the presence of acidosis or coma. The frequency with which a state bordering on coma is found, on examination, to be accompanied and caused by an acute septic infection in some part of the body needs no emphasis. A point of difficulty in diagnosis, however, may be mentioned, affecting predominantly the diabetic child, in whom the severity

of the underlying affection is usually high. It is sometimes not difficult to confuse abdominal symptoms due solely to incipient coma with those of a primary abdominal lesion, such as an acute appendicitis. Severe ketosis, for example, is not infrequently accompanied by nausea, vomiting, and severe epigastric or generalised abdominal pain of colicky type, with tenderness on pressure and muscular rigidity. Fever, however, is absent in ketosis alone, or the temperature is sub-normal, and with care the diagnosis can usually be made clear on other grounds of history and physical signs.

(a) *Pre-operative Treatment.* Examination of the urine and, if possible, an estimation of the blood sugar should be made at once to confirm the diagnosis and determine the grade of severity of the diabetes.

If the blood sugar is very high and the patient severely ill with abundant acetone bodies in the urine, 100 grammes of glucose should be given, together with insulin from 50 to 75 units subcutaneously. If he is unable to swallow or vomiting prevents absorption, the sugar should be given intravenously in a pint or more of saline, which is, moreover, of additional value in restoring the volume of circulating fluid. If the condition is less acute or if the patient is already under treatment with insulin, then smaller amounts of glucose and insulin respectively, e.g. half the above quantities, will be adequate, the general state and the blood sugar level being the final guides.

(b) *Precautions during Operation.* During the operation the prevention of shock and undue loss of heat is of importance, while considerations of anaesthesia and length of operation apply with even greater force than in the chronic case. Care must be taken with choice of stimulants, remembering the neutralising effect upon insulin possessed by adrenalin and pituitary extract, injection of which is followed by a rise of blood sugar.

(c) *Post-operative Treatment.* After the operation close observation is made of the patient's general state, the blood sugar is estimated two hours after return to the ward, and the urine collected, if necessary by catheter, at 2, 3 or 4-hour intervals according to the severity of the case and examined for sugar and acetone bodies. In cases with bladder obstruction it may be necessary to tie in a catheter to obtain the specimens at the times required and avoid error from the examination of residual urine.

If the pre-operative diabetes has been severe or the operation a serious one, it will commonly be found that the blood sugar two hours

after operation is again raised, although the urine which has collected meanwhile in the bladder may still be sugar-free from the pre-operative dose of insulin. Glucose and insulin are then administered again, in doses appropriate to the rise of blood sugar, the glucose being given if necessary intravenously with a continuous drip-saline or per rectum. No constant post-operative dosage can be advised, so variable are the factors in each case. Unless control by regular blood sugar and urine examinations is maintained, dangerous symptoms may arise at this stage from hypoglycæmia. The reaction of the tissues to insulin after so profound a disturbance is unstable and variable; the removal, moreover, of a large area of sepsis or the institution of open drainage is frequently followed by a sudden drop in insulin requirements as evidence of improvement, with serious danger of overdosage if such precautions are not carefully observed. This risk is, moreover, increased in a wasted, debilitated subject.

Subsequent control at this stage may be satisfactorily carried out by periodic examinations of the urine for sugar and acetone bodies, for example at 2-hourly intervals, and further treatment determined from the grade of reduction shown in the Benedict's tests. If a complete reduction is obtained in two successive specimens, a further injection of about 15 units of insulin with 30 grammes of glucose may be made, or proportionately less of each if the reduction is slighter. When the 2-hourly urine specimen becomes sugar-free, insulin should be withheld for the time and sugar alone given, as  $\frac{1}{2}$  oz. of orange juice, to minimise the risk of hypoglycæmia since the blood sugar may still be falling. The object of basing further insulin treatment upon the result of the urine specimen collected two hours earlier and not of that immediately obtained, is to counteract the slight lag with which the urinary sugar curve follows the blood sugar; in this way immediate treatment is not erroneously directed by the condition of "stale" urine which has been gradually collecting in the bladder during the previous two hours.

In severe cases, therefore, adopting the above regime, subsequent doses of insulin and glucose are either given or withheld at 4-hourly intervals, while unnecessarily frequent disturbance of the patient for blood sugar estimation at a time when he most needs rest is avoided. Later, of course, say after the first one or two days, blood sugar control will be resumed.

In less urgent cases, 3-hourly urine testing will suffice as a basis for 6-hourly insulin treatment, while in mild instances, 4-hourly and 8-hourly intervals respectively will prove adequate. In the more serious

case the shorter intervals adopted at the onset may be spaced out as improvement takes place.

The giving of abundant fluids in the post-operative stage is of no less importance than an adequate supply of carbohydrate. If there is any appreciable loss from vomiting the volume must be restored by other routes. Vomiting, moreover, adds to the difficulties of food absorption, and unless care is taken to make good the loss of carbohydrate rejected from the stomach a considerable risk is run of insulin-overdose. If there be any doubt, therefore, of the amount absorbed, the accompanying dose of insulin must be reduced or omitted, provided that the urine has remained sugar-free. The other main factor associated with risk of hypoglycæmia, namely the relief of acute sepsis, has already been mentioned.

*Hypoglycæmia.* It is essential, therefore, that the patient's attendants should be familiar with, and on the alert for, symptoms and signs of overdose. The first warning usually takes the form of marked subjective limpness and prostration, with trembling and a feeling of unrest and apprehension. Profuse sweating may occur, with pallor and coldness of the limbs. There may be a sense of constriction of the chest. The pupils are widely dilated, but the eyeballs do not show the striking loss of tension found in true diabetic coma. Undue acceleration or sometimes a marked slowing of the pulse is frequently present. With increasing severity, aphasia, restlessness or delirium, muscular twitchings or convulsions may in turn appear and finally coma develops. Symptoms usually become evident from 2 to 3 hours after the injection of insulin; the level of blood sugar at which they begin shows considerable variation in different subjects, being often in the region of 50 milligrammes per 100 cc. In chronic diabetics, however, in whom the blood sugar has long been raised to an abnormally high level, they may coincide with a blood sugar well over the 100 milligramme mark.

If symptoms suggestive of hypoglycæmia develop, the low blood sugar content should if possible be confirmed. Treatment lies primarily in giving glucose without delay. In mild cases 2-4 oz. of orange juice by mouth will suffice; in the more severe, 20 grammes of glucose should be given by mouth, appropriately flavoured, or by rectum, or again if the need be urgent 2 grammes should be given intravenously as 20 cc. of a 10 per cent solution. Recovery is accelerated by the injection of either 1 cc. of adrenalin hydrochloride 1/1000 subcutaneously, or 1 cc. of pituitrin intramuscularly, each of these substances being antagonistic in action to insulin as regards effect upon blood sugar.

When the immediate dangers of operation have been overcome, the next object is to plan a return by easy steps to a fuller diet. The diet given at this stage should in general terms be the same as would be given to the similar non-diabetic patient, together with an adequate amount of insulin to allow of its utilisation. In gastric cases, for example, milk will form the basis together with soft carbohydrates, Benger's food, custards and such-like, as the general state and digestive powers improve. In this regard it may be remembered that a pint of milk contains the equivalent of approximately 28 grammes of glucose and therefore requires 14 units of insulin. Until such time as the patient is able to take his pre-operation level of carbohydrate by mouth, any deficiency below this total amount is made good as glucose and given if necessary per rectum. Thereafter, a gradual resumption of the more customary diabetic diet is made, as the local abdominal condition will allow, and the insulin requirements modified accordingly. Periodic examination of the blood sugar is continued, say at weekly intervals, or not less often than once a month.

The daily requirement of insulin may be influenced and altered as a result of operation in different ways. In the majority of cases it is at least temporarily increased, often but little, in some two- or three-fold, by the general disturbance of metabolism. Where the case has been a chronic one and the operation relatively "clean," some such increase commonly results. In the frankly septic case, as for example the removal of an acutely or chronically infected gall-bladder or appendix, tonsillectomy, and even more the amputation of an infected gangrenous limb or excision of a carbuncle, the subsequent severity of the diabetes is often materially lessened and the dosage of insulin required may be correspondingly reduced. Similarly, the successful removal of a malignant neoplasm is commonly followed by an increase in sugar tolerance.

To summarise, enough has probably been said to indicate that, while the level of safety of surgery in the diabetic has without doubt been greatly raised, nevertheless much added care and observation are called for; before an operation is decided upon there should always therefore be reasonable evidence of its necessity.

**SECTION 9**

**SPLEEN**

**CHAPTER I**

**Indications for Splenectomy**

**by**

**RODNEY MAINGOT**

**CHAPTER II**

**Technique of Splenectomy**

**by**

**RODNEY MAINGOT**

**CHAPTER III**

**Complications and Physiological Effects of Splenectomy**

**by**

**RODNEY MAINGOT**

**CHAPTER IV**

**Radio-therapy in Splenic Diseases**

**by**

**WALTER M. LEVITT**



CHAPTER V

The Results of Splenectomy at the Mayo Clinic

CHAPTER VI

The Spleen and the Blood-Platelets

by

W. HOWEL EVANS

CHAPTER VII

Splenic Diseases of Surgical Importance

by

RODNEY MAINGOT

CHAPTER VIII

Rupture of the Spleen

by

RODNEY MAINGOT

CHAPTER IX

Egyptian Splenomegaly

by

H. E. S. STIVEN

## SECTION 9

## SPLEEN

### CHAPTER I

#### INDICATIONS FOR SPLENECTOMY

by

RODNEY MAINGOT

SPLENECTOMY may be called for :

(1) *In Cases of Rupture.*

(a) Direct injury.

(i) Penetrating wound.

(ii) Laceration.

(iii) Prolapse through external wound.

(b) Indirect injury.

(c) Spontaneous rupture.

(2) *To Remove a Focus of Disease.*

(a) Aneurysm of the splenic artery.

(b) Abscesses.

(c) Cysts.

(i) Hydatid.

(ii) Blood, serous, or lymphatic.

(iii) Dermoid.

(d) New growths.

(i) Innocent.

1. Fibroma.

2. Lymphoma.

3. Lymphangioma.

4. Hæmangioma.

- (ii) Malignant.
  - 1. Sarcoma.
  - 2. Endothelioma.
- (c) Splenomegaly in disease of bacterial or protozoan origin.
  - (i) Primary tuberculous disease.
  - (ii) Gumma.
  - (iii) Malaria.
  - (iv) Egyptian splenomegaly.
- (f) Gaucher's disease.
- (3) *In Anomalies of Position.*
  - (a) Thoracic, in cases of diaphragmatic hernia.
  - (b) Abdominal—wandering or floating spleen, often with torsion of the pedicle.
- (4) *To Reduce the Blood in the Portal Circulation.*
  - (a) Splenic anæmia (Banti's disease).
  - (b) Hepatic cirrhosis.
  - (c) Thrombophlebitic splenomegaly.
  - (d) Primary, essential, or idiopathic splenomegaly.
- (5) *Where there is Evidence that the Spleen is Engaged in Excessive Destruction of or Damage to the Cells of the Blood.*
  - (a) Essential thrombocytopenic purpura hæmorrhagica.
  - (b) Acholuric or hæmolytic jaundice with increased fragility of the erythrocytes.
- (6) *von Jaksch's Disease.*

The above list of indications for splenectomy is purely tentative, as any classification must be incomplete owing to our imperfect knowledge of the pathology of splenic diseases. Splenectomy has, of course, been performed for many conditions other than those indicated above, such as pernicious anæmia, the leukæmias, polycythæmia, hæmophilia, etc., but the days of splenectomy for mere splenomegaly are past. Splenectomy should now be advised only upon some definite indication, based upon combined clinical, hæmatological, and pathological considerations. Splenectomy may be successful even in the most desperate and unpromising cases, and where the operation offers the only prospect of cure or amelioration of symptoms the patient should be urged to face the ordeal.

## CHAPTER II

### TECHNIQUE OF SPLENECTOMY<sup>1</sup>

by

RODNEY MAINGOT

THERE are many ways of performing the operation of splenectomy, and these will vary with the technique employed by the individual surgeon and the particular circumstances which call for removal of the spleen. The operation may be one of great simplicity when the organ is of normal size, or, if enlarged, when there are no binding adhesions. On the other hand, in certain conditions in which splenectomy is clearly indicated, the technical difficulties may be so great that the operation may have to be abandoned altogether. In some cases of splenomegaly the organ may be so inextricably tethered to the diaphragm and adjacent viscera that it becomes a physical impossibility to mobilise the spleen and ligature off its anomalous vascular pedicles.

There are certain features in the technique of the operation which I propose to emphasise in this article, as they are not generally appreciated, or have been somewhat neglected in descriptions of the operation.

Figure 461 shows diagrammatically the two vascular pedicles of the spleen, and the somewhat complicated visceral reflections of the peritoneum in the region of the hilum. It demonstrates the necessity of securely ligaturing and dividing the gastro-splenic omentum before dealing with the true pedicle which contains the splenic artery and vein.

It is most important to ensure that the stomach is empty and flaccid before the operation is commenced, by passing a small stomach tube and aspirating the contents. If the stomach is distended with gas or fluid it will push the spleen into an even more inaccessible position, in addition to rendering all intra-abdominal manipulations more cumbersome, difficult, and not free from danger inasmuch as the stomach itself may receive some injury when the uppermost portion of the gastro-splenic omentum is ligated. It is unlikely that the stomach will contain any fluid except in cases of injury to the spleen; but if an inhalation anaesthetic is being given it is usually distended with gas.

<sup>1</sup> Reprinted (with illustrations) from *Surgery, Gynecology, and Obstetrics*, Jan., 1934. Vol. lviii, 62-66, by kind permission.



Fig 460.—THE BLOOD-VESSELS OF THE SPLEEN.

- A—Splenic artery.
- B—Splenic vein.
- C—Vasa brevia.
- D—Left gastro-epiploic artery.
- E—The tail of the pancreas.

(Adapted from Kirschner's "Operative Surgery," Lippincott, by kind permission of Julius Springer.)

A stomach tube, therefore, which has been introduced prior to the operation and is left *in situ* until it is completed, ensures an empty stomach, and simplifies the further steps in the operation.

There are four important stages in the operation: (1) the abdominal incision and exposure of the spleen, involving the question of choice of incision; (2) the freeing of adhesions, the mobilisation of the spleen, and its delivery through the abdominal wound; (3) the methods of securely ligaturing the vascular pedicles; (4) the closure of the wound and ensuring meticulous hæmostasis of the blood-vessels in the anterior abdominal wall.

(1) *Incision and Choice of Incision.* Numerous incisions have at one time or another been employed for splenectomy, but I shall describe only the four which I would myself recommend for the reasons given.

(a) Mid-line incision (see fig. 462, A). This is indicated in cases of rupture of the spleen, as it is easy to make and easy to close, besides affording satisfactory access to the spleen and permitting inspection of the liver and the other important viscera of the upper abdomen, which may or may not be implicated. If any difficulties are encountered during the operation, a ready approach to the spleen will be obtained by cutting the left rectus muscle transversely through one of its tendinous intersections.

(b) Paramedian incision (see fig. 462, B). This is recommended for the majority of cases of splenomegaly, and is the one preferred by most surgeons. The incision does not in any way damage the rectus muscle, gives a good exposure, and subsequently leaves a sound scar. There is, however, the drawback that the employment of this incision may protract the operation by a few valuable minutes.

(c) Transrectus muscle incision (see fig. 462, C). This is the incision I have used in the majority of my cases. It commences over the costal margin, and proceeds vertically downwards to the level of the umbilicus. It is placed at the junction of the middle and inner thirds of the left rectus muscle, and traverses all the structures of the anterior abdominal wall in the same plane. It gives ready approach to the spleen, and is particularly easy to sew up rapidly. I have never seen a post-operative

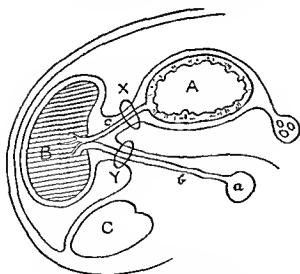


Fig. 461.—VASCULAR PEDICLES OF THE SPLEEN.

- X = False pedicle (gastro-splenic omentum).
- Y = True pedicle (lienorenal ligament).
- A = Stomach.
- B = Spleen.
- C = Kidney.
- a = Aorta.
- b = Splenic artery.
- c = Vasa brevia.

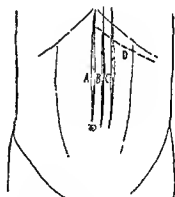


Fig. 462.—INCISIONS FOR SPLENECTOMY.

- A = Mid line
- B = Paramedian.
- C = Transrectus muscle.
- D = Subcostal.

ventral hernia develop after the employment of this incision, but I have two cases which show definite weakness of the inner fibres of the muscle.

(d) Subcostal incision (see fig. 462, D). This incision begins at the tip of the xiphisternum, and runs obliquely outwards and downwards, two finger-breadths below the costal margin. The rectus muscle and the muscles of the lateral abdominal wall are divided in the line of the incision. It is, in fact, similar to Koehler's gall-bladder incision on the opposite side, and should be employed for those cases in which splenectomy is called for in very obese patients. If this wound is carefully sutured there is no risk of subsequent hernia.

There are three important points, therefore, which should characterise all these incisions: (1) They should be large and generous, so that there is no hampering of the intra-abdominal manipulations. (2) Special pains should be taken to see that wound hæmostasis is thorough and complete. Neglect of this may, owing to the condition of the blood in such patients, lead to subsequent hæmorrhage, or a dangerous—possibly fatal—oozing. (3) Closure of the wound should be performed carefully, and tension sutures inserted as a precautionary measure against burst abdomen or post-operative ventral hernia, which are by no means infrequent complications.

(2) *Freeing of the Adhesions, the Mobilisation of the Spleen, and its Delivery through the Abdominal Wound.* As soon as the abdomen is opened the first step should be a rapid but complete exploration of the liver, the gall-bladder and its ducts, the pancreas, stomach, and duodenum. If not performed as a routine at this stage it may be forgotten or omitted after the splenectomy, and some important concomitant or independent intra-abdominal lesion be overlooked which will subsequently mitigate or completely destroy the benefits of the operation. It will be noticed that as a result of passing the stomach tube the stomach lies high up, tucked underneath the liver, empty and contracted. If, however, the viscus is still distended, the stomach tube should be reinserted and left in position during the remainder of the operation. Both edges of the wound are now well retracted to the right hand and to the left, and any adhesions which readily present themselves are carefully divided and ligatured. It is quite common to find slight adhesions between the spleen and the anterior abdominal wall, or between the spleen and the transverse colon. These should be picked up, divided between artery forceps, and ligatured off before proceeding further.

The spleen should then be carefully palpated, its size and consistency should be gauged, its mobility ascertained, and the presence or absence

of adhesions between the spleen and the diaphragm noted, before deciding upon the next step in the operation.

A careful search should be made for an *accessory spleen* or *splenculi*. It is stated that an accessory spleen is present in 10 per cent of cases. My experience is that the incidence has been under-estimated, and while in normal subjects this figure may be correct, in cases of splenomegaly and in certain other diseases of the spleen, such as essential thrombocytopenic purpura hæmorrhagica, it is very much greater. In seven out of thirteen cases of essential thrombocytopenic purpura hæmorrhagica I have found more than one spleneulus to be present—53 per cent. These spleneuli have no fixed anatomical position. Usually, however, they are found at the lower pole of the spleen, in the lower half of the gastro-splenic omentum, or in the great omentum. They vary considerably in size and shape, but are generally circular and about the size of a marble, although at other times they may be oval or disc-like. They receive their blood supply from branches of the left gastro-epiploic artery, vasa brevia, or from the splenic artery itself.

If a spleneulus is found at operation it should be excised in all cases except where the spleen has been removed for rupture, as it partakes of the same morbid processes or malign characteristics as its mammoth forebear, and cases are recorded in which after splenectomy a spleneulus has grown as large as a normal spleen. (Eeles and Freer, *B.M.J.*, 1921, ii, 515.)

The subsequent steps of the operation will be governed by the following factors: (a) the size of the spleen; (b) the presence or absence of adhesions between the spleen and the diaphragm; (c) the length and mobility of the lienorenal ligament.

If the spleen is small or normal in size, or even somewhat enlarged, if there are no adhesions (or, if any exist, they are very slight), and if the lienorenal ligament permits of some mobility of the organ, the procedure should be as follows: The lower two-thirds, or even more, of the gastro-splenic omentum is transfixed and ligatured off, the ligatures being placed nearer to the spleen than to the stomach, as it is very easy to damage the stomach or to include a portion of it in a ligature (fig. 463).

The remaining third of the gastro-splenic omentum is transfixed and ligatured after the spleen has been delivered through the wound. The left hand is then passed behind the spleen, which is drawn away from the diaphragm and the chest wall over to the right, thus bringing the posterior aspect of the lienorenal ligament into view. With scissors



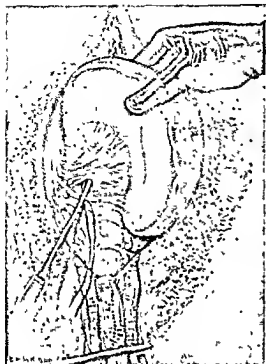


Fig. 463.—GASTRO-SPLENIC OMENTUM DISPLAYED. LIGATURE OF BLOOD-VESSELS IN GASTRO-SPLENIC OMENTUM.

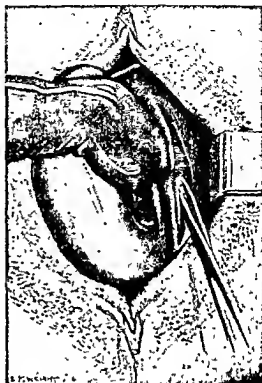


Fig. 464.—MOBILIZATION OF THE SPLEEN BY DIVISION OF THE POSTERIOR LEAF OF THE LIENO-RENAL LIGAMENT.

or a long knife the posterior leaf of this ligament is divided, when a further mobilisation of the organ will be rendered possible. The underlying areolar tissue and fascia propria are then further incised, and, with the finger or gauze dissection the tissues are separated, permitting of an even further freeing of the spleen.

This step is most important, and may be regarded as the key to the whole operation, as it is the lienorenal ligament which binds the spleen down in its hidden retreat in the abdominal cavity. When the lienorenal ligament is very mobile it is possible to deliver the spleen through the abdominal wound without employing this method. (Wilkie, *Amer. Jl. Surg.*, Oct., 1931, 340.)

(3) *Methods of Securely Ligaturing the Vascular Pedicles.* The spleen is now quite free, and can easily be drawn fully through the abdominal incision. After any remains of the gastro-splenic omentum have been dealt with and the tail of the pancreas identified and stripped away from the larger vessels, the main vascular pedicle is ready to be ligatured (fig. 465).

A large, hot, moist Cripps' pad is now placed in the space that

was formerly occupied by the spleen so as effectually to control any oozing from raw surfaces in this area. After isolating the vascular pedicle it is clamped with three large forceps.

In doing this the operator places the left index finger behind the pedicle from above downwards, and lifts the pedicle forwards to ensure that the forceps are applied by sight, and that in their application no damage is done to the pancreas, stomach, or colon. This is the three-clamp method of Fédoroff.

The spleen is then removed by severing the pedicle with a knife between the middle and distal hæmostats. The inner or medial hæmostat is then removed, thus leaving a groove or crushed area in the pedicle.

Figure 468 shows the method of ligaturing off this pedicle. Two stout 20-day chromic catgut ligatures are applied to the groove and tied side by side, after which each individual vessel distal to this is picked up with a hæmostat and ligatured off separately.

After ligaturing the last blood-vessel and removing the Cripps' pad, the appearance will be as presented in figure 470. A wisp of adjacent omentum is drawn over the rough surface of the pedicle and held in

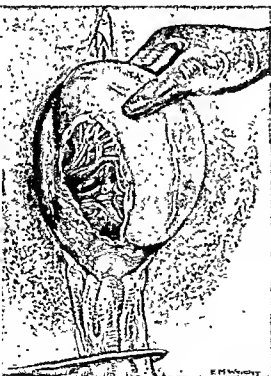


Fig. 465.—THE GASTRO-SPLENIC OMENTUM HAS BEEN DISSECTED AND THE BLOOD-VESSELS IN THE LIENORENAL LIGAMENT ARE SHOWN.

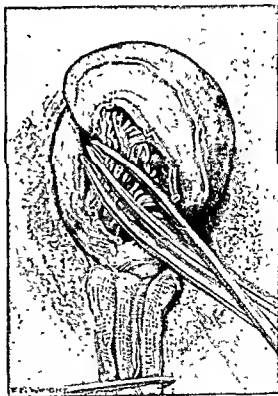


Fig. 466.—THE THREE CLAMP METHOD OF FÉDOROFF FOR DEALING WITH THE PEDICLE.

position by a stitch or ligature, so that no raw surface remains at the completion of the operation.

In cases of *splenomegaly* in which the spleen is greatly enlarged the procedure will be different. The gastric surface of the spleen and the gastro-splenic omentum at once come into view when the peritoneal cavity is opened. The spleen lies closely applied to the greater curvature of the stomach, the gastro-splenic omentum is vertically lengthened

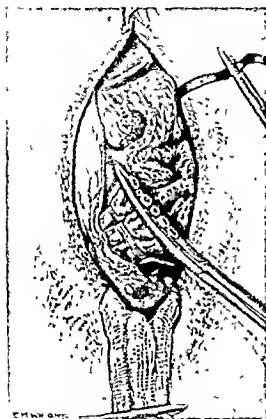


Fig. 467.—THE SPLEEN HAS BEEN REMOVED. THE CURVED GROOVED AREA WHICH REMAINS AFTER THE REMOVAL OF THE MEDIAL HEMOMENTUM IS SHOWN READY FOR THE APPLICATION OF THE LIGATURES.



Fig. 468.—TWO STOUT LIGATURES ARE APPLIED TO THE CURVED GROOVE, AND THE INDIVIDUAL BLOOD VESSELS DISTAL TO THIS ARE LIGATURED OFF SEPARATELY. NOTE THE POSITION OF THE CRIPP'S PAD.

and horizontally shortened, and the blood-vessels are enormously enlarged and increased in number. This enlargement is particularly prominent near the upper pole of the spleen, and is very noticeable in cases of splenic anemia. Each of these blood-vessels in the gastro-splenic omentum will have to be carefully and individually under-run with an aneurysm needle, and ligatured with thick catgut or silk.

After the blood-vessels in the gastro-splenic omentum have been dealt with, the right hand should be pressed to the back of the spleen.

Adhesions in this remote area are invisible, and will have to be separated with the finger. Bleeding may be sharp and troublesome at this stage, but the stripping and breaking down of adhesions will have to be proceeded with, as it is essential to free the organ posteriorly so

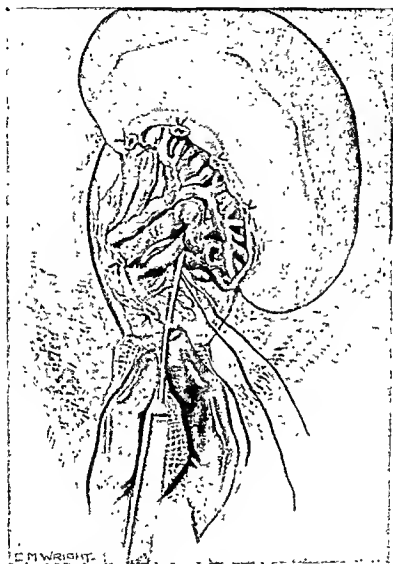


FIG. 463.—THE BLOOD VESSELS ARE LIGATURED SEPARATELY.

that it can be drawn or hooked over to the right to display the peritoneum which forms the posterior leaf of the lieno-renal ligament.

As soon as the spleen has been coaxed out of its bed, and has been drawn well over to the right, the subsequent stages for the further mobilisation of the spleen are similar to those described above. (See fig. 464.)

A hot Cripps' pad soaked in saline is packed against the diaphragm

to control bleeding for the time being. As complete a mobilisation as is possible should be attempted, and facilitated by turning the spleen over to the right on its pedicle, which acts as a hinge, and carefully separating the tail of the pancreas. In some cases of splenomegaly the splenic artery and vein may be enormously enlarged, friable, tortuous, and sacculated. In advanced cases of Banti's disease I have noted that their size may vary from  $\frac{1}{4}$  inch to  $1\frac{1}{2}$  inches in diameter.

Each individual blood-vessel is most carefully separated, and with scrupulous care an aneurysm needle, carrying a very stout ligature, is passed round it (see fig. 469), and the vessel ligatured in two places fully  $\frac{1}{4}$  inch apart. It is advantageous to tie off the main branches of the artery first, so that some of the blood from the spleen may be drawn back into the circulation. Each vessel that is doubly ligated is then severed between the ligatures, and this procedure is repeated step by step until the whole vascular sheath has been dealt with.



Fig. 470.—APPEARANCE AFTER SPLENECTOMY. THREE SUTURES HAVE BEEN APPLIED TO THE BLANKING POINTS ON THE DIAPHRAGM

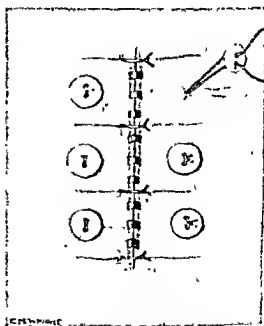


Fig. 471.—CLOSURE OF THE WOUND.

The veins are particularly friable, and the ligatures may cut through them and start a very troublesome hæmorrhage. I remember one case of splenomegaly in which I had to use No. 8 plaited silk to ligature the blood-vessels, as the catgut ligatures when tightened led to rupture and even severance of the venous trunks at the seat of ligation. It is possible, too, for the blood-vessels to rupture proximal to the ligatures, and I have known a case in which this occurred with a fatal result. It is obvious that in these cases, because of the increased size of the pedicle, the three-clamp method is unsuitable.

After dealing with the vascular pedicle, the Cripps' pad is removed, and any oozing surfaces on the diaphragm are picked up, under-run by a "snaking" suture, and tied off.

Drainage is unnecessary.

Before the wound is closed, and after complete hæmostasis is assured, a pint or two of warm normal saline is poured into the abdominal cavity. This often gives the patient that extra little fillip at a time when it is most needed. In cases of rupture of the spleen the blood in the peritoneal cavity can be citrated and used for auto-transfusion; but, so far, I have never had to resort to this measure. (Haulke, *Beitr. z. klin. Chir.*, 1921, cxxii, 389.)

(4) *Closure of the wound.* The wound is closed in layers with strong catgut, and three or four tension sutures are inserted to guard against the possibility of burst abdomen or post-incisional hernia. The tension sutures are placed through metal or mother-of-pearl buttons so as to prevent pressure upon or cutting of the skin.

The technique of splenectomy in cases of Egyptian splenomegaly is described on page 902.

## CHAPTER III

### COMPLICATIONS AND PHYSIOLOGICAL EFFECTS OF SPLENECTOMY

by

RODNEY MAINGOT

*Complications.* The post-operative complications are numerous, but may be epitomised as follows :

(1) *Shock.*

(a) Primary.

(b) Delayed.

(2) *Hæmorrhage.*

(a) Oozing from the wound.

(b) Hæmorrhage from the splenic bed due to severed adhesions.

(c) Hæmorrhage from omental adhesions.

(d) Slipped ligature.

(e) Rupture of a splenic blood-vessel proximal to the ligature.

(f) Hæmatemesis as the result of injury to the stomach wall or to the gastric veins.

(3) *Thrombosis.*

(a) Thrombosis of splenic vein and superior mesenteric vein. This is very prone to occur in cases of thrombocythæmia and where thrombophlebitis of the splenic vein is present.

(b) Thrombosis of other veins in the body, e.g. femoral vein or portal vein.

(4) *Peritoneal Effusion*, due to injury to the tail of the pancreas. This is usually associated with pyrexia. The effusion may take as long as a month to subside, and may possibly account for the condition known as splenic asthenia.

(5) *Sub-diaphragmatic Suppuration.* This may be caused by injury to the tail of the pancreas or some adjacent viscus, e.g. stomach or colon.

(6) *Left-sided Pleural Effusion, Empyema, Pulmonary Collapse, or Pneumonia Due in some Cases to Injury or Bruising of the Under-surface of the Diaphragm.*

(7) *Persistent Hiccough*, the result of irritation of the branches of the left phrenic nerve upon the under-surface of the diaphragm.

(8) *Burst Abdomen.* This may be attributable to :

(a) Injury to the tail of the pancreas with the liberation of pancreatic ferments and the consequent digestion of catgut sutures.

(b) The general condition of the patient—anæmic, debilitated, or suffering from some serious blood disorder.

(c) Hasty closure of the abdominal wall.

(9) *Post-Operative Ventral Hernia.* This may be caused by forcible retraction of the abdominal wall during operation, wound sepsis, or inadequate closure of the incision.

(10) *Intestinal Obstruction.* This may be due to a portion of small intestine becoming adherent to the raw surface of the vascular pedicle of the spleen, months, or even years, after splenectomy has been performed.

*Physiological Effects.* These may be briefly outlined as follows :

(1) *Changes in the Blood.*

(a) White blood-corpuscles. Immediately after splenectomy there is a polymorphonuclear leucocytosis which lasts for a few days. This is followed by a lymphocytosis which may persist for at least four months. During the first 24-48 hours after splenectomy there may be a leucocytosis of 30,000, but this gradually drops until at the end of the first week the count may be only 20,000. After a variable period, usually not less than six months, there may be a slight eosinophilia, and with this there may be, and often is, an increase in the number of mast cells.

(b) Red blood-corpuscles. There is a transient anæmia, but this disappears within two to three months. It is exceptional for the red blood-corpuscles to fall below 3,000,000 as the result of splenectomy. The fragility of the erythrocytes is found to be permanently decreased. Estimation



of the hæmoglobin will show a moderately low percentage during the first week or two following removal of the spleen, but this gradually rises, until at the end of about two months it is practically normal. The platelets, normal at about 250,000 to 400,000 per cubic millimetre, are increased after splenectomy. They may rise by several thousands within the first few days, but after a month has elapsed their numbers diminish to a figure even below the normal count, and during the ensuing months they fluctuate, until eventually a more or less normal number is again found in the circulating blood.

(2) *Enlargement of the Lymphatic Glands.* There is generally enlargement of all the lymphatic glands in the body, due to compensatory increased hæmatopoietic activity.

(3) *Changes in the Bone-Marrow.* The yellow marrow in many of the long bones is gradually replaced by red marrow, this change usually being complete within a period of six months.

(4) *Changes in the Reticulo-Endothelial System.* The specialised cells of the remaining portions of the reticulo-endothelial system in other organs undergo a marked proliferation.

(5) *Hypertrophy of Splenuli.* (See page 809.)

(6) *An Increase of Iron in the Tissues,* as shown by Hill and Flack.

(7) Where splenectomy has been performed the process of *Regeneration of the Blood*, following a severe hæmorrhage, is said to take place more slowly. Such patients will less readily develop jaundice after the absorption of hæmolytic substances. It should be noted that there is no evidence whatsoever that following the operation of splenectomy the resistance of patients to infection is diminished in the least degree.

(8) As 25 per cent of the total amount of portal blood passing through the liver comes from the spleen, the result of splenectomy will be to *Relieve the Liver* of some of its burden, especially when it is cirrhotic.

*An Alternative to Splenectomy* is ligation of the splenic artery, and this method may be advised when the operation presents insuperable technical difficulties in the removal of the organ, such as might

be seen in a case of chronic splenomegaly where the spleen is inextricably welded to the diaphragm, or where dense, extensive perisplenic adhesions render the operation not only formidable and prolonged, but even unwarrantable and impossible in a patient who is already a very poor operative risk.

Where this operation is undertaken the final results are often disappointing.

CHAPTER IV  
RADIO-THERAPY IN SPLENIC DISEASES  
By  
WALTER M. LEVITT

(1) EFFECTS OF X-RAYS ON NORMAL SPLEEN

(a) *Direct Effects.* Changes in the spleen are observed very soon after an application of X-rays. Heincke described degenerative changes in the follicles one hour after intensive radiation. Macroscopically, the effect of radiation is shown by marked shrinking of the organ, which becomes darker in colour. Microscopically, a great increase in pigment is observed, partly intracellular in the pulp cells, partly free. The follicles are reduced in size, and the cells in number, and large intercellular spaces are seen. The degenerative process commences at the centre of the follicle and spreads outwards. The nuclei of the lymphoid cells disappear and the chromatin collects in the intercellular spaces, and is afterwards taken up by phagocytes. The pulp tissue is seen to be more open in arrangement than normal, and here also a marked deficiency in the cell content is observed. Abnormal cells in the form of epithelioid cells are also seen. These cells are three or four times as large as a lymphocyte and have an eosinophil protoplasm.

(b) *Indirect Effects.* Various indirect effects have been claimed for X-rays from the irradiation of the spleen by X-rays. The two most important of these are: (i) The shortening of the clotting time of the blood (Stephan, v. d. Hutten, and others); (ii) The production of lymphocytosis (Wetterer). Various attempts have been made to turn these effects to account in X-ray therapy. Thus, irradiation of the spleen has been advocated in the treatment of the purpuras (Meda, Pancoast, and others) and hæmophilia, and also as a prophylactic against excessive hæmorrhage in cases in which operations are to be undertaken in very vascular regions (Partsch). Repeated small doses of X-rays to the spleen are also of value in certain forms of menorrhagia

(Nürnberger, Werner, and others). The lymphocytosis-producing effect has been made use of in the treatment of tuberculosis. Among other indirect effects claimed for the irradiation of the spleen is the prevention or control of anaphylactic phenomena. Thus, splenic irradiation has been suggested in the treatment of bronchial asthma and of urticaria.

(c) *Can Radiation replace Splenectomy?* The effects described upon the splenic cellular elements suggest that with sufficiently intensive irradiation the function of the spleen might be capable of being destroyed, or at any rate so depressed by X-ray treatment as to replace splenectomy in the diseases in which the latter operation is indicated. Clinically, however, trial of the method has so far failed to produce satisfactory results in splenic anaemia. Nor would the effect of X-rays in purpura hæmorrhagica appear to be as certain or as rapidly obtained as that of splenectomy. The present position, therefore, is that X-ray treatment would only be employed in such cases when for some reason splenectomy was contra-indicated.

## (2) X-RAY THERAPY IN DISEASES AFFECTING THE SPLEEN

*The Leukæmias.* This group of diseases forms by far the commonest indication for irradiation of the spleen. They are all characterised by progressive loss of strength, dyspnoea on exertion, and tendency to hæmorrhage. Examination reveals an enlarged spleen, which may be of enormous proportions and may fill the whole abdomen; the liver may also be enlarged. In the lymphatic type of leukæmia, and occasionally in the myeloid type, the lymphatic glands, both superficial and deep, may be enlarged and may form very large masses. The two main types of the disease are distinguished by the blood picture. While in both types the total leucocyte count is greatly increased, often to 500,000 or more, in lymphatic leukæmia the lymphocytes preponderate, forming often 90 per cent of the total white count, while in the myeloid type the polymorphonuclear leucocytes, and their more primitive generations, the myelocytes and the transitional cells, preponderate. In acute cases, and in the end-stages of the chronic cases, the parent non-granular myeloid cells (so-called myeloblasts), may form a considerable proportion of the total count, and this may be taken as an indication of great activity of the disease. Concurrently with the increasing leucocyte count, there is a progressive deficiency in the red cells and hæmoglobin, to which may be attributed many of the symptoms.

The value of X-rays in the treatment of the chronic leukaemias was discovered very soon after the discovery of the rays themselves. When correctly applied, the white cells can soon be reduced to normal numbers and approximately normal proportions. The red cells and hæmoglobin rise, and the general condition of the patient is restored. This improvement is maintained for a period which varies from months up to even a few years, but sooner or later a recurrence appears. The recurrence is treated in the same way as the original condition, and a further, though usually shorter, remission is obtained. With successive recurrences, the remissions become progressively shorter, and eventually the effects of the radiations fail to keep pace with the ever-rising leucocyte count. At this stage, treatment has to be abandoned and the patient dies from profound anaemia, hæmorrhages, or perhaps from pressure from glandular swellings.

Improvement can be obtained in the treatment of leukaemia from irradiation of any region of the body, but there is little doubt that the best results are obtained when the spleen is irradiated. The irradiation of this organ has to be carried out with extreme caution and must be controlled by frequent blood examinations. Over-irradiation may easily cause acute marrow failure and consequent death of the patient. When, however, splenic irradiation is carried out with due care and skill the results obtained are better than those gained from the irradiation of any other region. Irradiation of the bone-marrow is frequently advocated in leukaemia, but in the writer's experience this method has the disadvantage that much more radiation has to be applied to obtain the desired effect on the leucocyte count, while the red cells and hæmoglobin are not so rapidly and completely restored.

The splenic irradiation is carried out in carefully graded daily doses, and treatment usually requires about a fortnight for its completion. In the later stages of the disease, when the remissions are much shorter in duration, the comfort of the patient is often better maintained by less intensive treatment at more frequent intervals—every month, or even every fortnight. X-ray treatment is of very doubtful value in the treatment of acute leukaemia, and should only be used for the relief of symptoms such as may arise from pressure from the glandular masses.

*Polycythamia.* Irradiation of the spleen is recommended (Pancoast, Groszlik) in this condition, in which there is a great excess of red cells and hæmoglobin in the blood. In the writer's experience in a

small number of cases, little or no benefit has resulted from this form of treatment. The doses recommended are considerably greater than those that are given in leukæmia.

*Lymphadenoma.* Lymphadenoma only affects the spleen in the late stages of the disease, and by that time the disease has usually become generalised. In such cases, it is usual to treat the spleen, not as an isolated organ but as part of a general regional irradiation of the upper abdomen or of the whole abdomen, as may be required.

## REFERENCE :

KNOX, R. and LEVITT, W. M. *A Text-book of X-ray Therapeutics* (A. & C. Black, Ltd.), 1932.

## CHAPTER V

### THE RESULTS OF SPLENECTOMY AT THE MAYO CLINIC<sup>1</sup>

#### SPLENECTOMIES

April 1, 1904, to January 1, 1931.

	Patients.	Hospital deaths.	Subseq. deaths.	Living
Splenic anemia . . . . .	184	17	72	92
Hemolytic jaundice . . . . .	128	4	11	110
Peruicious anemia . . . . .	62	4	57	1
Hemorrhagic purpura . . . . .	57	4	3	49
Myelogenous leukemia . . . . .	46	3	42	1
Cirrhosis of liver . . . . .	44	8	25	11
Chronic infectious splenomegaly . . . . .	32	8	15	8
Luetic splenomegaly . . . . .	10	1	4	5
Tuberculosis of spleen . . . . .	10	1	3	5
Gaucher's disease . . . . .	9	2	2	5
Lymphocytic splenomegaly . . . . .	8	0	5	3
Ruptured spleen . . . . .	8	2	0	6
Indeterminate hemorrhagic disease . . . . .	3	0	0	3
Polycythemia . . . . .	3	1	1	1
Acute aplastic anemia . . . . .	3	0	3	0
Chronic aplastic anemia . . . . .	2	0	1	1
Acute and subacute septic splenomegaly . . . . .	2	0	2	0
Hemorrhagic cyst of spleen . . . . .	2	0	1	1
Wandering spleen . . . . .	2	0	0	2
Sarcoma of spleen . . . . .	2	0	2	0
Hodgkin's disease . . . . .	2	0	1	1
Reticuloendotheliosis . . . . .	2	0	1	1
Marble bone disease . . . . .	1	0	1	0
Chronic hemolytic anemia . . . . .	1	0	0	1
Indeterminate congenital jaundice . . . . .	1	1	0	0
Multiple sclerosis (Pick's) . . . . .	1	1	0	0
Eosinophilia with splenomegaly . . . . .	1	0	1	0
Neutrophilia with splenomegaly . . . . .	1	1	0	0
Hemangioma . . . . .	1	0	1	0
Infarction of spleen (resection $\frac{1}{2}$ of spleen) . . . . .	1	0	0	1
Secondary splenectomy . . . . .	11	0	4	7
Indeterminate . . . . .	6	1	3	2
Total . . . . .	616	59	261	317

<sup>1</sup> The following valuable tables, which at my request were kindly supplied for this work by Dr. H. Z. Giffin, of the Mayo Clinic, show better than any other records the brilliant results which have been obtained from splenectomy by a group of surgeons of the highest technical skill, working in one of the best equipped and staffed clinics in the world. I wish to express my thanks and appreciation to Dr. W. J. Mayo and Dr. H. Z. Giffin for their great courtesy and co-operation in this matter.—(Euton.)

SPLENECTOMY

<i>Splenic anemia</i>	.	.	.	.	.	.	.	184
Hospital deaths	.	.	.	.	.	.	.	17
Number traced	.	.	.	.	.	.	.	164
Subsequent deaths	.	.	.	.	.	.	.	72
Living	.	.	.	.	.	.	.	92
In good condition	.	.	.	.	.	.	63	
In fair condition	.	.	.	.	.	.	22	
In poor condition	.	.	.	.	.	.	7	

12/31/1908 to 1/1/1934.

SPLENECTOMY

<i>Chronic infectious splenomegaly</i>	.	.	.	.	.	.	.	32
Hospital deaths	.	.	.	.	.	.	.	8
Number traced	.	.	.	.	.	.	.	23
Subsequent deaths	.	.	.	.	.	.	.	15
Living	.	.	.	.	.	.	.	8
In good condition	.	.	.	.	.	.	5	
In fair condition	.	.	.	.	.	.	3	

7/2/1908 to 1/1/1934.

SPLENECTOMY

<i>Hemolytic jaundice</i>	.	.	.	.	.	.	.	128
Hospital deaths	.	.	.	.	.	.	.	4
Number traced	.	.	.	.	.	.	.	121
Subsequent deaths	.	.	.	.	.	.	.	11
Living	.	.	.	.	.	.	.	110
In good condition	.	.	.	.	.	.	97	
In fair condition	.	.	.	.	.	.	11	
In poor condition	.	.	.	.	.	.	2	

6/30/1911 to 1/1/1934.

SPLENECTOMY

<i>Luetic splenomegaly</i>	.	.	.	.	.	.	.	10
Hospital death	.	.	.	.	.	.	.	1
Number traced	.	.	.	.	.	.	.	9
Subsequent deaths	.	.	.	.	.	.	.	4
Living	.	.	.	.	.	.	.	5
In good condition	.	.	.	.	.	.	5	

12/4/1914 to 1/1/1934.

SPLENECTOMY

<i>Hemorrhagic purpura</i>	.	.	.	.	.	.	.	57
Hospital deaths	.	.	.	.	.	.	.	4
Number traced	.	.	.	.	.	.	.	52
Subsequent deaths	.	.	.	.	.	.	.	3
Living	.	.	.	.	.	.	.	49
In good condition	.	.	.	.	.	.	42	
In fair condition	.	.	.	.	.	.	6	
In poor condition	.	.	.	.	.	.	1	

3/7/1923 to 1/1/1934.



## SPLENECTOMY

<i>Gaucher's disease</i> . . . . .	9
Hospital deaths . . . . .	2
Number traced . . . . .	7
Subsequent deaths . . . . .	2
Living . . . . .	5
In good condition . . . . .	4
In fair condition . . . . .	1

1/7/1907 to 1/1/1931.

TABLE I

POST-OPERATIVE LENGTH OF LIFE IN 3 SUBSEQUENT HEMORRHAGIC  
PURPURA DEATHS AND 49 CASES LIVING

<i>Prominent features in cause of death.</i>	<i>Cases</i>	<i>Less than 1</i>	<i>Years of post-operative life.</i>									
			1	2	3	4	5	6	7	8	9	10
Hemorrhage . . . . .	1						1					
Atrophy of liver with jaundice	1							1				
Lobar pneumonia . . . . .	1			1								
Total . . . . .	3			1			1	1				
Living—Good condition . . . . .	42	5	1	1	1	5	4	5	5	5	5	2
Fair condition . . . . .	6		1	2		1				2		
Poor condition . . . . .	1	1										
Total . . . . .	49	6	2	6	1	6	4	5	5	7	5	2

3/7/1923 to 1/1/1931.



TABLE 3

POST-OPERATIVE LENGTH OF LIFE IN 72 SUBSEQUENT SPLENIC ANEMIA DEATHS AND 92 CASES LIVING

Prominent features in course of death	Total cases.	Less than 1	Years of post-operative life.																			
			1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Hemorrhage . . . . .	29	0	5	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Thrombosis . . . . .	9	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Pneumonia . . . . .	5	3	2	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Verrucae of liver . . . . .	4	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Paralytic stroke . . . . .	3	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Leukemia . . . . .	2	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Carcinoma of throat . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Carcinoma of esophagus with metastases to larynx . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Carcinoma of duodenum and pylorus with obstruction . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Malignant tumor kidney . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Following gallbladder operation . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Uteral regurgitation . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Bright's disease . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Summer diarrhea . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Shock following operation for obstruction . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Heart attack following operation elsewhere . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Psychonephritis and uremia . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Nephritis and peritonitis . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Acute ulcerative colitis . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Infection following abrasion on wrist . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Fungus and lung abscess . . . . .	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Unknown . . . . .	5	1	2	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Total . . . . .	72	27	10	8	4	4	3	1	1	1	3	1	1	2	3	1	2	1	1	1	1	1
Living—Good condition . . . . .	63	8	3	2	4	5	3	1	1	1	1	3	1	2	3	1	1	1	1	1	1	1
Fair condition . . . . .	22	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Poor condition . . . . .	7	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
Total . . . . .	92	9	5	4	6	12	1	6	7	7	4	3	4	4	3	3	3	3	3	3	3	3

12,341,008 to 1,119,341

12 34/1908 to 1.1/1934

## CHAPTER VI

### THE SPLEEN AND THE BLOOD-PLATELETS

by

W. HOWEL EVANS

THE blood-platelets may fairly be described as the enigma of the blood stream. They were first adequately described by Bizzozero (1882), and since that time controversy over them, in one way or another, has never ceased. At first, thanks to the physiologists, their existence was doubted and much ingenuity exercised to prove them artefacts. That period has passed, very largely owing to the attention focussed on the platelets by the dramatic results of splenectomy in purpura hæmorrhagica. The platelets are now recognised as an antigenically independent element of the circulating blood; in spite of the considerable accretion of knowledge in recent years, their *precise* relationship to the clinical conditions of hæmorrhagic disease and intra-vascular clotting still eludes us, though it is clearly intimate and of importance.

#### THE BLOOD-PLATELETS : ORIGIN : MORPHOLOGY : FUNCTION

[Detailed reviews are given by *Hittmaier (1927)* and *Mackay (1931)*. The relation of platelets to thrombosis is discussed by *Dawbarn, Earlam, and Evans (1928)*, and *Evans (1929, 2)*. Blood coagulation is exhaustively discussed by *Pickering (1928)*.]

The origin of the mammalian blood-platelet is still under dispute. From all considerations it is clear that the platelet arises from some precursor in the bone-marrow. (*Onke, 1915*; *Bedson, 1923*; *Bedson and Johnston, 1925*.) On morphological grounds *Wright (1910)* suggested that platelets arose from the megakaryocyte of the bone-marrow by cytoplasmic budding; no more convincing theory has yet been advanced in spite of much research. (*Sabin, 1923*; *Bedson and Johnston, 1925*.)

The platelets are, as seen in wet preparations, oval or round bodies

averaging 2-3 microns in size. Even in suitable diluting fluids they tend quickly to become irregular in outline, to assume irregular forms, and even to disappear altogether. In purpura hæmorrhagica and in other conditions where their numbers are small, they show variation in size, large forms up to 5 microns being common. In conditions where there is rapid regeneration, e.g. after splenectomy, etc., small forms not above 1 micron are also present in fair numbers.

By the ordinary Romanowsky stains the cytoplasm appears faintly blue, and the cell-body contains a variable number of purplish granules. These may be collected in the centre quasi nucleus, scattered, or collected in areas at the periphery: it has been stated that the granules vary inversely with the size of the platelets. No satisfactory demonstration of a nucleus has been made, and mitosis has not yet been observed; indirect metabolic evidence does not suggest that the platelets are nucleated. Morphological differences, particularly those relating to the size and number of the granules, have been described as of specific importance in purpura hæmorrhagica. It now seems clear that these differences may be observed in any condition where the numbers are scanty, and it may be categorically stated that the morphology of the platelets as at present understood bears no relation to their functional capacity.

It is established from the studies of Duke (1913) on the survival of transfused platelets in thrombocytopenic conditions, that their life is short, not exceeding two or three days. This estimate of time has found general acceptance.

The usual method of counting is an indirect one using sodium citrate, all direct methods being useless. The area to be pricked, usually the finger, is thoroughly cleansed with ether, immersed in the citrate solution, the prick made through the drop, and a small quantity of blood allowed to flow into some sort of paraffined container. This mixture of blood and diluent is thoroughly mixed, and counted in a hæmoeytometer chamber, a red cell approximate to platelet ratio being thus obtained. A red cell count made at the same time enables the total number to be calculated. According to the majority of observers, the figures regarded as normal usually range between 250,000 and 400,000. (See Mackay, 1931.)

As already briefly mentioned, the life of the platelets in the blood stream is short, and all the available evidence points to their removal by the reticulo-endothelial system, and particularly by the spleen. Direct evidence of intracellular destruction is not actually forthcoming but the indirect evidence can hardly be gainsaid. Thus it is easy, by

teasing a fragment of spleen in citrate solution, to demonstrate large masses of platelets. It has been observed (Myers, Maingot, and Gordon, 1926; Galloway, 1931-2) that the blood from the splenic artery contains more platelets than that from the vein, though it must also be said that Holloway and Blackford (1924) experimentally, and Cumings (1933) in human cases, failed to find this difference.

It has long been known that splenectomy causes a prompt and considerable increase of the platelets in the circulating blood, and similarly, if the reticulo-endothelial system is blockaded by dyes (Bedson, 1926) a similar rapid increase of comparable magnitude is evoked. The blockade increase can also be obtained in animals whose blood has been allowed to return to normal after splenectomy, suggesting that after the removal of the spleen the remaining part of the reticulo-endothelial system has taken over its functions, but that, as might be expected, its capacity to deal with platelets can again be upset by blockading. Just how the normal regulation of the platelets is effected is not clear; probably, as suggested by Bedson (1926), the reticulo-endothelial cells of the spleen being essentially phagocytic take up and destroy platelets that have become effete. When the spleen is removed there is a temporary flooding of the blood stream as production exceeds removal, until the remaining reticulo-endothelial tissue takes over.

To digress briefly, it is by no means easy to find out who first recognised the platelet increase after splenectomy. After some search of the earlier literature, it would appear that the credit should go to Bond (1896). In two cases after splenectomy—a rare operation in those days—he noted in stained films large masses of granular protoplasm which he suspected to be an aggregation of blood-platelets. Collected in Hayem's fluid after pricking the finger through a drop of that fluid, the granular masses were still present, but if the blood was received into dilute osmic acid—a fixative—there were no masses, but instead many discrete platelets. He noted that the granular masses and large numbers of platelets were present in the blood of three patients after splenectomy up to four months after the operation, but the granular masses could not be found in a case fifteen months after splenectomy. As Bond at the same time found the platelets similarly numerous in "advanced phthisis" and "leucocythæmia," both conditions in which high platelet counts are known frequently to occur, it is clear that we can admit the accuracy of his observations. He remarks: "The fact that these masses, with their antecedent increase in number of platelets, gradually increase after splenectomy . . . is clear proof

that the blood-platelets are not formed entirely or even chiefly in the spleen."

Whether this attribution be correct or not, the rise of the platelet count after the then-fashionable operation of splenectomy for pernicious anæmia was observed by Eppinger and his co-workers in Vienna before the War. (Eppinger, 1913; Von Decastello, 1914.) An American, Moffitt (1914), mentions that it was pointed out to him when he visited Vienna, and it was quickly known in America at any rate. Lee, Minot, and Vincent (1916) refer to it, and describe three cases of thrombosis or phlebitis arising after splenectomy for pernicious anæmia at a time when they describe the platelet count as being "very largely increased." Minot, Denny, and Davis (1916) mention a fatal case of "widespread" thrombosis after splenectomy for pernicious anæmia. Ottenberg and Rosenthal (1917) mention a count of 970,000 platelets in a case of pernicious anæmia after splenectomy.

#### THE COURSE OF THE HUMAN BLOOD-PLATELET COUNT AFTER SPLENECTOMY

Despite the fact that much interest has been taken in this subject recently, there are very few detailed records. I have found detailed charts of one case of acholuric jaundice reported by Dyke (1925); eight cases of splenic anæmia reported by Rosenthal (1925); eleven of my own series (1928); three by Brock and Rake (1929), and three by Galloway (1931). A number of figures are mentioned by Spence (1928) in his summary of the purpura cases. Lesehke and Wittkower (1926) give detailed figures of many purpura cases, and a few maxima in a variety of other conditions.

From these cases, if we omit for the moment the purpura ones, as the figures are somewhat capricious, it may be stated generally that following splenectomy for ruptured normal spleen, acholuric jaundice and splenic anæmia, there is a rapid elevation of the count, manifest in 24 hours, and progressing to a maximum in somewhere between two and three weeks. Then there is a much slower fall, an approximately normal level not being reached for about 90 days or even longer. Thereafter the platelets tend to remain rather on the upper side of normal, subject to variation such as might be met with in normal persons (infections, parturition, etc.). This has been my personal finding in a total of twelve cases other than purpura hæmorrhagica observed for five years or over; the findings of the other observers mentioned seem in fairly close agreement.

The height of the curve varies ; it is lowest as a rule in the purpura cases, and in the other types it appears to reach an average level of about 1,000,000 (maximum of observers quoted 1,600,000, Rosenthal ; minimum, 624,000, Galloway).

In purpura hæmorrhagica, as is now well known, the behaviour of the platelets is more irregular. Generally speaking, the rise would appear to be more rapid ; in two of my cases the count was quadrupled in six hours ; similar rapid increase has been noted by Myers, Maingot, and Gordon (1926). In another case, however, there has been no appreciable rise after seven years, although the patient has remained free from purpura.

We may therefore conclude that following splenectomy for causes other than purpura hæmorrhagica the usual course is that the blood-platelets quickly rise, reach a maximum somewhere in the third week, have fallen to figures nearly but usually not quite normal in three months, and thereafter remain on the upper level of normal, subject to fluctuations from ordinary causes.

#### THE FUNCTIONS OF THE BLOOD-PLATELETS

The most obvious and important functions of the platelets appear to be in relation to blood clotting, whether extra- or intra-vascular, and to hæmorrhagic disease.

It is generally conceded that the platelets are at least of great importance in extra-vascular clotting, and that by their disintegration they provide material which in some way or other is intimately concerned in the production of thrombin or fibrin ferment. Blood deprived of platelets by various methods will not clot spontaneously, but does so on addition of platelets. (Cramer and Pringle, 1913 ; Lee and Vincent, 1914 ; Wright and Minot, 1917 ; Gichner, 1927.) Tait and Burke (1926) from direct dark-field observation considered that they saw fibrin threads arise in the track of spherules shot off from disintegrating platelets, and went so far as to suggest that the unruptured thrombocyte might be regarded as prothrombin. It has also been considered that the delay in coagulation in hæmophilia is due to qualitative defects in platelets present in normal numbers, the prothrombin being slowly liberated.

Pickering (1928), however, would deny the primary role of the platelets, considering that the initial step in clotting is a physical change in the colloidal equilibrium of the plasma, and Roskam (1927) and Feissley (1933) think that platelets are only active in virtue of the



plasma absorbed on the surface. It is, of course, a puzzling thing that in purpura hæmorrhagica (and other thrombocytopenic conditions), in spite of the virtual absence of platelets, the clotting time in vitro is normal or nearly so; it is also known that in animals bled at the height of digestion, if the plasma be deplateletised by filtration through charcoal or a Berkefeld filter (Cramer and Pringle, 1913), it still clots rapidly. Mills (1927) has also shown that after protein meals the clotting time in vitro is shortened and the blood-platelets show a more marked tendency to clump. (In respect of post-operative thrombosis he therefore suggests a low protein diet.) It would thus appear that blood can clot in the presence of minimal numbers of platelets, and that the products of protein digestion in some way accelerate the process, whether platelets be present or not. It may be that this is normally a subsidiary mechanism; clearly we are not yet in possession of all the factors concerned.

"In intra-vascular clotting the conditions and mechanism are different. Welch and Aschoff have shown that in the so-called static thrombi found in the veins of the lower limb, the cerebral sinuses and the auricles, the first step in the building of a thrombus is the adhesion of masses of platelets to the vessel wall. Progressive deposition of platelets goes on but fibrin does not appear in the mass. Finally, when sufficient slowing of the blood stream has occurred, the peripheral blood column rapidly clots, forming a red portion of the clot consisting of a mingled mass of red cells, white cells, platelets, and fibrin similar to the extra-vascular clot. The essential and primary fact in intra-vascular clotting is an alteration of the physical relationship between the vessel wall and the platelets. What determines the adhesion of the platelets is not clearly known. While injury to the intima may certainly cause it, such injury has never been convincingly shown in the static thrombi mentioned. The chief factors discussed by Welch and Aschoff are the retardation of the blood flow and increase in the number of platelets." (Dawbarn, Earlam, and Evans, 1928.) The above description of the method of formation of the intra-vascular clot has received experimental proof at the hands of Rowntree and Shionoya (1927) in experiments with an artificial circulation.

#### THE BLOOD-PLATELETS IN RELATION TO CLINICAL THROMBOSIS

The factor of fluctuations in the platelets in the circulating blood has received some attention recently, because an increase in the platelets has been observed not only after the specific operation of splenectomy,

but also after any operative procedure, parturition, and fractures. (Hueck, 1926 ; Dawharn, Earlam, and Evans, 1928 ; Willinsky, 1930 ; Galloway, 1931 (1) ; Brock, 1933.)

The Liverpool work on this subject arose from the work of Rosenthal (1925) on splenic anæmia, later to be discussed. Rosenthal correlated the fairly frequent occurrence of thrombosis after splenectomy for splenic anæmia with a persistent elevation of the platelet count. This work appeared while the Liverpool workers were casting about for some new method of attack on the old problem of post-operative thrombosis and embolism. It was then found that after operations the platelets showed a considerable increase, usually beginning about the sixth day, reaching a peak on the tenth day, remaining high for a further few days, and becoming normal about the end of the third week. The degree of this platelet reaction varied ; it was in general proportion to the severity of the operation, but varied in different individuals with the same operation. It was shown to be unassociated with age, rest in bed, anæsthetics, loss of blood, or sepsis. It was pointed out that the period of the platelet increase, namely, the second week after operation, was precisely that period of time during which post-operative thrombosis and embolism commonly occurred. The same platelet reaction was observed after parturition, especially by Cæsarean section, and fractures. Clotting time *in vitro* seemed to show a tendency to shortening during the platelet reaction. It is clear that the platelet reaction does not account for the *inception* of clotting ; if for any other reason clotting begins, its spread will obviously be helped by a thrombocytosis. A further factor in favour of thrombosis at this time is the increase of fibrinogen in the blood shown to occur by Allen (1927) ; while it is also known at any rate in pregnancy and tuberculosis that there is a reduced suspension stability of the blood. Hence, given all or most of these factors, there is a definite bias in favour of thrombosis, and the fact that it is relatively so rare after operation, etc., is clear evidence of the adaptability of the defensive mechanism.

It appears that the factor underlying the platelet reaction is the absorption of break-down products following the inevitable trauma. As regards infections, Reimann (1927) has demonstrated the platelet reaction in convalescence from pneumonia, i.e. during the period of absorption of the exudate, and Broek (1933) has called attention to the frequency of thrombosis in advancing phthisis ; the occurrence of high platelet counts and also of increased sedimentation rate in this condition are well known.

This type of platelet reaction is evidently not the same as that after splenectomy, which is due to the ablation of the bulk of the platelet-controlling mechanism; it is of shorter duration and usually much less marked, though some few of our post-operative counts reached 1,000,000. It can only be ascribed to a stimulation of the marrow by unknown break-down products, and in this sense, i.e. that of a physiological response to a stimulus, would appear to be inevitable; it would not appear that there is any prospect of preventing or modifying it in any way. It is therefore apparent that the post-splenectomy curve is the resultant of a specific and a non-specific stimulus.

It should be mentioned here that Evans and his co-workers have never suggested that there is any direct relationship between the actual numbers of the platelets and the incidence of thrombosis. Many of the post-splenectomy series and some of the post-operative and post-partum figures showed very high counts without suffering thrombosis. It was contended that the time relations of the platelet reaction and the observed clinical thrombosis were in such close agreement that the platelet reaction must be regarded as in some way or other of great importance. As illustrating that thrombosis does not depend on the mere height of the platelet curve, an unpublished case personally observed may be quoted. A multipara aged 47 was submitted to operation for a supposed ovarian cyst which proved to be a large spleen. It was removed; no pre-operative counts were made. The post-operative platelet counts from the 10th to the 365th day were:

<i>Day after operation.</i>	<i>Number of platelets.</i>
10th	1,850,000
13th	1,330,000
14th	1,289,000
18th	1,420,000
24th	932,000
29th	1,021,000
115th	949,000
365th	1,020,000

This patient died from lobar pneumonia three months after the last count; no thrombosis occurred at any stage in spite of the persistent thrombocytosis.

## PLATELETS IN RELATION TO SPLENECTOMY

To return to Rosenthal's work on splenic anæmia. It seems clear that this is the chief type of splenic disorder in which thrombotic complications are common after splenectomy. Apart from the four cases occurring after splenectomy for pernicious anæmia already mentioned, and one case after splenectomy for purpura hæmorrhagica mentioned by Leschke and Wittkower (1926), I have not found any records in other conditions. Fatal mesenteric thrombosis, ascribed to an extension from the ligatured splenic vein, is mentioned as an exceedingly dangerous sequel by Wilkins (1923); also by Mayo (1925), Rosenthal (1925), Evans (1929, 1), Pemberton (1931), and Bryce (1932). Rosenthal also mentions systemic thromboses and Pemberton pulmonary embolism. The danger of post-operative thrombosis in splenic anæmia is, therefore, a very real one.

At the present moment it must be admitted that we are very much in the dark as to the underlying processes involved in "splenic anæmia." McNee's (1931) important investigations lead to the hope of eventual clarification, and at the same time point to the probability of more than one type within the group, with possibly differing pathogeny; as yet our knowledge does not permit of any clear pre-operative distinction. It is clear that sclerosis and thrombophlebitis of the splenic and portal veins is of frequent occurrence in "splenic anæmia," and it is tempting to think that such changes may be an important factor predisposing to mesenteric thrombosis. Pemberton's recognition of oesophageal varices by X-rays and oesophagoscopy points to a possible means of pre-operative assessment of the extent of the obstruction in the portal system which may prove of value. There can be no doubt that a *successful* splenectomy is the best, if not the only hope for the sufferer from splenic anæmia; equally, he seems exposed to a definite risk of portal, or mesenteric, or other thrombosis which may be fatal. The problem is, can this risk be constantly associated with any specific pathological picture? For instance, is it specially likely to occur in the presence of gross thrombophlebitic changes in the portal drainage area? And have we any pre-operative means of estimating the probability of thrombosis?

In this connection Rosenthal's work naturally excited great interest; it has already been mentioned how it gave rise to the discovery of the platelet reaction in other conditions. Disregarding pathological anatomy, Rosenthal suggested that two types of splenic anæmia might be differentiated on the pre- and post-operative

behaviour of the platelets. In one, which he called the thrombocytopenic, the platelets were low before splenectomy, described a "normal" curve afterwards, and the results were good. These patients showed subjective and objective evidence of hæmorrhagic tendency.

In the other, the thromboeythæmic group, there was usually a normal pre-operative count, but after removal of the spleen the platelets remained persistently elevated (one million and over) for months and even years. One of the cases died with widespread portal thrombosis, and three suffered non-fatal systemic thromboses. This group showed hæmatemesis, but no evidence of general hæmorrhagic tendency.

It is clear that the first—thrombocytopenic—type exists, and probably is the commoner. Ziegler (1914) suggested that in splenic æmia there was an associated hæmorrhagic diathesis, and Frank (1917) referred to "Splenopathie mit Hypoleukie und Thrombopenie." In these cases there may be hæmorrhages from many sites, e.g. epistaxis, bleeding gums, menorrhagia, and purpura. In a series of cases reported in 1929, I found examples of all these, and noted in some cases prolongation of the bleeding time, and slight defect in the clotting time, in all these respects being in agreement with Rosenthal. It is my practice to make several platelet counts in order to be sure that the thrombocytopenia is permanent.

Of ten cases of thrombocytopenic type personally observed after splenectomy, none suffered thrombosis. Of five patients observed for three years or over, three have remained free from symptoms and have maintained normal platelet counts. One patient died three years after operation with recurrent hæmatemesis; another had a single hæmatemesis one year after operation. Both these patients showed normal or somewhat elevated platelet counts when seen after the hæmatemesises. Hæmatemesis alone, without bleeding from other sites, may be due to portal obstruction—which may be progressive in spite of splenectomy—and is not necessarily evidence of general hæmorrhagic tendency. Four more cases (all under one year) described normal platelet curves and remain well so far.

The only case of thromboeythæmic type in my experience died a month after splenectomy with a persistently elevated platelet count, and at autopsy it was found that thrombosis had spread from the splenic pedicle into the superior mesenteric vein with gangrene of a loop of small bowel; the portal vein remained patent. As this result was in agreement with Rosenthal's findings, it seemed reasonable to suggest that the thrombocytopenic type offered the better operative

risk; and that in the *thrombocythæmic* case operation entailed considerable risk, and should only be undertaken in full realisation of that risk. (Evans, 1929, 1).

Recently Bryce (1932), in discussing splenectomy and thrombosis, reported two cases which do not clarify the problem. The first case apparently conformed to the recognised syndrome of splenic anaemia; there was splenomegaly with ascites and (at autopsy) fine hepatic cirrhosis. There was a history of recurrent hæmatemesis, and the pre-operative platelet count was 38,000. This patient died on the tenth day after splenectomy; autopsy showed recent thrombosis spreading from the splenic vein into the portal vein.

The second case, as Bryce pointed out, was of more doubtful diagnosis. The spleen was large; the blood picture showed anaemia with apparently macrocytosis. The case is unfortunately scantily reported, and no pathological report of the spleen is given. Observed over five months the platelets fluctuated between 285,000 and 136,000. During this same period systemic thromboses occurred in the veins of both legs. Splenectomy was performed as a last resort, with misgiving on account of this thrombotic tendency; up to four months later the expected thrombosis had not occurred, and the platelet count showed fluctuations within a range which could be considered "normal." (Maximum, 700,000.) This case cannot be accepted as a clear-cut thrombocythæmic splenic anaemia; it is possible that the systemic thromboses had a purely local cause, though the pre-operative apprehension of thrombosis can be readily understood.

The first case, however, is important in showing that even an apparent thrombocytopenic case may not be immune from fatal thrombosis. It is clear that we have not sufficient data to be too dogmatic on the pre-operative criteria for the selection of cases. Bryce points out the complete lack of knowledge as to the possible correlation between the platelets and the incidence of thrombophlebitic changes in the portal system. This is a matter for the future, and it behoves all concerned with cases of "splenic anaemia"—physician, surgeon, and pathologist alike—to study their cases in the fullest possible detail.

Meanwhile, it still seems reasonable to think that the cases showing subjective and objective general bleeding tendency associated with persistent thrombocytopenia offer the best operative risk. Even so, it is not possible to postulate complete cure, as is shown by the frequency of later recurrences of hæmatemesis, which may be due to progressive changes in the portal area and liver, the prime agent of which is

unaffected by the splenectomy. Further, if future events fail to confirm the impression that thrombosis is a more or less inevitable sequel to splenectomy in the thromboerythæmic case, we must be prepared to modify our opinion and not refuse the benefit of operation to such patients. In this connection, McNee (1931) and Bryce (1932) have suggested ligation of the splenic artery in place of excision of the spleen. This would have the advantage that there would be no starting-point of thrombosis in the splenic vein; it might also be advisable in those cases presenting formidable adhesions. It is an experiment which would be of great interest; the weak point seems to be that the patient with an enormous spleen would not be relieved of his tumour.

A point which seems elementary, but which is nevertheless frequently overlooked, is that whatever the type selected for operation, every effort should be made to get the patient into the best possible condition for operation. Many cases are very anæmic when first seen, but can be remarkably improved with rest, transfusions, and full doses of iron, as in the intervals between the hæmorrhages the improvement of the anæmia may be rapid. (Davidson, 1934.) It is reasonable to assume that if the patient's general condition be improved, his protective mechanism against clotting should also be improved.

There is little to be said about the risks of post-operative thrombosis in conditions other than splenic anæmia, as they seem to be quite rare. The cases after splenectomy for pernicious anæmia are now historical curiosities; the odd case in purpura hæmorrhagica does not vitiate the excellence of the results in the chronic cases. In purpura, indeed, the chief risk seems to be the opposite, i.e. failure to arrest the hæmorrhagic tendency. In other conditions such as acholuric jaundice, rupture, cysts, syphilitic splenomegaly, etc., the risk is probably just that which might be expected after any major operation, and need never be regarded as a deterrent. The fact that thrombotic sequels are so rare, in spite of the high platelet levels normally found, is further evidence that thrombocytosis *alone* is not sufficient to induce thrombosis.

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## CHAPTER VII

### SPLenic DISEASES OF SURGICAL IMPORTANCE

by

RODNEY MAINGOT

(1) *Aneurysm of the Splenic Artery.* Only a few cases of aneurysm of the splenic artery have been recorded. Most of these were unrecognised during life and only discovered at post-mortem examination. Rupture frequently occurs, giving rise to a rapid, and often fatal, internal hæmorrhage. Literature only reports a few cases where a pre-operative diagnosis was made or where operation—usually splenectomy—was performed with success.

Sometimes arterio-sclerosis, new growths of the body or tail of the pancreas, embolism, syphilis, or trauma have been held to be responsible factors, but an investigation in the majority of cases has disclosed no cause for the disease.

The rarity of the condition is evidenced by the fact that of all aneurysm cases revealed at autopsy aneurysm of the splenic artery is found in only about 5 per cent.

The symptoms may be vague, and mild epigastric upset may be the sole complaint until violent, sudden, and copious hæmorrhage proclaims the presence of a grave internal catastrophe. A pulsatile tumour situated to the left of the mid-line in the epigastrium, a bruit heard with the stethoscope, and splenomegaly are unusual findings. Rupture of a splenic aneurysm may give rise to signs and symptoms similar in every respect to those of spontaneous rupture of the spleen.

The treatment is ligature of the splenic blood-vessels at a site proximal to the aneurysmal dilatation, followed by removal of the aneurysmal sac and the spleen.

(2) *Splenic Abscess.* Abscess of the spleen is rarely seen in cold and temperate climates. The frequency with which the spleen escapes abscess formation in infections in general is probably linked up with its abundant content of phagocytic cells and its normal function as the scavenger of stray organisms entering the blood stream. Abscess may,

however, develop during the course of any chronic or acute blood infection and in certain parasitic diseases. It may thus occur :

- (a) In acute specific infectious diseases, the commonest being enteric fever.
- (b) In pyæmia ; here multiple abscesses are usually present.
- (c) In infective endocarditis. In this condition the breaking down of a septic infarct leads to abscess formation.
- (d) In injuries.
- (e) In pneumococcal septicæmia.
- (f) In certain staphylococcal infections, e.g. carbuncle and furunculosis.
- (g) In infections associated with gangrenous appendicitis, acute salpingitis, and any infection of a grave character.
- (h) In malaria.
- (i) In dysentery.
- (j) In relapsing fever.
- (k) In suppurating hydatid cyst of the spleen.

The possibility of a splenic abscess must not be overlooked in cases of infection and in diseases peculiar to tropical and sub-tropical climates, accompanied by high temperature, repeated rigors, abdominal pain and tenderness, and local signs suggestive of suppuration in the left hypochondriac region and in the base of the left lung.

In the early stages of splenic abscess the symptoms are vague and anomalous, there being no characteristic feature until the abscess has attained a considerable size and has started to stretch the capsule of the spleen or has actually burst and produced a localised peritonitis in the region of the splenic bed. The condition being rare, the possibility of its occurrence is often forgotten.

In all well-established cases the patient looks gravely ill, and there may be, and often is, anorexia, occasional bouts of vomiting, diarrhoea, asthenia, rapid loss of weight, rigors, and high temperature. Pain in the upper left quadrant of the abdomen is constant and severe, and respiratory movements of the left side of the chest may be restricted and painful. When the abscess is situated in the upper pole of the spleen, infection may spread through the diaphragm and give rise to pleurisy or empyema. The signs and symptoms in such cases at once suggest subphrenic suppuration or empyema. When the lower pole is principally involved, tenderness below the costal margin may be

exquisite, guarding of the muscles of the left half of the abdomen may be marked, but especially so in the left hypochondrium and left post-renal angle, and there may be œdema and pitting on pressure in this area.

A splenic abscess may burst :

- (a) Between the convex surface of the spleen and the diaphragm, and become localised as one form of subphrenic abscess.
- (b) Into the general peritoneal cavity, and give rise to a diffuse peritonitis.
- (c) Into the left pleural cavity, and produce an empyema.
- (d) Through the abdominal wall.
- (e) Into the stomach, colon, or jejunum.

The following investigations should always be made :

(a) Examination of the blood. This will include a leucocyte count, blood culture, Widal test, etc. In all cases of splenic abscess there is leucocytosis, even when the patient is suffering from typhoid—the commonest cause of a splenic abscess.

(b) Splenic puncture. A long needle is attached to a 10 cc. Record syringe, and plunged into the spleen through the tenth intercostal space in the mid-axillary line. During the puncture and subsequent aspiration the patient is instructed to hold his breath. The aspiration is performed as quickly as possible, as respiratory movements will cause movement of the needle with possible damage to the spleen. The material withdrawn is submitted for pathological investigation.

As a method of investigation splenic puncture is not to be lightly undertaken, as the risk of spreading infection to adjacent areas is not negligible.

(c) Radiography. A straight X-ray will often show that the left cupola of the diaphragm is elevated and immobile on screening, and a suspicious shadow in the splenic region may indicate the position of the pus.

### *Treatment.*

- (a) Drainage of the abscess ; or
- (b) Splenectomy.

Splenectomy is the best treatment if it can be performed with safety. If, however, the spleen is firmly bound down by dense adhesions

and suppuration is extensive, the technical difficulties alone will preclude removal of the organ, and drainage will have to be substituted. Again, if there are localising signs, such as redness of the skin and œdema in the subcostal region, if on splenic puncture pus has been located, or if the patient's general condition is such as to prohibit an exploratory laparotomy, the abscess should be drained.

The position of the incision and the method of approach will vary according to the site of the abscess. For instance, when the upper pole is involved and there is a possibility of intra-thoracic suppuration, the posterior transpleural route will be the one of choice; whereas if the abscess is pointing, the incision should be made directly over it; if pus is found, a drainage-tube should be introduced and secured to the abdominal wall by a stitch.

In cases of doubt the incision should be made from the tip of the twelfth rib and extended upwards along the under-border of the eleventh rib. When the incision has been sufficiently deepened the abscess cavity should be sought for and cautiously explored with the finger.

(3) *Cysts of the Spleen.* These may be classified as follows:

(a) Primary non-parasitic cysts.

(i) single.

(ii) multiple.

(b) Dermoid cysts.

(c) Parasitic cysts—hydatid.

From a surgical point of view cysts of the spleen are relatively unimportant.

*Single cysts* are exceedingly rare and occur chiefly in young adults. Such a cyst increases slowly in size, until eventually it may assume enormous proportions and may be mistaken for a hydronephrosis, an ovarian cyst, or a cyst of the tail of the pancreas. The majority of these cysts are due to trauma—hæmorrhagic cysts of the spleen, but in certain cases they may possess a wall of fibrous tissue and may be lined with columnar or cubical cells. These cysts may be filled with clear, serous, or creamy viscid chocolate-coloured fluid, streaked with yellowish flakes of cholesterol. Clinically they cause little or no inconvenience apart from the pressure occasioned by their increase in size.

There are two varieties of *multiple cysts* of the spleen:

(a) Polycystic, i.e. the spleen is riddled with numerous small cysts, with a similar accompanying condition of the kidneys and liver. The

number and size of such cysts is always much smaller in the case of the spleen than in the primarily affected organ, the kidney.

(b) A condition in which small multiple serous cysts occur and are found accidentally at exploratory laparotomy or at autopsy. They are of no importance, probably congenital, and are not associated with polycystic disease.

*Dermoid cysts* of the spleen are rare and call for no special comment.

*Hydatid cysts* may occur in any part of the body, and are occasionally found in the spleen.



FIG. 422.—LARGE SOLITARY HEMORRHAGIC CYST OF THE SPLEEN.  
(Museum, University College Hospital.)

Exploratory puncture should not be performed where the presence of such a cyst is suspected. Splenectomy is the ideal operation if there are no adhesions, or where, if present, they can be dealt with without difficulty and without danger of rupture of the spleen. If extensive adhesions render mobilisation of the spleen impossible the wisest and safest procedure is to anchor the cyst to the parietes, and then, after injecting it with formalin, to incise it and drain away its contents.

(4) *New Growths of the Spleen.* These are also strikingly uncommon and may be classified as :

(a) Innocent.

(i) Fibroma.

(ii) Lymphoma.

(iii) Hæmangioma.

(iv) Lymphangioma. (ii), (iii), and (iv) are often difficult to distinguish from true sarcomata.

(b) Malignant.

(i) Primary.

(ii) Secondary.

Unlike the liver, the spleen is very rarely the seat of secondary carcinomatous deposits. It is difficult to explain the reason for this, although it is conceivable that malignant cells become "phagocytosed" before they can multiply, much in the same way as the red blood-corpuscles are removed from the circulation by the splenic pulp. Primary malignant tumours are seldom seen, but when they occur they are usually sarcomatous.

Pathologically it is sometimes difficult to determine whether these sarcomata are, in fact, lympho-sarcomata or endotheliomata.

It is very doubtful if primary carcinoma of the spleen ever occurs.

Up to 1929 Tasker Howard collected 116 cases of *primary sarcoma of the spleen*. The ultimate results of splenectomy for sarcoma were shown to be unsatisfactory as the majority died of metastases within eighteen months. Some unexpectedly good results did, however, follow removal of the spleen.

(5) *Tuberculosis of the Spleen.* This may be *primary* or *secondary*. Tuberculosis of the spleen is often seen in cases of general tuberculosis,

the spleen in such cases usually being moderately enlarged and studded with numerous grey miliary tubercles, most marked beneath the capsule, which may, in prolonged cases, grow by fusion into small discrete yellow areas of caseation. Seldom is there time for softening and liquefaction to occur. At autopsy healed tubercles are found in the organ comparatively frequently in cases in which tuberculosis has occurred elsewhere in the body.

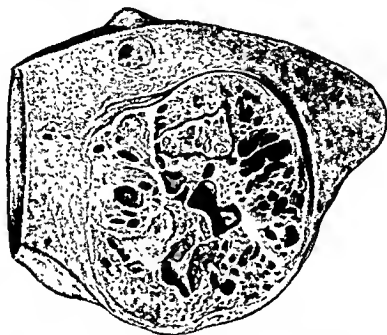


Fig. 473—ANGIO-SARCOMA OF THE SPLEEN. PART OF A SPLEEN, THE SECTION OF WHICH SHOWS A WELL DEFINED CIRCULAR GROWTH MEASURING 2½ INCHES IN DIAMETER AND CONSISTING OF SOFT FRIABLE TISSUE CONTAINING NUMEROUS LARGE BLOOD SPACES. A SECOND SMALLER NODULE OF GROWTH FORMS A PROJECTION ON THE EXTERNAL SURFACE.

(Museum, Royal College of Surgeons.)

*Primary tuberculosis of the spleen is rare. There may be one solitary abscess, but it is more usual to find the spleen riddled with small breaking-down caseous masses. A few cases of primary tuberculosis of the spleen treated by splenectomy have been described. Polycythæmia has been a marked feature in some of these cases.*

(6) *Syphilis of the Spleen.* The spleen may show evidence of infection with *spirochaeta pallida* in :

- (a) Congenital syphilis.
- (b) Acquired syphilis.

In congenital syphilis there may be a diffuse splenomegaly due to interstitial splenitis, or multiple gummata may be present.

In the secondary stage of syphilis the spleen is frequently enlarged as a result of diffuse interstitial inflammation of the organ. The enlarged spleen is seldom felt, but in this connection it should be remembered that the organ will not be palpable until it is at least twice its normal size. The degree of splenic enlargement should not be gauged by percussion, as this alone is always an unreliable method of diagnosis.

In tertiary syphilis the spleen may be enlarged as the result of interstitial changes or the formation of localised gummata. In this stage the signs, symptoms, and blood pictures may closely mimic those of splenic anaemia; in fact, it may be impossible to differentiate between the diseases until the spleen has been removed and subjected to microscopical investigation.

Too much reliance must not be placed upon serological tests in making a differential diagnosis between splenic anaemia and syphilis of the spleen, as the Wassermann reaction may, in an undoubted case of tertiary syphilis, be either positive or negative. The blood picture in cases of gumma of the spleen usually shows a secondary anaemia.

*Treatment.* Anti-syphilitic measures in cases of syphilitic splenomegaly should be persevered with for as long as possible. In anomalous cases of splenic enlargement which do not conform to any known type, a short intensive course of anti-syphilitic treatment should also be given a trial, as some of these cases may respond to such measures.

In intractable cases, and especially in those where the Wassermann reaction is persistently positive in spite of a carefully planned regime of treatment, and in which the patient, in addition to suffering from splenic enlargement, is anaemic, debilitated, and obviously retrogressing, splenectomy should be undertaken as advised by W. J. Mayo. Mayo has recorded 10 such cases, 5 of which showed a remarkable improvement after splenectomy, the Wassermann reaction, which before was persistently positive, speedily becoming negative, and the patients being restored to apparent good health.

(7) *Malarial Splenomegaly.* In the tropics malaria is by far the commonest cause of splenic enlargement. Splenectomy has been performed for malarial splenomegaly in a considerable number of cases



in the past, the reasons for advocating removal of the spleen being as follows :

(a) The large size of the organ and its liability to injury. The mortality following operation for ruptured malarial spleen is over 60 per cent.

(b) Enlargement of the spleen associated with undue mobility. Here excision was undertaken to forestall torsion of the pedicle, or the tendency of the spleen to rupture, either spontaneously or as the result of some injury.

(c) As advocated by Jonnesco, to remove a possible breeding ground for the parasites in intractable and long-standing cases which show no response to medicinal treatment.

Many "malarial spleens" which have failed to respond to anti-malarial treatment are now known to have been due to kala-azar. It must be a very rare thing indeed now to have to remove a spleen for malarin alone, except of course in cases of rupture, as medicinal treatment, combined with radiation, will often prove successful.

The mortality for splenectomy will be found to vary within wide limits, according to whether the organ is fixed by adhesions or whether it is mobile ; in the former condition the mortality is over 60 per cent, whereas in the latter it is only about 5 per cent.

(8) *Wandering or Floating Spleen.* This is a rare condition, as shown by W. J. Mayo, who reports that of 646 splenectomies performed up to 1934 at the Mayo Clinic only two were for wandering spleen. The causes of this condition may be :

(a) Congenital. Here the ectopic position of the spleen may be due to absence of the supporting phrenico-colic ligament. Occasionally the spleen may be situated in a retroperitoneal position, or in the thorax as in congenital diaphragmatic hernia.

(b) Injury. As a result of direct or indirect injury the suspensory ligaments of the spleen may tear or become stretched and elongated.

(c) Splenomegaly. Here the weight of the spleen may stretch the supporting peritoneal folds and the organ become unduly mobile.

(d) Visceroptosis. It is very doubtful, however, whether this is a predisposing cause.

The spleen may wander :

- (a) Into the thorax in cases of diaphragmatic hernia.
- (b) Into any part of the abdominal cavity, showing a predilection for the left iliac fossa.
- (c) Into the hollow of the pelvis : or
- (d) Even into a large hernial sac.

In the migrations of the spleen the following complications may occur :

- (a) Twisting of the pedicle.
- (b) Engorgement and consequent enlargement of the spleen.
- (c) As the result of (b) hæmorrhagic cysts may form in its substance and the spleen may undergo cystic degeneration.
- (d) Atrophy of the spleen.
- (e) Other organs may become involved as the result of pressure or of twisting.
- (f) "Dislocation." The movable spleen is said to be "dislocated" when as a result of localised chronic peritonitis it becomes fixed in an abnormal position and remains so.

Clinically there is usually no difficulty in recognising the condition, as the wandering spleen has a characteristic shape and generally lies in a superficial position. It can often be coaxed back into its original position in the abdominal cavity. Occasionally, however, it may be mistaken for :

- (a) A uterine fibroid with a long pedicle.
- (b) An ovarian cyst or growth of the ovary.
- (c) A pregnant uterus.
- (d) A polycystic kidney.
- (e) Hydronephrosis.
- (f) Carcinoma of the stomach or of the splenic flexure.

The symptoms are usually *mild* and may include a dull ache in the epigastrium from engorgement, flatulent dyspepsia from the dragging on the stomach, pressure effects, and a variety of symptoms akin to those of movable kidney.

When torsion of the pedicle occurs the symptoms are *acute* and may resemble those of a severe abdominal catastrophe. There is sudden

acute agonising abdominal pain, prostration, vomiting and distension; in fact, a combination of symptoms frequently seen in a case of twisted ovarian cyst or acute hæmorrhagic pancreatitis. A diagnosis may be impossible until the abdomen has been opened.

The acute symptoms are due to the thrombosis of the blood-vessels of the pedicle which have long been twisted, rather than to rotation of the pedicle itself which, as a rule, is a slow process. As soon as the blood-vessels in the pedicle thrombose, the spleen becomes a massive infarct, and localised peritonitis ensues. It may, in addition, become gangrenous, slough, or produce a localised abscess or generalised peritonitis.

*Treatment.* Splenectomy is the approved treatment for movable spleen with complications, such as torsion of the pedicle, and is also recommended by most authorities for uncomplicated cases. A few mild cases have been treated with abdominal supports or by *splenopexy*. We are, however, averse to these methods and would recommend splenectomy as the operation of choice in all such cases as soon as the condition is recognised, as it is usually only a matter of time before complications develop.

(9) *von Jaksch's Disease—Pseudo-Leukæmic Anæmia of Infants.* It is now generally accepted that von Jaksch's anæmia is not a primary splenic disease or blood disorder, nor are there valid reasons for viewing the condition as a hybrid form of pernicious anæmia, leukæmia, or Banti's disease occurring in infancy or childhood. It is rather a form of secondary anæmia associated with splenomegaly. Cases occur during the first two years of life, especially about the end of the first year; but the condition is rarely seen before six months or after the age of three. It results either from a lack of suitable food, vitamins, and fresh air, from congenital syphilis, or as a sequela of certain infectious diseases, notably measles. It may arise from a debilitated condition or a nutritional disease such as rickets, which is undoubtedly the most significant factor. Thus rickety children often have enlarged spleens and a secondary anæmia conforming to the von Jaksch type. When these cases are cured by medicine, dieting, and physio-therapy, the spleen retrogresses and the blood picture slowly reverts to normal. It is usual for such cases to respond well to treatment.

In infancy and childhood any anæmic condition is likely to be associated with splenomegaly. It is to be remembered, moreover, that the relative size of the organ is greater during the first few years of

life than in the adult. The splenic enlargement may be explained by the fact that during foetal life the spleen is an important hæmatopoietic organ, and that where anæmia occurs in infancy or childhood the spleen again resumes this function, while at the same time retaining its power to destroy effete, immature, or anæmic erythrocytes. This heavy augmentation in function is responsible for the increase in size of the spleen.

The blood pictures obtained in this disease will vary considerably, at one time closely mimicking those of pernicious anæmia, while at another time resembling the myelocytic or lymphocytic types of leukæmia. Usually, however, the following findings will be recorded: red blood-corpuscles=1,000,000 to 2,000,000; white blood-corpuscles=20,000 to 100,000; hæmoglobin=20-30 per cent (the hæmoglobin percentage is usually very low); and the anæmia of secondary type with colour index=0.5 to 0.7; differential count=lymphocytes in very large numbers, a few myelocytes, and an occasional megaloblast. Normoblasts are found in all cases, and there is usually anisocytosis and polychromasmia.

Clinically, the spleen is felt to be greatly enlarged—it is smooth, hard and not tender, while the swollen liver may extend two to three finger-breadths below the costal margin. The lower half of the abdomen is protuberant. There is no evidence of ascites. The lymphatic glands may be slightly enlarged, but this is not a constant feature.

*Treatment.* Medical measures such as a nutritious diet rich in vitamins, the liberal administration of iron and arsenic, and ample fresh air and sunshine, will often effect a cure. Blood-transfusions may be efficacious. Deep X-ray treatment is of no service. Splenectomy has been performed for this disease but in my opinion operative interference is only indicated where doubt arises as to the true pathology of the splenic tumour or where, in spite of prolonged and thorough medical treatment, the child becomes gradually worse and there is no diminution in the size of the spleen. Splenectomy presents no technical difficulties and is associated with a low mortality—about 8 per cent.

(10) *Thrombophlebitic Splenomegaly.* This may be a primary or a secondary condition in which the splenic vein or the portal vein and its branches are the seat of chronic endophlebitis accompanied by thrombosis and splenic enlargement. When the disease complicates splenic anæmia it is said to be *secondary*; the ætiology of the *primary* cases is still very obscure.

The clotting which occurs in the splenic or portal veins is thought to be secondary to the phlebitis, as in nearly every instance chalky plaques or other signs of degeneration are found in the wall of the thrombosed vein. The thrombus itself, which may either be recent or old, fibrotic or calcified, may completely block the lumen of the vein; or it may become tunnelled, permitting of a precarious circulation. The spleen is



*Fig 474.—THROMBOPHLEBITIC SPLENOMEGALY. THE SPLENIC VEIN IS FILLED WITH AN OLD AND PARTLY TUNNELLED THROMBUS. THE WALL OF THE VEIN IS IRREGULARLY THICKENED.*  
(Museum, University College Hospital.)

of enormous proportions, in fact some of the largest spleens on record have been seen in connection with this disease. Microscopically there is an immense increase in the splenic pulp and a generalised fibrosis. At operation, in all cases of splenomegaly of doubtful pathology and in cases of splenic anaemia, the splenic and portal veins should be carefully examined for any signs of phlebitis prior to the performance of splenectomy. If phlebitis is found to be present in the splenic vein,

as large a segment as possible of the diseased venous channel should be excised, together with the spleen.

The condition is often overlooked owing to the difficulty of diagnosis, but doubtless many more cases would be recognised if at operation more care were taken in palpating and inspecting the splenic vein. After operation the enlarged and thickened venous stumps of the splenic pedicle should in all cases be subjected to the closest scrutiny for evidence of endophlebitis.

Splenectomy is indicated for thrombophlebitic splenomegaly, but the operation will be formidable and associated with considerable risk owing to the presence of numerous adhesions which bind the spleen to the diaphragm and adjacent viscera. The vasa brevia are ballooned and friable to a degree, and these vessels will have to be individually isolated and secured before proceeding with the mobilisation of the spleen which is always a difficult and hazardous procedure in such cases. This is followed by the careful separation and double ligature of each blood-vessel in the main splenic pedicle before the spleen itself is cut adrift.

(11) *Splenomegalic Cirrhosis.* Splenomegalic cirrhosis may closely resemble Banti's disease, but it runs an acute instead of a chronic course, and instead of a leucopenia as in Banti's disease there is nearly always a moderate leucocytosis.

The rationale of the operation of splenectomy in these cases is not only to remove a focus of disease, i.e. the spleen, but also to produce a very marked reduction of the blood in the portal circulation, a quarter of which is ordinarily computed to come from the spleen, and to ease the burden which is imposed upon the already overtaxed and damaged liver. This affords the hepatic cells some respite and grants them opportunity to display their unmatched powers of regeneration.

As these patients are anæmic from repeated hæmorrhages, debilitated, and suffering from the effects of a serious auto-intoxication, it is not surprising that the mortality from splenectomy in such a condition is high, and that the results of this operation, even when combined with omentopexy, are discouraging. As medicinal measures have little or nothing to offer in the way of amelioration or cure, removal of the spleen, hazardous as it may be, should be advised, except in those cases which present hopeless operative risks. Unexpectedly good results will occasionally follow and justify this bold policy.

After excision of the organ the ascitic fluid is completely evacuated by suction, and a modified Talma-Morison operation is performed.

Drainage of the abdominal cavity is not advised as this will almost invariably give rise to peritonitis.

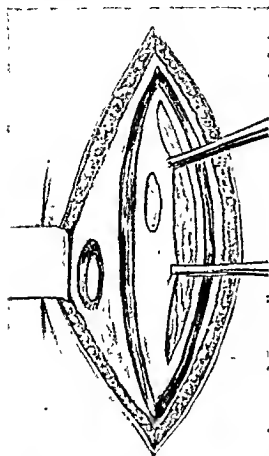


FIG. 475.—OMENTOPEXY. FIRST STAGE. THE PARIETAL PERITONEUM HAS BEEN DISSECTED FREE FROM THE RECTUS MUSCLE. THE PERITONEUM IS SEIZED WITH TWO PAIRS OF ALLIS FORCEPS, DRAWN TOWARDS THE MIDDLE LINE, AND AN OVAL OPENING MADE THROUGH IT.

AN OVAL OPENING IS THEN MADE IN THE RECTUS MUSCLE AT A LOWER LEVEL THAN THE OPENING IN THE PERITONEUM SO AS TO PREVENT ANY KINKING OF THE OMENTUM WHEN IT IS DRAWN THROUGH THESE APERTURES.

### (12) *Gaucher's Disease.*

This interesting disease, with its obscure ætiology, was originally described by Gaucher in 1882. He regarded the condition as a primitive epithelioma of the spleen. It was later thought to be an endothelioma. Mandelbaum and Downey (1916), as a result of exhaustive research work, founded the present conception of the disease as being due to some metabolic disturbance. It was Lieb and Epstein (1924), however, who by chemical analysis of the spleen demonstrated that the lipid nature of the Gaucher substance was, in fact, kerosin which belongs to the cerebrosides. The disease is now regarded as being due to disturbances in lipid metabolism, in which an abnormal substance — kerosin — is deposited in certain cells (Gaucher cells) of the reticulo-endothelial system.

The many important features of this disease may be grouped under the following headings:

(a) *The onset.* The onset is insidious, there being no sign of the disease at birth. The majority of the children are well developed and healthy, and progress along normal lines. The first indication of the disease may be the presence of enlargement of the spleen which may be accidentally discovered. The disease may commence during the first months of life, or may be first noticed as late as the third, fourth, or fifth decade.

(b) The course may be acute in children, but it is, as a rule, chronic in adults. In the average case the condition is slowly progressive, but there are instances where the patient has lived for twenty years or more after the disease was first discovered.

(c) Sex incidence. The disease is commoner in females than in males in the proportion of two or three to one.

(d) The disease is congenital and familial. In about one-third of the cases examined by Professor Ludwig Pick, a great authority on the subject, several members of one generation were affected, but the disease was limited to this generation only. As many as four cases in one family have been reported. So far as is known, no instance has been recorded of the disease being transmitted from parent to child.

(e) Splenomegaly. The spleen undergoes a slowly progressive enlargement until in a late stage it may fill the greater part of the abdominal cavity. Some of the largest spleens ever seen belong to this group. The microscopical appearances are characteristic and are described later.

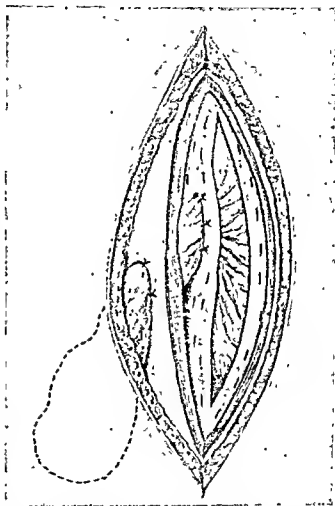


Fig. 476.—OMPHALOCELE. SECOND STAGE. THE OMENTUM HAS BEEN DRAWN THROUGH THE OPENING IN THE PERITONEUM AND IN THE MUSCLE, AND STITCHED INTO POSITION. THE DISTAL END OF THE OMENTUM IS LOOSELY PACKED INTO A POCKET MADE FOR IT IN THE SUBCUTANEOUS TISSUES. NUMEROUS VERY SMALL SLITS ARE MADE IN THE PARIENTAL PERITONEUM BEFORE THIS LAYER IS RETURNED. THE ABDOMINAL WALL IS THEN CLOSED IN THE USUAL MANNER.

(f) The liver is moderately enlarged, and as the disease advances this enlargement progresses. The liver rarely assumes great proportions or extends to more than three or four finger-breadths below the costal



margin. After splenectomy the enlargement of the liver may remain stationary for years, and in a few cases some retrogression has been noticed. There is no ascites.

(g) Blood changes. There is usually a moderate hypochromic anaemia. Lencopenia, due to diminution in the polymorphonuclear leucocytes or lymphocytes, is stated to be a constant feature. It is certainly present in the majority of cases, but not in all. After splenectomy there is frequently a leucocytosis. This is well shown in the case I have described in which the white blood cells rose from 2,200 immediately before operation to 11,000 six days after splenectomy, and nearly three years later a white count of 15,000 was recorded. Thrombocytopenia is present in unsplenectomised cases. After removal of the spleen the platelet count mounts rapidly, often to a normal figure, as I have shown. This pre-operative platelet deficiency may in part account for the hæmorrhages from the gums, purpura, and hæmaturia which are so frequently seen. After splenectomy such bleeding is very rare, and the blood picture shows an all-round improvement.

There may also be, as Potter and McRae have shown, a temporary remission of the disease and improvement both in the blood and in the general condition of the patient following the administration of liver extract.

The characteristic Gaucher cells have never been demonstrated in the blood stream at any time, although they have been obtained from the spleen by puncture, or from the bone-marrow—especially of the tibia and sternum—through a small trephine hole. The material obtained on splenic puncture will often show quite clearly, in fresh or stained smears, the large typical Gaucher cells.

(h) Pigmentation of the skin. A peculiar, diffuse or blotchy pigmentation, limited principally to the face, neck and hands, imparts a greyish or yellow-brown to ochre tone. All these pigmentations, as Pick has shown, are expressions of a general hæmochromatosis which is constantly present in Gaucher's disease, and which becomes more marked as the disease progresses.

(i) Pinguecula-like thickenings of the ocular conjunctivæ. There may be brownish-yellow, wedge-shaped thickenings of the sclerotics, but this condition was only present in some 20 per cent of all the recorded cases, and was absent in the case I shall presently describe.

(j) Bone changes. The skeletal changes observed in Gaucher's disease are :

- (i) Thickening of the shafts of the long bones. In the femur, however, skiagrams may show a punched-out appearance in the lower end.
- (ii) Localised swellings over the bones. Such swellings may closely resemble osteomyelitis or a superficial abscess. The majority of such swellings have been operated upon as cases of osteomyelitis, and the true pathology of the condition has been overlooked.
- (iii) Pathological fracture.

(k) Nerve symptoms. Children suffering from the disease are prone to spastic irritative contractions and tremors of central type.

(l) Lymphadenopathy is exceedingly rare. A case, however, was recorded in which a diagnosis was made after excision and biopsy of an enlarged inguinal gland which contained Gaucher cells.

(m) Disturbances of the organs of internal secretion. Such disturbances, as noted by Norhertzen, are by no means infrequent. Cases of dwarfism, infantilism, and general dystrophies, have been seen in association with Gaucher's disease. Enzer considers that the reports of Gaucher's disease show that examinations of the brain have been neglected, and that in view of the relationship of Gaucher's disease to lipid metabolism, important changes may be present in the brain.

(n) Causes of death :

- (i) Cachexia.
- (ii) Rapidly progressing anaemia and hæmorrhages.
- (iii) Intercurrent disease. The commonest lethal intercurrent disease in infants is broncho-pneumonia ; in adults pneumonia, tuberculous disease, and cancer.
- (iv) Splenectomy. The mortality of splenectomy for Gaucher's disease is about 20 per cent.

(o) Differential diagnosis. A diagnosis will often have to be made from :

- (i) Splenic anaemia.
- (ii) Acholuric jaundice.
- (iii) Hanot's biliary cirrhosis.
- (iv) von Jaksch's anaemia.
- (v) Leukæmia.

(p) Very few symptoms are present until the spleen has increased considerably in size.

(q) Pathological features. The outstanding feature of the disease is the presence of *Gaucher cells* which are found in the Malpighian bodies, in the venous sinuses of the spleen, in the bone-marrow, and

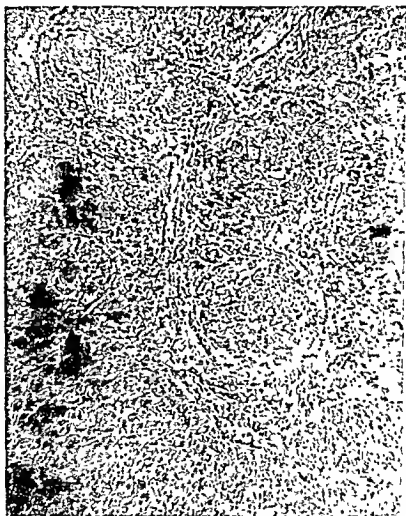


Fig 477.—SECTION OF SPLEEN SHOWING LARGE COLLECTIONS OF GAUCHER CELLS.  
[Author's coll.]

in the sinusoids of the liver. They are in all probability modified reticular cells. Collections of these peculiar, large (about 20-80  $\mu$  in diameter), clear, vesicular cells with their small nuclei (1-20 in number) will be seen in the sections, crowded together and often in alveolar arrangement. The cytoplasm under a high magnification appears wrinkled. This wrinkling is due to a maze of minute threads, woven

together in an irregular network. The wrinkles are the remains of the spongioplasm in the interstices of which the Gaucher substance (kerasin) is stored.

The nuclei may be single or multiple, more often than not numerous, even as many as twenty sometimes being crammed into one cell.



Fig. 473.—HIGHER MAGNIFICATION OF THE GAUCHER CELLS SHOWN IN FIG. 477.

(r) Incidence. Potter and McRae (1933) state that the total number of reported instances of the disease, including two of their own, is now over 100. Splenectomy has been performed in a large number of these reported cases with a mortality of about 20 per cent.

(s) Value of splenectomy. Professor Ludwig Pick, in an authoritative and comprehensive article in the *American Journal of Medical Sciences* (1933), as a result of his extensive experience, offers the

following important conclusions with regard to the value of splenectomy :

**"VALUE OF SPLENECTOMY.** Here opinions differ. In a recent compilation a not inconsiderable number of cases, 19·5 per cent, did not survive the splenectomy. It is admitted that in a number of the cases there is an improvement in the general nutrition and the condition of the blood and a disappearance of the hemorrhagic diathesis. On the other hand, it may be emphasised that in other cases the disease process itself progresses, and that the involvement of the skeletal system, as I have stated, becomes evident only after the splenectomy. Since the spleen, in the course of Gaucher's disease, represents solely or almost entirely an organ depot, its extirpation can, in my opinion, have no influence on the process. There is, therefore, no rational indication for its removal. The symptomatic indication is another matter. In cases of especially severe anemia, hemorrhagic diathesis or where there is a particularly large organ, splenectomy may be indicated. However, the prognosis of the operation becomes worse in just these cases of severe anemia and copious hemorrhages, because of the poor general condition of the patient. Radiotherapy of the spleen has also been tried. The splenic enlargement may be diminished by this means, but there is no noticeable influence upon the course of the disease."

In view of the rarity of Gaucher's disease the case which I saw in consultation with my colleague, Dr. Bernard Myers, at the Royal Waterloo Hospital, and upon which I subsequently performed splenectomy (1931), although described elsewhere (*Medical Press and Circular*, June 28, and July 5, 1933) is, in my opinion, worthy of further review.

**Case of Gaucher's Disease.** J. W., female, aged two years seven months, was admitted to hospital on September 8th, 1931. **History:** This case was admitted for observation and investigation because of a left-sided abdominal swelling which was first noticed at four months of age. An important observation in the clinical history of the case was that the abdominal tumour at first varied in size, but latterly grew steadily bigger. Family history was negative, and as far as could be ascertained there were no other members of the family who had suffered from "abdominal tumours" or enlarged spleen. Broncho-pneumonia in November, 1930 was the only past illness of note. There were three definite attacks of epistaxis, the last one of which was very severe, but no history was elicited as to other forms of hæmorrhage. The motions were always light in colour, but there had been no jaundice. The child's complexion had always been sallow, and sometimes it was brown. Although the appetite varied, development was apparently proceeding on normal lines.

On examination the patient was very pale and waxen; there was considerable wasting of the limbs; the conjunctivæ were normal, although the sclerotics were pale blue. The abdomen was very protuberant, this being specially marked on the left side. A large tumour could be felt occupying the left half of the abdomen and spreading right across, bulging into the right iliac fossa. The margin of the tumour was notched, firm, and sharp, but not tender. The enlarged spleen could be pushed slightly towards the right, but not in a vertical plane. The liver was enlarged three finger-breadths below the costal margin. There was no clinical evidence of free fluid in the abdomen. The urine was normal.

Numerous examinations of the blood were performed, both before and after the operation, as detailed in the table given below. The hæmoglobin was 36 per cent on

admission, and 16 per cent on the day before operation. No pre-operative blood-transfusions were given. The Wassermann reaction was negative, and the bleeding time was over six minutes. Clotting time was normal. It will be seen that six days after the operation the hæmoglobin was 21 per cent, from which it gradually rose to 75 per cent within seven months, and nearly three years later it was 97 per cent. Shortly after operation the bleeding time was seven minutes. Whereas there had been a thrombocytopenia, after operation the platelets became numerous.

Soon after admission it was realised that the child was rapidly going downhill, and that splenectomy offered the only prospect of amelioration or cure. Desperate as the condition was, the operation was performed on September 17th, 1931, under a general anæsthetic. This proved to be a simple and straightforward undertaking. The spleen, although very large and weighing 39 oz., was easily delivered through the abdominal wound, and the whole operation occupied only ten minutes. There was no post-operative shock. The patient developed broncho-pneumonia and was seriously ill for a few days, but subsequent to recovery from this her progress was rapid and uneventful.

Nearly two years later she was again admitted to hospital with a peculiar soft fluctuating tumour which was situated on the inner aspect and at the junction of the middle and lower thirds of the right thigh. This tumour was about the size of a golf ball and was red and painful. The child did not appear to be unduly ill and the temperature was only slightly raised. It was thought to be a "cold abscess," or a subperiosteal abscess of low grade infection. The tumour was incised and its contents evacuated. It appeared to originate from the bone, which was slightly eroded. The walls of the abscess and the portion of bone involved were curetted and the space was packed with gauze soaked in eusol.

The wound healed very rapidly and caused no inconvenience. Some of the material collected from the wound, and presumably from the marrow, showed, on microscopic examination, the typical foamy Gaucher cells. The presence of these cells does not, however, necessarily imply that the primary lesion here was not a localised osteomyelitis, as Gaucher cells are frequently found in the marrow of the shafts of the long bones in cases of Gaucher's disease.

When examined on 15/5/34 she was looking very well. There had been considerable constipation of late. There were no complaints of pains in the bones, no hæmorrhages, and her complexion was good. *The liver had grown larger and extended to the umbilicus.*

The details of the blood count performed on 15/5/34 are recorded on the accompanying table, and show no anaemia, leucocytosis, and numerous platelets.

*"Report on the Spleen.* Weight when received at laboratory shortly after removal was 39 oz. The surface of the spleen was smooth, and the capsule not specially thickened. A few small old infarcts were seen, but no recent ones. No major adhesions had been formed. On section the organ appeared rather tough, and contained extensive zones in which the normal colour was replaced by a slaty-pink. The most striking feature of the microscopical sections was the replacement of large areas of the pulp by large relatively solid masses of cells of the Gaucher type. (See fig. 478.) Some of these cells exceeded  $30\ \mu$  in diameter, and were multinuclear. They stained very faintly, and their protoplasm had often a decidedly reticulate appearance. They did not appear to stain with either Sudan III or osmic acid. It was not possible to examine them by polarised light. Apart from the solid masses of cells, a few free groups were seen within the sinuses. The spleen itself was congested. The Malpighian bodies were apparently not hypertrophic." (F. A. Knott.)

## A CASE OF GAUCHER'S DISEASE.—REPORT ON BLOOD EXAMINATIONS (F. A. KNOTT)

Date.	Red blood cells.	White blood cells.	Polym.	Lymphos.	Hyalines.	Eosinophils.	Basophils.	Hb	C I.	Platelets.	Notes.
9/9/31	3,200,000	3,100	46%	33%	18%	2%	1%	36%	0.56	75,000	Red cells very pale and irregular shapes. Van den Bergh reaction—negative direct and indirect. W.R.—negative. Fragility test normal. Slight hemolysis in 0.15% saline; none in 0.175%. No excess of urobilin in the urine. Clotting time normal, bleeding time definitely prolonged—6 mins. Faces, large amounts of stercobilin.
10/9/31	1,350,000	2,200	41%	39%	14%	2%	1%	16%	0.7	diminished	Red cells very pale and irregular, and tend to basophilic staining. No nucleated reds. Bleeding time—over 6 mins. Clotting time normal.
11/9/31											Notes by house-surgeon on 10/9/31 read: Child has not progressed. Anuria severe and has increased. Condition desperate. Operation decided upon without pre-operative transfusion. Note.—Hb.=16%.
23/9/31	2,000,000	11,000	—	—	—	—	—	21%	0.52	120,000	Bleeding time 7 mins. Note.—No blood-transfusions were given after the operation.
30/10/31	2,800,000	11,800	—	—	—	—	—	29%	0.52	+	
6/11/31	3,010,000	11,000	—	—	—	—	—	33%	0.52	262,000	
20/11/31	3,300,000	9,100	—	—	—	—	—	40%	0.69	172,000	
30/11/32	4,500,000	12,000	—	—	—	—	—	75%	0.93	+	
11/11/32	1,500,000	9,500	61%	28%	7%	3%	1%	88%	0.97	low, approximately 50,000	Bleeding time mildly prolonged.
2/11/33	4,500,000	8,700	67%	23%	7%	2%	1%	88%	0.97	+	Reds practically normal.
23/2/31	4,400,000	19,000	35%	35%	23%	6.5%	0.5%	79%	0.80	plentiful	Reds show no abnormality. No myelocytes.
15/5/34	4,800,000	15,000	52%	23%	7%	7%	1%	97%	0.99	numerous—about 300,000	Reds show no significant abnormality.

## SPLENECTOMY

(13) *Splenic Anæmia—Banti's Disease*. These titles are often used interchangeably, but it is more usual in this country to reserve the term Banti's disease for the later stages of splenic anæmia associated with the development of cirrhosis of the liver.

The ætiology and several factors relating to the pathology are still unknown. Therefore, where the ætiology of any splenic condition resembling Banti's disease has been discovered as a result of clinical, pathological, and serological investigations, such a disease at once passes out of this category.

The disease is commoner in males than in females, and often starts in early adult life, 20–30, though it may occur at any age. It passes through three stages :

*The first stage:* Gradual enlargement of the spleen. This stage may last for many years, and, as a rule, is unassociated with any important symptoms.

*The second stage:* The onset of secondary anæmia with a marked tendency to hæmorrhage of all kinds, especially of gastro-intestinal origin.

*The third stage:* The development of secondary cirrhosis of the liver and the terminal phase of ascites.

The essential features of the disease may be briefly summarised as follows :

(a) Great chronicity. The disease runs a chronic course and may last as long as ten years. It progresses slowly, gradually increasing in severity, and advancing without reprieve to a fatal issue unless arrested by operative intervention. Medical measures, however persistently and skilfully applied, can do little to avert this ultimately fatal issue, but the anæmia will show a temporary improvement with massive iron medication, a method of some value in severe cases before operation is undertaken.

(b) Splenomegaly. A characteristic feature of the disease is the progressive splenic enlargement. In the first and early stages a slight or moderate splenomegaly is the only indication of disease, there being a complete absence of symptoms in most cases. The enlarged spleen is often discovered merely by chance during a routine abdominal examination. It should be remembered that in the very early stages of splenic anæmia, when the spleen is only just palpable, a blood examination may show no abnormality except perhaps such a slight degree of anæmia that it could hardly be regarded as of the secondary type. It is in such cases, however, where there is slight splenic enlargement, unassociated with any marked alteration in the blood, that



surgery offers its most brilliant results ; but the diagnosis must always remain in doubt until the organ has been removed and submitted to microscopical examination. It needs considerable courage to recommend operation in the presence of such negative findings, and it requires much faith on the part of the patient to yield to a comparatively severe operation when he considers himself to be in fair health. If operation is deferred and medical measures are adopted in such cases, in time the slowly progressive enlargement of the spleen and the characteristic changes in the blood will reveal the relentless nature of the disease, while the operation will be further complicated by peritoneal adhesions.

During the second and third stages the spleen increases in size, but it rarely attains to enormous proportions. On abdominal examination its surface is smooth, the notches can be identified, and there is no tenderness on deep pressure unless infarction has occurred. On auscultation no friction sounds are audible, although occasionally hæmic murmurs due to eddies in the large dilated venous channels can be heard. (Rolleston.)

On removal of the spleen its capsule will be found to be thickened (perisplenitis), and there may be, scattered irregularly over its surface, adherent strands of fibrous tissue (adhesions). The splenic vein will often be found to be thickened, enlarged even to the size of a finger, and may be the seat of endophlebitis. On section the cut surface will be seen to be fibrous or flabby and of a peculiar maroon or grey-pink colour. Old or recent infarcts may also be discerned.

On microscopical examination the main feature is a great increase in the connective tissue and a reduction in the number of pulp cells. This fibrosis is responsible for the great thickening of the splenic capsule, of its trabeculae, and of the walls of the splenic veins. It also affects the Malpighian follicles.

(c) Changes in the blood. There is an anæmia of the secondary type with leucopenia, but this, as previously stated, only arises after the disease has been in progress for some time. The blood count may be normal at the inception of the disease, and there may be a leucocytosis after a severe bout of hæmorrhage, or when septic complications are present. But a polymorphonuclear leucopenia is a characteristic feature and becomes worse as the disease advances. In a well-established case examination of the blood will often reveal :

(i) *Red blood-corpuscles.* These are greatly reduced in numbers, usually to about half the normal count, i.e. 2,000,000 to 2,500,000 cells per cubic millimetre. No erythroblasts.

(ii) *Poikilocytosis* is often present to a marked degree.

(iii) The *hæmoglobin* percentage is low—down to about 50 per cent. After a severe hæmorrhage it drops to a very low figure, but subsequently, and especially after a blood-transfusion and the administration of large doses of iron, it rises once more.

(iv) *White blood-corpuscles*. Leucopenia affecting the granular cells is the rule except in the instances mentioned. There may be a relative lymphocytosis. The usual white blood count averages 4,000 cells per cubic millimetre. No myelocytes.

(v) *Colour index*. This is low.

(vi) *Platelets* (see page 835).

(d) A marked tendency to hæmorrhages. A brisk hæmorrhage may announce the presence of the disease and thus lead to its detection. Hæmorrhage may occur in all the stages of the disease; whilst it is rare in the first stage, it becomes commoner in the late stages.

Vomiting of blood is the most serious complication of splenic anæmia, and occurs in fully 50 per cent of all cases which come under observation. The blood in such instances comes from the œsophageal varices and from the dilated vasa brevia.

This serious complication—recurrent gastro-intestinal hæmorrhage—is little influenced by removal of the spleen, as in fully 50 per cent of such cases subjected to splenectomy further hæmorrhages will occur, sometimes proving fatal.

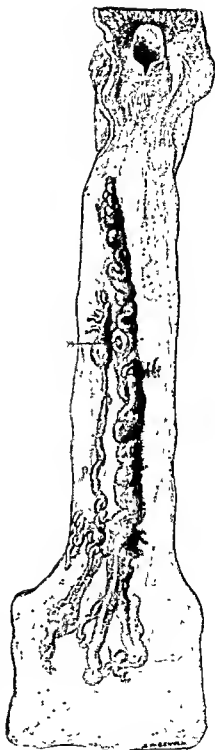


Fig. 479.—RUPTURED OESOPHAGEAL VARIX IN A CASE OF BANTI'S DISEASE. AN EXTENSIVE VARIX OCCUPIES THE ANTERIOR WALL OF THE OESOPHAGUS FROM THE LOWER END TO WITHIN 1½ IN. OF THE CRICOID. JUST ABOVE ITS CENTRE IS AN ULCERATED OPENING. (By courtesy of the Manchester University and the "British Journal of Surgery.")

(e) *Cirrhosis of the liver.* As the disease becomes advanced the liver enlarges and presents the clinical picture of Laennec's multilobular cirrhosis. The hepatomegaly is eventually followed by shrinkage. The changes which are found in the liver depend upon the stage of the disease. During the first and early stages, the organ may appear perfectly normal. Later on it may be enlarged, then shrunken and fibrotic. Ascites is an indication that the disease is very advanced, and when present to a marked degree, necessitating frequent tapplings, it is a sign that death cannot long be delayed. In the terminal stages the patient may appear lemon-tinted, but true jaundice is rare, and, if present, is slight.

(f) *Changes in the bone-marrow.* The bone-marrow is hyperplastic, the typical red marrow of a secondary anaemia being found.

(g) There is no general involvement of the lymph glands.

(h) *Dyspeptic symptoms.* Indigestion, vomiting, nausea, and epigastric discomfort are not characteristically found, and when present they are late manifestations, being due to hepatic insufficiency consequent upon cirrhosis, or to the weight and pressure of the greatly enlarged spleen.

*Treatment of Splenic Anaemia.* As previously emphasised, medical treatment for this condition is of no permanent benefit, and operation should be advised in all cases except where the patient is regarded as too grave an operative risk. Where the following conditions are present splenectomy may be contra-indicated:

(a) Marked hepatic insufficiency.

(b) Grave anaemia resulting from repeated hæmorrhages which frequent blood-transfusions have failed to control. Here it is doubtful whether removal of the spleen will have any effect in checking these hæmorrhages. Again, the operative risk in an exsanguinated patient is almost prohibitive.

(c) In the terminal stage when, in addition to ascites, cachexia is advanced.

Operation will, however, sometimes prove surprisingly successful, even in the most desperate cases, and, where splenectomy is found to be impossible owing to the presence of numerous adhesions, ligature of the splenic artery combined with omentopexy should be performed.

The best results are obtained when the operation is undertaken in the early stages of the disease—the earlier the better.

Pre-operative measures include :

- (i) Rest in bed for a long period.
- (ii) A nutritious diet and the liberal administration of sugar.
- (iii) Intramuscular injections of iron and arsenic, together with large doses of ferrous salts by mouth.
- (iv) Blood-transfusions.
- (v) Radiation of the spleen by radium or X-rays.

If the spleen is very large, applications of X-rays, or preferably radium, are advocated in order to reduce its size. It should be borne in mind, however, that radiation of the spleen has two disadvantages :

It will have a slightly deleterious effect on the already impoverished blood : and

Although effective in reducing the splenic tumour, it will add to the difficulties of the operation by increasing the number of adhesions. Where it is used in excessive doses, without efficient screenage, or even sometimes where its application has been carried out with every care and precaution, it may produce a severe localised chronic adhesive peritonitis and cause adjacent viscera in the region of the spleen to become matted together to such an extent as to interfere seriously with, or even actually to prevent, approach to the organ at operation.

*The operation of splenectomy* has already been described (see page 805), but the following details with regard to technique in cases of splenic anaemia are worthy of special attention.

Hæmostasis must be complete. During the whole operation, from the commencement of the incision to the closure of the wound, there must be no loss of blood. The numerous bleeding points in the wound must all be separately picked up and ligatured. The wound must be quite dry before the tetra-cloths are applied and the peritoneum opened. The blood-vessels in the gastro-splenic omentum will, in this disease, be found to be unduly enlarged, sacculated, thin-walled and friable. Some of these vessels in the upper portion of the gastro-splenic omentum are often enlarged out of all proportion to their neighbours. The gastro-splenic omentum itself is longitudinally lengthened and transversely shortened. The spleen, therefore, closely abuts upon the greater curvature of the stomach. Each blood-vessel in the gastro-splenic omentum will, therefore, have to be individually underrun with an aneurysm needle and doubly ligatured with silk or strong catgut to prevent hæmorrhage and injury to the stomach or

adjacent colon. The blood-vessels in the lienorenal ligament are usually very thick, and some of the individual vessels may be larger than the diameter of an ordinary finger. They may be tortuous and very friable, crumpling up and lacerating on ligature. Inspection of the vascular stump after the spleen has been removed will show the numerous gaping mouths of these severed vessels.

It is most exceptional to find a mobile spleen in cases of splenic anæmia. Adhesions are usually numerous and strong, and anchor the spleen to the diaphragm. In early cases, however, the adhesions are easily separated and mobilisation of the organ is not a difficult matter. But when there is cohesion between the opposing surfaces of the spleen and diaphragm, as may occur in the later stages of the disease, their separation may be impossible.

When dealing with such a case, Moynihan had to remove a piece of the diaphragm. The resulting gap—about 2 inches long—was closed with only the slightest difficulty and the patient recovered.

All visible and accessible adhesions should be divided between hæmostats and ligatured. Those that occupy the splenic bed, however, will have to be separated by blunt dissection by hand or with a swab, and the bleeding controlled by pressure with large, hot swabs packed as tightly as possible into the space.

After removal of the spleen the bleeding points on the under-surface of the diaphragm and adjacent area are secured and tied, or under-run by a "snaking" suture as described by W. J. Mayo. These gauze packs, if correctly placed, are invaluable in controlling the oozing and in facilitating the subsequent steps of the operation.

If there is ascites the fluid should be evacuated by an electric suction apparatus while the operation is in progress.

Pemberton, of the Mayo Clinic, advocates ligation of the coronary vein at the completion of the operation, with a view to reducing the incidence of post-operative hæmatemesis. He considers that "Since the hæmorrhage commonly results from rupture of greatly dilated varices situated beneath the mucous membrane of the lower end of the œsophagus, it has been suggested that this complication might possibly be minimized by tying the coronary vein, with the view of reducing the enormous turgescence by breaking communication with the portal circulation."

Pemberton also holds the view that omentopexy as a means of permitting additional collateral circulation in selected cases is a valuable supplementary measure to splenectomy.

The wound should be very carefully closed and tension sutures should be inserted to guard against the possibility of burst abdomen.

*Mortality and Late Results.* The mortality of splenectomy for splenic anæmia in early cases is about 10 per cent, but in late cases it may be as high as 25 per cent. Pemberton shows that of 167 patients with splenic anæmia and Banti's syndrome who were subjected to splenectomy at the Mayo Clinic, 16 died in hospital—an operative mortality of 9·6 per cent.

“Of the 151 patients who survived the immediate effects of the operation, eighty are known to be living, three of them eighteen years after operation. Two are still living, fifteen and seventeen years after operation, and fifteen have lived from ten to fifteen years. Ten of the sixty-eight patients who recovered from the operation but who died later lived for more than nine years, one for eighteen, one for thirteen, and three for twelve years. Although the causes of many of the subsequent deaths were not attributable to the disease itself, it is of interest that more than a third were directly attributable to hæmorrhage.”

Later figures (1934) from the Mayo Clinic show that of 184 patients with splenic anæmia there were 17 hospital deaths, 72 subsequent deaths, and 92 living.

(14) *Essential Thrombocytopenic Purpura Hæmorrhagica.* The following are a few of the alternative titles which have been applied to this disease :

- (a) Purpura hæmorrhagica.
- (b) Essential purpura hæmorrhagica.
- (c) Essential thrombopenia (Frank).
- (d) Thrombocytopenia (Eppinger).
- (e) Thrombocytolytic purpura (Kaznelson).

Purpura hæmorrhagica may be defined as an essential or idiopathic hæmorrhagic disease characterised by bleeding from the mucous membranes (especially the gums), petechiæ, secondary anæmia, marked reduction in the number of thrombocytes (platelets) in the circulating blood, and a prolonged bleeding time.

The following are some brief notes relating to the *history* of purpura hæmorrhagica.

Werlhof (1775) was the first to describe purpura hæmorrhagica. Denys (1887) noticed that platelets were absent from the blood in a case of purpura. Hayem (1896) showed that there was a flat-topped clot when blood was allowed to stand in a test-tube. Duke (1910) demonstrated that the bleeding time was prolonged in these cases

owing to a diminution in the number of platelets, and to a deficiency of fibrinogen. Hess (1916) showed the importance of the positive capillary resistance test (tourniquet test), and was the first to suggest that the spleen should be removed in this disease. Kaznelson (1916), at Hess's suggestion, performed the first splenectomy for this condition.

The disease is three times commoner in females than it is in males, and although it occurs most frequently in childhood and early adult life and is rare after forty, no age is exempt.

There is no reason to believe that the condition is familial, hereditary or congenital. There are many forms of purpura, but in the majority the blood changes are secondary, as is seen in cases of so-called symptomatic purpura septicæmia, typhoid fever, small-pox, scurvy, poisoning by certain drugs (e.g. arseno-benzol, iodides, etc.), and some skin diseases.

In primary purpura hæmorrhagica—the form under description—the blood changes appear to be primary and the disease has an unknown (idiopathic) causation. Removal of the spleen is contra-indicated in the secondary or symptomatic purpuras. Splenectomy should never be performed in a case of purpura where the platelet count is high or within normal limits. A painstaking clinical examination, combined with a series of investigations (which will be briefly described later), are necessary to establish a correct diagnosis.

The indications for splenectomy for purpura hæmorrhagica are usually quite clear when once an accurate diagnosis has been made. Although splenectomy is indicated in almost every case of essential purpura hæmorrhagica, delay may be advisable in certain instances, as some forms of the chronic disease are prone to very long remissions, spontaneous cure, or cure by effective medicinal measures.

Two forms of purpura hæmorrhagica are described :

- (a) The acute form ; and
- (b) The chronic relapsing form.

In the *acute* form the patient suddenly begins to bleed without any previous warning. There is a severe uncontrollable oozing of blood from the mucous membranes and also into the subcutaneous tissues, occurring with dramatic suddenness and continuing until the patient is bled white, becomes critically ill, or dies in a few days. Hæmorrhages may also occur in the brain or spinal cord, giving rise to nervous symptoms. There may be much extravasation of blood into the subcutaneous tissues (hæmatoma), especially over pressure points ; petechiæ, which vary in size and colour, are always numerous and

often scattered all over the body. They may appear as small spots in the skin, bright red, purple, or dark brown, involving chiefly the extensor surfaces of the extremities and the anterior aspect of the trunk. Bleeding into the internal organs may be evidenced by hæmatemesis, melæna, hæmaturia, or menorrhagia. In certain anomalous cases the *only* symptom may be severe recurrent attacks of hæmatemesis, persistent or intermittent bouts of painless hæmaturia, or incoercible menorrhagia. When the disease assumes this cryptic form the diagnosis is beset with many difficulties and misgivings. An understanding of the true significance of the thrombocyte count and other tests for purpura hæmorrhagica in cases of unexplained hæmorrhage, and a knowledge of the waywardness and disguises that the disease may assume, will, however, prevent unnecessary examinations or rash precipitate surgical measures.

Many cases of "essential hæmaturia" or severe menorrhagia may be unrecognised instances of the hæmorrhagic diathesis.

It is rare for an acute case of purpura hæmorrhagica to undergo spontaneous remission and remain permanently cured.

As soon as the disease is accurately diagnosed, I have consistently advised splenectomy for the *acute* form of purpura hæmorrhagica for the following reasons:

(i) Repeated blood-transfusions have, according to my experience, very little or no effect in arresting or even temporarily ameliorating the factors which determine a fatal issue. Numerous writers, however, state that they have found blood-transfusion to be of very definite value. I have given whole blood or citrated blood repeatedly to a number of cases, but I have not been able to convince myself that these patients have materially benefited by these means. Again, unless the blood is very carefully matched, transfusions may be, and often are, followed by very dangerous complications, such as profuse hæmoglobinuria from lysis of the donor's cells.

(ii) Spontaneous recovery is never to be relied upon, and must always be regarded as an exceptional phenomenon in an acute case.

(iii) The patients may die from loss of blood. The most effective method of producing an immediate hæmostasis in such cases is splenectomy.

(iv) The operative mortality is not as high as is generally stated in literature, i.e. about 40 per cent. Eliason and Ferguson (*Annals of Surg.*, Vol. xcvi, No. 5, Nov., 1932, pp. 801-829) show that of the last twenty-two acute cases recorded there were only three deaths—13.6 per cent. I have operated upon four acute cases without a



death. The immediate post-operative and late results in these four cases were in every respect most gratifying. It seems to me not improbable that a number of cases classified in literature as acute essential purpura hæmorrhagica were instances of aplastic anæmia upon whom splenectomy was performed at great risk and with a high immediate mortality.

In the *chronic* form essential purpura hæmorrhagica is characterised by remissions and exacerbations—milder attacks of hæmorrhage occurring at irregular intervals. During a remission the patient may appear to be in fairly good health. Suddenly, without any warning, he will be plunged into the throes of an acute attack with all the usual symptoms, such as bleeding from the gums, asthenia, pyrexia, anæmia, mental depression, the appearance of bruising of the subcutaneous tissues, petechiæ, and perhaps the occasional vomiting of blood or the passing of blood-tinted urine. After a variable period of days or weeks the symptoms abate, and the health slowly improves. Occasionally an intermittent hæmaturia or intractable menorrhagia may be the legacy of an unduly severe recurrent attack.

*Diagnosis.* A diagnosis is made on the following points :

- (a) Low platelet count.
- (b) Spontaneous extravasation of blood into or under the skin and mucous membranes of the body.
- (c) Prolonged bleeding time.
- (d) Absence of clot-retraction.
- (e) The appearance of petechiæ under the skin, distal to a tourniquet blocking the venous but not the arterial flow.
- (f) Secondary anæmia without constant changes in the erythrocytes.
- (g) No constant variation in the leucocytes, but usually an increase rather than a decrease.

*Observations on the Diagnostic Tests.*

(i) The tourniquet or capillary resistance test. This is performed in the following manner: pressure midway between the systolic and diastolic is maintained for three minutes on the arm, either by a rubber bandage (Bier), or by a blood-pressure instrument. If hæmorrhagic areas (petechiæ) appear between the lower end of the bandage and the wrist, the test is said to be positive. This test is usually—though not always—positive in purpura hæmorrhagica.

(ii) The low platelet (thrombocyte) count. The normal platelet count varies within wide limits—200,000 to 400,000 per cubic millimetre, the average being about 250,000. In purpura hæmorrhagica the platelet count is reduced to 50,000 or less, and in a severe case no platelets may be seen on successive examinations. The platelet count is always low in the acute cases and during the acute exacerbations of the chronic recurrent forms. It is an important diagnostic test, also indicating the severity of the condition, and is a valuable aid in deciding upon the best method of treatment to be adopted.

That the spleen may destroy platelets in virtue of its content of reticulo-endothelial cells in normal health is not definitely proved, but there is an accumulation of evidence that in diseases such as essential purpura hæmorrhagica it does so to an abnormal extent. The clinical confirmation of this destructive power was afforded in two cases of my own in which I collected blood from the splenic artery and the splenic vein, prior to removal of the spleen, and had platelet counts done on each specimen. In both cases a very much smaller number of platelets was found in the specimen from the vein than in that from the artery.

(iii) The failure of the clot to retract. Although failure of the clot to retract in a test-tube is said to be a characteristic feature, I have noticed retraction to be present in a few of my cases. A flat-topped blood clot may occur in blood of perfectly healthy individuals who give no history of hæmorrhages; too much reliance therefore cannot be placed upon this test.

It is interesting to note that the blood coagulation in essential purpura hæmorrhagica is normal in contrast to that which obtains in hæmophilia.

(iv) The prolonged bleeding time. The normal bleeding time is from 3–4 minutes. In cases of purpura hæmorrhagica it is increased to 20, 30, 60 minutes, or even longer.

(v) The size of the spleen. Is the spleen enlarged in essential purpura hæmorrhagica? The statement that splenomegaly occurs in a large number of cases has not been in accord with my own experience. On no single occasion during numerous critical clinical examinations could I detect any splenic enlargement in four acute and nine chronic cases. Again, in each of these thirteen cases the spleen was carefully weighed as soon as it was removed from the body. Eleven were found to be of normal weight and size, whilst two were definitely lighter, smaller, and more fibrotic than normal.

*Differential Diagnosis.* Essential thrombocytopenic purpura hæmorrhagica will have to be distinguished from :

(i) Symptomatic or secondary purpura such as may occur in acute septicæmia.

(ii) Aplastic anæmia.

(iii) Leukæmia.

(iv) Acute toxic purpura due to certain drugs and poisons, e.g. arseno-benzol.

(v) Hæmophilia.

*Treatment.* There are two methods of treatment :

(a) Medical.

(b) Splenectomy.

It should be remembered that mild chronic cases may undergo spontaneous cure. We therefore recommend persistence with medical measures for the mild cases. Operation is advocated for the acute and chronic relapsing varieties.

*Medical Treatment.* This includes :

(i) *The administration of blood.*

1. Blood-transfusion.

2. Autogenous blood by intramuscular injection.

It is said that repeated transfusions may be effective in arresting the hæmorrhage and in producing a remission, often without subsequent recurrence, in a number of cases. This, however, has unfortunately not been my experience. Dixon (*B.M.J.*, Vol. i, p. 16, 1923) gives a favourable report of four cases treated by intramuscular injections of 20-30 cc. of autogenous blood.

(ii) *Radio-therapy.*

1. Radium.

2. X-rays.

3. Ultra-violet light.

Radiation of the spleen and occasionally of the longer bones has been tried, but the results as far as I can ascertain have not been very satisfactory.

(iii) *Non-specific protein shock.*

Injections of the following have been given :

1. Antivenin ; snake venom. (*Proc. Roy. Soc. Med.*, xviii, II.)
2. Horse serum.
3. Milk.
4. Peptone.

(iv) *The use of coagulants.* A number of these, including coagulen, thromboplastin, hæmoplastin, etc., have been given an extensive trial. They have not proved successful in my cases.

(v) *Diets rich in vitamins B and C, and liver.*

(vi) *The administration of certain drugs.*

1. Salvarsan.
2. Calcium, with or without parathormone.
3. Iron and arsenic.
4. Thyroid extract, singly or combined with other ductless gland products.

*Splenectomy.* None of the medical measures advocated above, either singly or combined, can be relied upon to control the extensive hæmorrhages which are present in some of these cases. Removal of the spleen, however, ensures a complete and spontaneous hæmostasis, as abrupt as that which follows the application of a ligature to a spouting artery. Visible proof of the immediate hæmostatic effect of splenectomy was afforded in two of my cases in which bleeding from the gums was so profuse as to interfere very considerably with the administration of the anæsthetic. As soon as the spleen was removed the hæmorrhages ceased instantaneously. This phenomenon has been attributed by some to a fall in the blood-pressure with accompanying shock, rather than to an excessive flooding of the circulation with platelets after the splenectomy. The cause, however, is immaterial, for the fact remains that the platelet count immediately rises by leaps and bounds after splenectomy, hæmorrhage ceases at once, a healthy colour and tone of the body are soon regained, convalescence is rapid and uneventful, and the late results are most satisfactory.

In splenectomy for purpura hæmorrhagica we remove a focus of disease—possibly a parasitic organ. The disease is in some ways akin to hæmolytic jaundice. The spleen in hæmolytic jaundice has a vicarious appetite—an avidity for red cells; in essential purpura hæmorrhagica for platelets.

A number of cases have been recorded where, even after splenectomy,

mild relapses have occurred and the disease has assumed its pristine form. Recurrence is always likely if an accessory spleen or splenuli are unobserved or, if seen, not removed at the original operation. Recurrence may also sometimes be traced to untreated septic foci, such as dental caries, disease of the accessory sinuses, septic tonsils, etc. Removal of these foci of infection is the best prophylactic measure against recurrence. If after thorough eradication of all septic foci there is recurrence, I would advise exploratory laparotomy and a search for splenuli, and, where found, their removal.

In seven of my thirteen cases, i.e. in approximately 50 per cent, splenuli were present, but they were always removed, this being followed by a careful scrutiny of the remnants of the gastro-splenic omentum, of the great omentum, and of other likely sites for ectopic splenic remains.

*Results of Splenectomy.* Eliason and Ferguson reviewed the results obtained in 213 reported cases. They found that the operation mortality for the whole group was 13.1 per cent; but in the cases collected from the last four years the mortality was only 7.08 per cent in 113 cases.

In *acute purpura* there were 35 cases treated by splenectomy with twelve deaths—34.3 per cent. In the last 22 cases, however, there were only three deaths—13.6 per cent.

In the *chronic purpuras* there were 160 cases with eleven deaths—7 per cent.

My own figures show 9 chronic cases with one death and 4 acute cases without a death, i.e. a total of 13 cases with one death—7.9 per cent.

Results obtained at the Mayo Clinic show 57 cases, with 4 hospital deaths, 3 subsequent deaths, and 49 living, 42 of which are in good health.

*Cases.* The following are some very brief notes of four cases of essential purpura hæmorrhagica which came under the care of Dr. Bernard Myers and myself:

*Case 1—ordinary clinical type.* W. B., female, aged 19. She had her first attack of purpura at the age of 10. Prior to admission to hospital (1926) she had numerous recurrent attacks, lasting on an average from two to three weeks. On admission she was suffering from an acute attack which had been in progress for two months and showed no signs of abating. There was profuse bleeding from the gums, purpuric spots in the skin and in the mucous membrane of the mouth, and menorrhagia. A blood examination showed a very marked secondary anaemia, and only a few platelets were present. On a number of occasions the platelets were entirely absent. The

bleeding time was twenty minutes, the tourniquet test was positive, and the spleen was impalpable. All the usual methods for controlling hemorrhage were tried without success. Splenectomy was accordingly performed as this seemed to offer the only prospect of cure. The profuse bleeding from the gums, which was a very noticeable feature during the anæsthetic, ceased as soon as the spleen was removed. This interesting phenomenon was noticed by four of us who were present at the operation.

The patient made a splendid recovery, and now, some years after the operation, she is fit and well and has had no recurrence of her symptoms. Menstruation, which was troublesome prior to operation, now runs a normal course. When she was last examined (about five years after splenectomy was performed) the blood count showed red blood cells, 4,650,000; white blood cells, 6,700; hæmoglobin, 79 per cent; colour index, 0.84. *Differential count*: Polymorphs, 51 per cent; eosins, 2 per cent; basophils, 1 per cent; large hyalines, 10 per cent; lymphos, 36 per cent. The red blood cells showed mild anisocytosis and a few were pale. The platelet count showed 110,000 per cubic mm. Clotting time was normal, 4½ minutes; the bleeding time was 5½ minutes, and the capillary resistance test was negative.

*Case 2—"Essential hæmaturia" due to purpura hæmorrhagica.* S.C., female, aged 6. At the commencement of the disease she was treated as a case of Henoch's purpura. Her symptoms began with sudden abdominal pain, purpuric subcutaneous eruptions, and swelling of the joints, especially the ankle joints. About a week after the onset of the illness she was admitted to hospital as a case of acute intussusception, but fortunately was not operated upon. The purpuric rash and the effusion into the joints disappeared, but she developed hæmaturia which persisted continuously for the ten months prior to my seeing her and until splenectomy was undertaken.

On examination she was very pale, there were no visible purpuric spots, and the spleen was not palpable. A lengthy pathological examination was then done, resulting in the exclusion of nephritic and of any bacterial toxæmia. The blood examination showed a severe form of secondary anæmia and a very low platelet count. The bleeding time was prolonged, but the tourniquet test was negative.

On cystoscopy the bladder was found to be normal, and bright red blood could be seen coming through both ureteric orifices. Ureteric specimens were collected and examined, and an ascending pyelogram of each kidney was taken. The ureteric specimens were sterile and contained a large quantity of blood, a little albumen, but no other abnormal constituent. The pyelograms were normal.

The child was given a prolonged course of medical treatment, but as this proved unavailing splenectomy was advised. In the summer of 1926 the spleen was removed, together with a large spleniculus, and a quantity of free fluid was mopped up from the abdominal cavity. The spleen was not enlarged, in fact, it appeared to be smaller than normal and fibrotic.

On the day after operation the urine, instead of being bright red, was dark brown in colour, and within three days it was normal except for a trace of albumen. The child made a rapid and uninterrupted recovery. For the first year after the operation the urine contained a trace of albumen, but never any blood.

I saw this patient again in August, 1934, when she was in perfect health, the urine being normal.

*Case 3—The chief feature was profuse menorrhagia.* Mrs. D., aged 37. The first attack of purpura occurred when she was aged 14. She had many recurrent mild attacks, but her main complaint was incessant uterine hæmorrhage. Owing to this

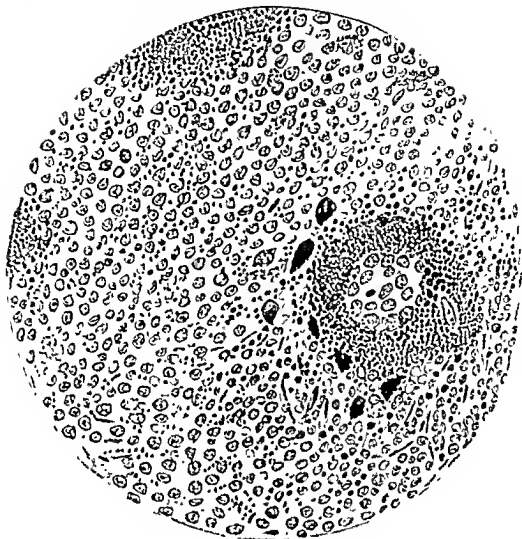


Fig 840. CASE 2. ESSENTIAL PURPURA HEMORRHAGICA TREATED BY SPLENECTOMY. DRAWING OF SECTION OF SPLEEN, LEITZ 8 MM. APPOCHROMATIC, OC. 10X, SHOWING ENDOTHELIAL HYPERPLASIA OF SPLEEN PULP AND PHAGOCYTIC GIANT CELLS. Below: SAME GIANT CELLS, LEITZ 1/12 IMMERSION, OC. 10X, SHOWING PHAGOCYTOSIS OF RED CELLS, POLYNUCLEAR AND PLATELETS (MYERS, GORDON AND MAINWORTH).  
(By the courtesy of the "British Journal of Children's Diseases.")

profuse menorrhagia she was on several occasions admitted to hospital for in-patient treatment, and was subjected to a number of minor operations upon the womb.

At length she was admitted to the Royal Waterloo Hospital in a collapsed condition due to a severe uterine hæmorrhage which followed a miscarriage. This hæmorrhage persisted for two months in spite of minor gynæcological procedures and blood-transfusions. She was bled white and very nearly died. In addition to the loss of blood from the uterus there was a purpuric rash and hæmorrhage from the gums.

On examination the blood showed red blood cells, 2,000,000, and very few platelets. The capillary resistance test was strongly positive in two minutes; the bleeding time was fifteen minutes. Recovery following the usual styptic treatment was slow, but all hæmorrhages ceased and she was eventually discharged and sent to a convalescent home.

Within two months of her leaving hospital she had to be readmitted on account of a severe recurrence of all the symptoms. Splenectomy was performed. Myers writing on this case (*St. Bartholomew's Hospital Journal*, 1933) gives an account of the subsequent history:

"Although nearly eight years ago there has been no further attack of purpura, no hæmorrhages of any kind, even after the extraction of teeth, and menstruation, which was the bugbear of her life previous to operation, frequently necessitating her admission to hospital, became normal and remained so. She looks at present the picture of health. It is interesting to note that the capillary resistance test has remained negative since splenectomy, and the bleeding time is also normal, while the platelets increased to nearly 200,000 within a few days of operation. The last blood examination performed a few months ago showed: Red blood cells, 4,800,000; hæmoglobin, 87 per cent; colour index, 0.89; white blood cells, 9,000. Differential count. Polymorphonuclears, 64 per cent; eosinophils, 2 per cent, basophils, 1 per cent; large hyalines, 7 per cent; lymphocytes, 26 per cent. The red blood corpuscles showed no significant abnormality. The platelets were 194,000 per c.mm. Clotting time normal, 3½ minutes; bleeding time normal, 3½ minutes. Capillary resistance test negative."

*Case 4—In which there were nervous symptoms.* A description of this case is given by Myers (*St. Bartholomew's Hospital Journal*, 1933). He writes:

"E. W., age 10, was under the care of my colleague, Dr. H. Dunlop, who kindly asked me to see her with him about nine months ago. For nearly twelve months previously she had suffered from purpuric spots on the skin. A few large purpuric spots were seen on the lips, inside the mouth, and on the hard palate, and blood oozed freely from them. Similar spots were found on the tongue later on. Epistaxis became troublesome. The platelet count was 28,000. She developed nervous symptoms, with paralysis of the muscles of the right eye, the pupil being completely paralysed. There was weakness of convergence and incomplete ptosis. The lower part of the left side of the face became weak, and also the left arm; the latter was ataxic. Left plantar reflex was indefinitely extensor, the right being flexor. All cleared up completely, the right eye being the last to do so and the pupil last of all. We looked upon the cause as being probably a slight purpuric hæmorrhage in the brain. Although the paralysis completely cleared up, and her mentality remained perfectly normal, the bleeding continued from the nose, lips, and mouth, and fresh crops were apparent on the skin. Blood-transfusion was performed and improved the condition for about



three to four weeks, during which time there was no bleeding from the mucous membranes, but the platelet count, which was 20,000 before transfusion, remained at about the same figure afterwards. Splenectomy was performed nearly ten months ago. No further attack of purpura has happened since, and she has remained in excellent health. After splenectomy the platelet count went up to 400,000, the figure immediately before operation being 28,000.

"It is interesting to note in this child that the capillary resistance test was negative until just before the operation, when it became slightly but definitely positive; the bleeding time increased from 7 to 15 minutes. The capillary resistance test is now negative and the bleeding time normal."

(15) *Hæmolytic or Acholuric Jaundice.* The condition may be defined as a hæmolytic disease, affecting primarily the spleen, and secondarily the liver, gall-bladder, and biliary passages, characterised by anæmia, reticulocytosis, increased fragility of the red blood-corpuscles, "crises," acholuric jaundice, and splenomegaly.

There are many varieties of hæmolytic jaundice, but the only form for which surgery may be required is that which is associated with increased fragility of the red cells. The outstanding characteristics of the disease are by no means always constant in appearance. Lord Dawson (*Lancet*, No. 5688, p. 516, Sept. 3, 1932) has shown that in certain cases jaundice may be absent or the spleen may not be palpable; or again, that the fragility of the erythrocytes may not be constantly increased. In suspected or anomalous cases the advisability of undertaking numerous blood examinations and tests for fragility of the red blood-corpuscles is emphasised, as splenectomy is contra-indicated where the red cells do not show a lessened resistance to hypotonic salt solutions.

The disease may be an acquired affection, but it is usually congenital, and when so it is often familial. The congenital and acquired types present many features in common, the main distinction between them being age of onset, severity of symptoms, and the course of the disease.

The *congenital* form, which was originally described by Claude Wilson (1890), starts in infancy or childhood and runs a mild chronic course. The disease is hereditary and may be transmitted through healthy parents. There are few symptoms; the general health is little affected, and the lemon-tinted complexion calls for such slight comment that Chauffard regarded these patients as being "more jaundiced than ill."

The peaceful course of the disease is at lengthy and infrequent intervals interrupted by a crisis, the signs and symptoms of which often mimic those of gall-stone obstruction.

The *acquired* form, which is most frequently seen in adult life, is more severe in character, more sudden in onset, and more rapid and eager in its course. The crises, too, are more frequent, more prolonged, and more grave than in the cases of the congenital type. As the disease advances the anæmia becomes extreme, until the stage is reached when the patient is more anæmic than jaundiced. Whilst the prognosis is often good in the congenital form, it is always grave in the acquired cases. The immediate and late results of splenectomy for the acquired cases are usually not quite so uniformly satisfactory as for those of the congenital type.

### *Clinical Features.*

(a) *Splenomegaly.* In a few cases, as shown by Lord Dawson, the spleen may not be palpable. A splenomegaly, however, of moderate proportions, or occasionally of great size, is usual. Whilst in some mild congenital cases it may take many years before the spleen protrudes its increasing girth below the costal margin, in the more acute and active acquired type, commencing in adult life, this growth is quick and steadily progressive. In some instances the rapidity of growth is astounding, and within a few weeks of the onset of the disease the spleen may occupy the left half of the abdomen, reach across the middle line, and protrude into the right iliac fossa.

During a crisis the spleen always enlarges and becomes painful and tender on pressure. The pain is due to the rapid stretching of the capsule or to perisplenitis.

(b) *Hæmolytic Jaundice.* The patient's complexion is pale yellow. The jaundice is mild and, although varying in degree, it persists throughout the life of the patient. It often causes no inconvenience, and is not associated with any important symptoms except during a crisis.

The jaundice is due to hæmolysis and not to obstruction of the common bile-duct. It is not accompanied by any of the usual toxic manifestations such as itching, brachycardia, depression, yellow vision, etc., except, of course, in those cases where the disease is complicated by obstruction of the common bile-duct with pigment gall-stones; here jaundice of the obstructive type is present. Such obstruction may account for some of the signs and symptoms seen in severe crises. The fæces are normal in colour and contain bile-pigment. The urine may be highly coloured—amber-brown—from the presence of urobilin, but does not contain bile—hence the term “acholuric.” Where, however, the common bile-duct is obstructed (a not infrequent complication in cases of the acquired type) bile will for the time be

found in the urine and the stools will be putty-like and free from pigment.

(c) *Blood Changes.*

(i) *Anæmia of the secondary type.* This is slight in the mild chronic cases, but becomes more marked and may be intense in the severe cases. During a crisis the anæmia becomes rapidly worse and the red blood-corpuscles may show a surprising fall of two or three millions within a few hours.

(ii) *Abnormal fragility of the red blood-corpuscles.* This may be an inherited characteristic. The erythrocytes show a lessened resistance to hypotonic salt solution, and rapidly undergo hæmolytic. Hæmoglobin is set free and an excess of bilirubin circulates in the blood stream and tints the skin and other tissues pale yellow.

The anæmia which results is due to the excessive destruction of the fragile red blood-corpuscles, but replacement with immature cells from the marrow may, for some time and to a large extent, keep pace with the destruction. These immature or young corpuscles are called *reticulocytes*. They are found in large numbers in the circulating blood and are a characteristic feature of the disease. In some cases of acholuric jaundice the reticulocyte count may be very high—60-80 per cent, the normal figure being approximately only 1 per cent.

(i) Normoblasts may be present.

(ii) Colour index. This is usually less than 1.

(iii) The white blood-corpuscles are generally found in normal numbers, but there may be a slight leucocytosis.

(iv) In very severe and intractable cases the blood picture, apart from the marked rise in reticulocyte count, may closely resemble that of pernicious anæmia.

(d) *Crises.* In this disease acute exacerbations or crises frequently occur. They are due either to an excessive hæmolytic destruction—a form of protein shock—or to obstruction of the common bile-duct with biliary mud or pigment stones. Such crises are characterised by :

(i) Pyrexia.

(ii) Constitutional symptoms such as malaise, anorexia, headaches, and depression, etc.

(iii) Deepening jaundice.

(iv) Acute anæmia.

(v) Temporary hæmoglobinuria (in some cases).

- (vi) No petechiæ and no itching of the skin.
- (vii) Tenderness over the spleen which often becomes greatly and rapidly enlarged. There may also be tenderness over the liver and in the epigastrium.
- (viii) Wasting.
- (ix) An increase of urobilin in the urine.
- (x) A marked increase in the reticulocytes; nucleated red blood-corpuscles may also appear in the blood stream.

(e) *Tendency to the Formation of Pigment Gall-stones.* Chronic cholecystitis, cholangitis, and the formation of pigment gall-stones or black biliary mud often occurs in acbolic jaundice. Pemberton reported that the gall-bladder was diseased (with or without gall-stones) in 81 cases out of 118—68·6 per cent. These pigment gall-stones contain bilirubin, and occasionally there may be not even a trace of cholesterin. The bile in such cases is thick and dark-coloured. These pigment stones often give rise to common bile-duct obstruction, and there is little doubt that many of the so-called crises are really attacks of common bile-duct obstruction. The liver may become cirrhotic late in the disease, and when this occurs the prognosis, both as regards the mortality and the late results following splenectomy, is less satisfactory.

*Causation.* The disease is regarded by many authorities as being due to the formation in the bone-marrow of immature, young, and unusually fragile red cells which readily undergo lysis. The spleen, in coping with the destruction of these unduly fragile cells, enlarges in proportion to the demand made upon it in the removal of these cells from the circulation.

In the acquired form, however, the spleen itself may be primarily at fault by assuming an unnaturally ravenous appetite for red blood-corpuscles. In order to satisfy the peculiar demands of the spleen in this disease the bone-marrow has to work overtime, and in so doing produces a large number of defective and unduly fragile red cells for which the spleen has an even greater avidity.

*Treatment.* Splenectomy is the best treatment for hæmolytic jaundice of the type described, and the results are very satisfactory. Lifelong jaundice disappears in a few days, with marked improvement in the general health and well-being of the patient. The fragility of the red cells may show a return to normal, or almost to normal, in the



increase the jaundice, and may lead to scanty urine and even uræmia.

These observations on the futility of pre-operative blood-transfusion in cases of acholuric jaundice are well exemplified in the case I am about to describe. The mortality is very low, as shown by the figures of the Mayo Clinic, where 128 cases were operated upon with four deaths—a mortality of 3·1 per cent. Approximately 87 per cent of the

ON BLOOD AND OTHER EXAMINATIONS

Blood-transfusions.	Notes.
—	Nucleated reds +. Anisocytosis. Poikilocytosis +. Parasites nil. Stools: no ova, no protozoa. Urine: sp. gr. 1·014, alb. + +, sugar nil, urates + +, urobilin + +, casts nil, a few leucocytes. W.R.=0. Sputum, no T.B. present. B.P. systolic 98, diastolic 35.
—	—
—	<i>Fragility of red blood cells.</i> Result: fragility increased. Hemolysis started at 0·6%, was complete at 0·45%. Control hemolysis started at 0·45%, and was complete at 0·3% or 0·35%.
—	<i>Radiologist's report.</i> Screen: diaphragm movements good, heart shadow + +, especially the left ventricle. Liver and spleen + + +. Radiogram: confirmatory. Consolidation at right base, no effusion. No evidence of pericardial effusion. Heart enlarged, dilatation on both sides of heart.
300 cc.	She had a slight rigor following the blood-transfusion. Group II. Universal donor.
—	—
300 cc.	Universal donor.
—	—
Whole blood 450 cc. saline glucose 100 cc.	Rigor, jaundice + +. Fragility is increased to 0·6% saline.
—	—
—	Reticulocytes + + +.
—	—
Whole blood 460 cc. saline glucose 90 cc.	Followed by rigor.
Whole blood 460 cc. 5% saline glucose 170 cc.	Jaundice has deepened.
—	Spleen: huge, colour greyish, no adhesions, free fluid + +. Liver + +, gall-bladder and biliary passages normal. Section shows that the spleen is engorged with red blood cells in various stages of disintegration. The majority of them have lost their hb. (spleen sent to Tropical Hospital).
—	No post-operative blood transfusions were given.
—	—
—	Transitions 2%. Myelocytes 2%. Poikilocytosis and anisocytosis +. No reticulocytes.
—	Poikilocytosis and anisocytosis +. No reticulocytes.
—	Poikilocytosis and anisocytosis = 0. Platelets = 182,400 per cubic mm. Fragility normal.

patients who recovered from the operation are living, and 84 per cent of these are in good health.

It should be emphasised that where operation is undertaken for acholuric jaundice careful examination should be made of the gall-bladder and biliary passages before the spleen is removed. Where the case is complicated by gall-stones it is best to remove the spleen in the first instance before proceeding with the surgical requirements of the biliary system. In suspected cases, however, where it is obvious that the patient is not fit to stand more than one operative procedure, we would advise splenectomy in the first instance to remove the excessive strain which is thrown upon the bone-marrow, this being the most urgent need of the moment. When the patient has sufficiently recovered from the effects of the splenectomy and his general condition has improved, the gall-bladder is removed, the stones are extracted from the common duct, and drainage of the biliary ducts is instituted. If an accessory spleen or splenuuli are present they should in all cases be removed.

*A Case of Acholuric Jaundice of the Acquired Type.* The following case of hæmolytic jaundice, occurring in a young girl under the care of Dr. Bernard Myers and Dr. W. E. Cooke, was of great interest, not only in diagnosis but also in proving that a remarkable recovery will sometimes follow splenectomy in desperate cases, both as regards the blood picture and the general health. It also demonstrates that no beneficial effects can be expected from pre-operative blood-transfusions (of which this patient had four); in fact, they may be injurious and productive of severe complications such as rigors, anaphylactic shock, and deepening of the jaundice. After splenectomy blood transfusions are unnecessary owing to the very rapid regeneration and all-round improvement in the blood.

Here is Dr. W. E. Cooke's account of the history of the case prior to admission to the Royal Waterloo Hospital:

" M. T., female, aged 13], born at Portsmouth, went to Malta when five years of age, stayed in Malta five years. Admitted to the Hospital for Tropical Diseases, 1920, for supposed kala-azar. Splenic puncture performed here, but no Leishman-Donovan bodies found. Transferred to the London Hospital as out-patient and given X-ray treatment (13 treatments). No definite improvement. Was attending school and kept fairly well up to re-admission here. On February 28th, 1927, she became sick and jaundiced, pain felt in right shoulder, ankles swollen.

On admission, 5/3/27, she was pale and anæmic. Marked pulsation of vessels of neck, crepitations heard over both lower lobes of lungs anteriorly and posteriorly, and dullness over both bases and respiratory rub below left nipple. Pulse rapid, regular, tension poor. Heart enlarged. Apex beat sixth space about one inch outside nipple line. Sounds lacking in tone. Abdomen protuberant; veins marked; liver enlarged up to fifth rib and downwards to level of umbilicus. Spleen enlarged down to one inch below umbilical level. Some oedema of foot and ankles. Generally thin and wasted.

With treatment (expectorants, diuretics, and digitalis) the moist sounds in the chest disappeared, and the oedema diminished. Then a definite double bruit appeared.

most audible in the apical area, but her condition generally was improved. Since admission her temperature has been swinging each day between 98.4° and 101°. Pulse 112-120. Respirations 24-32.

On 27/3/27 she was not so well and showed signs of increasing anæmia. The blood count was down to 2,000,000 red blood cells.

I transfused her on 29/3/27 with over half a pint of blood (300 cc.) and she had a slight rigor subsequently. She is Group II.

On 4/4/27 red blood cells were 2,030,000; hb. 60 per cent.

On 11/4/27 red blood cells were 1,400,000; hb. 40 per cent.

On 13/4/27 I again transfused her with another 300 cc. of blood, and the following day red blood cells were 1,200,000; hb. 40 per cent. Donors in both cases were 'universal donors.'

From 18/3/27 until 7/4/27 she was taking liq. arsenicalis, and had worked up to 6 minims daily.

On 7/4/27 Dr. Myers saw her with me in consultation.

To-day as she leaves us the liver is the same or slightly less in size than on admission, but the spleen has increased in size and is now nearer the symphysis pubis.

I fear the prognosis is very poor, but in the hope that you may be able to fit her for operation, which is rather a desperate hope, we transfer her as arranged."

Although it was realised that this patient was a grave operative risk, the spleen and liver tremendously enlarged, the abdomen distended to its fullest capacity with free fluid, the pulse quick and feeble, the hæmoglobin in the region of 20 per cent, it was felt that splenectomy, hazardous as it might be deemed, offered the only hope, and it was therefore undertaken on 6/5/27.

At operation the gall-bladder and biliary passages were found to be normal, and the liver was greatly enlarged. A large quantity of yellow free fluid was mopped up from the abdominal cavity, and after prolonging the incision the enormous spleen, which was greyish in colour, was removed.

The patient made a remarkable and uneventful recovery, the jaundice disappeared, and her complexion became normal. Reference to the chart will show the progressive improvement that took place in the condition of her blood.

I saw her again on 28/4/33, when she was in perfect health; the liver was not enlarged, the abdominal scar was sound, and examination of the blood showed a normal blood count, and fragility also normal.

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## CHAPTER VIII

### RUPTURE OF THE SPLEEN

by

RODNEY MAINGOT

RUPTURE of the spleen is yearly becoming a commoner accident, and it has been computed that in all abdominal injuries its incidence now varies from 15-25 per cent. This incidence may be attributed to such factors as the increasing number of road accidents, the introduction of the aeroplane, and greater interest in active sports. Schmidt, in 1932, found that ruptured spleen was fifteen times commoner than it was in 1913, and called attention to the interesting fact that, whereas between 1913 and 1926 he saw about two cases a year, between 1926 and 1932 he had treated approximately 32 cases a year. This is in accord with the statistical records of several hospitals situated in the thickly populated industrial centres where a large number of accidents occur yearly.

*Causes.* Ruptured spleen may be caused by *direct* or *indirect* violence: or again, it may occur *spontaneously*.

*Direct injuries* are caused in various ways, resulting from such accidents as a fall from a height, a crush, a blow, a stab, a gunshot wound, etc. They may be associated with external wounds, and on rare occasions the spleen may actually prolapse through the wound.

*Indirect injuries* of the spleen are due to sudden twisting movements of the body, to acute flexion, extension, or rotation of the trunk, or to jarring such as might result from jumping from a height.

The commonest single cause of ruptured spleen is a fall from a height—40 per cent of cases; motor car accidents account for about 20 per cent of cases, and violent sports for about 15 per cent. These figures are difficult to assess correctly and are only approximate. About 20 per cent of all cases of ruptured spleen are associated with other intra-abdominal injuries, and many are complicated by additional injuries to other parts of the body. As might be expected, the victims are principally young adult males, who by reason of their occupation or habits are chiefly exposed to this type of injury.

*Spontaneous Rupture* may occur in a *normal* or in a *pathological*

spleen. While the former is exceedingly rare, the latter, although not often seen in this country, is by no means infrequent in tropical climates where malaria is rife.



Fig. 481.—RUPTURED SPLEEN. (*Museum, Royal College of Surgeons.*)

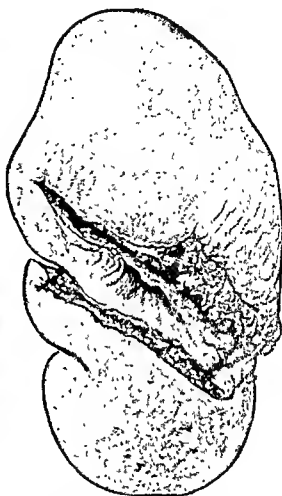


Fig. 482.—RUPTURED SPLEEN. ANOTHER VIEW OF THE SPLEEN DEPICTED IN FIG. 481. (*Museum, Royal College of Surgeons.*)

Spontaneous rupture of the pathological spleen occurs most commonly :

- (1) In malaria.
- (2) In typhoid fever.
- (3) In acute general infections.
- (4) In blood dyscrasias.
- (5) During pregnancy, parturition, and the puerperium.
- (6) In cases associated with splenomegaly from any cause.

In all these conditions the spontaneous rupture is probably dependent upon some slight unnoticed trauma to the softened and congested

spleen. Again, the vascular endothelium of the spleen may be damaged by the circulating toxins which are present in some of these conditions. These toxins cause increased permeability and softening of the smaller radicles of the splenic blood-vessels which disrupt when the intra-splenic pressure is unduly raised from any cause, leading to extravasation of blood under the capsule of the organ. This subcapsular hæmatoma will by the increase of tension produce a pressure necrosis and secondary softening of the splenic pulp, and fresh blood-vessels will eventually be eroded, until bleeding of such a severe nature is produced that the capsule yields and is torn asunder, when the peritoneal cavity becomes flooded with blood.

A correct pre-operative diagnosis is very difficult to make in cases of spontaneous rupture. More often than not an exploratory operation is advised on account of an unexplained intra-peritoneal hæmorrhage, the cause of which is not revealed until the abdomen has been opened. The operation to be advised in all cases is splenectomy.

Spontaneous rupture in a normal spleen, as stated above, is exceedingly rare, and probably less than twenty authentic cases have been reported in this country. Shorten (1919) was one of the first to describe a case. Susman (1927) collected six cases and reported another. Hamilton Bailey (1930) recorded eleven cases from literature, and added one of his own. Byford (1930), Nixon (1932), Dardinski (1932), Black (1933), and Halliwell (1933) have each reported one case. Dardinski was of the opinion that the rupture of the spleen in his case was probably due to an increase of pressure in the organ produced by the damming back of blood from the liver which was filled with secondary deposits. Susman suggests that the majority of cases of rupture in a normal spleen are, in fact, cases of traumatic rupture in which a trivial injury has been sustained, but has been forgotten by the patient. The rent in the spleen is quickly sealed off by a coagulum or by omentum, so that there is little or no intra-peritoneal bleeding at the time of the original injury. After an interval of a few days or even weeks, however, the plug becomes dislodged and permits of a copious and unchecked hæmorrhage. Susman also quotes cases of apparent spontaneous rupture in which the patients remembered later that they had received a blow or some such injury.

We are in accord with this view, and hold that the spleen, owing to its normal friability, is probably the only viscus in the abdomen which is liable to laceration on the receipt of some slight external injury. Torsion of the pedicle of a normal but unduly mobile spleen may

produce degenerative changes in the organ and the congestion necessary for rupture, as observed by Halliwell.

*Naked-eye Appearances of Ruptured Spleens.* In cases of injury to the spleen the organ may be :

- (1) Torn into two portions.
- (2) Completely severed from its pedicles and be found lying loose in the peritoneal cavity.
- (3) Reduced to pulp within its capsule.
- (4) Punctured, as in gunshot wounds and stabs.
- (5) Fissured.
- (6) Lacerated. There is no particular site of election for the laceration. In certain instances a subcapsular hæmatoma may form which may either be absorbed or give rise to further hæmorrhage at a later date.

*Clinical Types of Ruptured Spleen.*

- (1) Spontaneous rupture.
- (2) Where there has been a severe injury followed shortly afterwards by syncope and the death of the patient.
- (3) Where there has been injury followed by shock, a latent period, and then signs of internal hæmorrhage.

In Type 2, there has been a severe injury and the patient is admitted in a collapsed, and often unconscious condition. Death often follows before a diagnosis of intra-peritoneal injury can be made, and even where such a diagnosis is made it is fruitless to attempt operation with the patient in a moribund state. In such cases the spleen is often cut adrift from its pedicles or torn in half, and the abdominal cavity is flooded with blood, while the presence of other injuries, e.g. fractured base of the skull, will often proclaim the futility of operative interference.

In Type 3, the patient has sustained an injury, this being followed first by shock, and then by a latent period during which the patient has recovered from shock. This latent period may last only a short time—sometimes less than twenty-four hours—but it may be prolonged for several days, a week, or even as long as two or three weeks, before signs of internal hæmorrhage are evident. This is the type most frequently seen in surgical practice.

*Symptomatology.*

In cases of ruptured spleen the signs and symptoms will vary according to the severity and rapidity of the hæmorrhage and to the time that has elapsed between the receipt of injury and the examination of the patient. There are as a rule three definite stages, the signs and symptoms of which are distinct :

(1) The stage immediately following the injury. This is the initial period of shock—a reflex, sympathetic disturbance such as may be associated with any abdominal injury. This well-recognised stage needs no further elaboration here.

(2) The latent period. This is the time during which, to all appearances, there is no hæmorrhage from the spleen and the patient has recovered from primary shock. This deceptive stage varies considerably in length; it may be a very short while—a question of a few hours at the most—before signs of internal hæmorrhage become evident; it may be more protracted, even to twenty-four hours; or again, there may be a lapse of as much as a week or more before there is any recurrence of the bleeding, this being known as the “delayed” type of ruptured spleen. During this latent period in 50 per cent of cases there may be no abnormal signs and symptoms found on examination; in some instances the patient will be *apparently* normal again in twenty-four hours after the injury, while in over 30 per cent he will be discharged as fit and may return to work for several days before the catastrophe of a violent intra-abdominal bleeding occurs.

In half the cases seen during this latent phase the following points may be elicited on careful examination: (a) a slight, but continuous aching pain below the left costal margin, or perhaps diffused over the whole abdomen; (b) left shoulder pain (Kehr’s sign); (c) some tenderness in the left flank; (d) slight muscular guarding in this region; (e) local tumefaction; (f) a low degree of pyrexia; (g) a slight icteric tinge of the conjunctivæ; (h) sallowness of the skin.

*Pain* is the commonest symptom of ruptured spleen; if continuous and located to the left hypochondriac region, it is very suggestive of such injury. Phrenic pain, i.e. pain referred to the left shoulder, is a result of irritation by blood clot of the branches of the left phrenic nerve on the under-surface of the diaphragm, and is very often felt, although inquiries as to its presence are seldom made.

*Tenderness* is not marked, but when present it is a valuable sign.

*Rigidity* is not constant, but here again, where it is persistent, it is suggestive of some intra-abdominal lesion.

(3) The stage of rapid bleeding into the peritoneal cavity. During this stage the familiar signs of internal hæmorrhage may be evident.

The symptoms of pallor, faintness, thirst, vertigo, tinnitus, palpitation and tremor, with cold extremities, nausea, sweating, and rapid pulse, are caused by a sudden anæmia. In any suspected case of severe hæmorrhage a hæmoglobin estimation will give a useful indication as to the degree of anæmia present, allowing time, however, for dilution of the blood from the fluid reserves of the body. If the hæmoglobin estimation falls below 50 per cent, it will indicate that the patient has lost a large quantity of blood, although immediately after hæmorrhage the hæmoglobin percentage is normal, or even increased on account of shock. In cases of severe hæmorrhage there is an abrupt and often considerable drop in the blood-pressure. The signs and symptoms of internal hæmorrhage are only conclusive when bleeding has been in progress for some considerable time.

In the majority of cases the symptomatology is relatively unimportant, and on examination the most that will be found is a certain degree of pallor and sweating, some tumefaction of the abdomen, and cold extremities. The temperature, after a sharp fall, gradually rises to 99° or 100° F. The pulse-rate is increased and mounts rapidly, until eventually it becomes thready and almost imperceptible. The respirations are increased and shallow. When "air-hunger" is present a respiratory count may be impossible.

At this stage the abdomen may be slightly distended, and tenderness and rigidity may be elicited in the left hypochondrium. Shifting dullness and Ballance's sign may give added confirmation to the diagnosis.

*A Case of Ruptured Spleen of the "Delayed" Type.* V. C., aged 13, born with an enormous hæmangioma of the right lower limb for which she underwent eighteen operations without success. Whilst in the schoolroom she tripped and fell, hitting her left side against a form. She experienced a sharp, stabbing pain over the left lower ribs, collapsed, and was put to bed in a very shocked condition. When seen by the doctor an hour or two after the accident her temperature was sub-normal and her pulse almost imperceptible at the wrist. She was blanched, almost unconscious, and her body was drenched in cold perspiration.

She made a very rapid recovery, and the next day expressed herself as being quite fit. Within three days she returned to school and continued there until the seventh day after the fall, when during a meal she again suddenly collapsed. I saw her on the following day, i.e. the eighth after the injury. On examination I found her very pale and prostrate, her temperature sub-normal, and the pulse uncountable at the wrist. The abdomen was distended and slightly tender on the left side, particularly below the costal margin. There was shifting dullness in both flanks.

*Operation.* Left paramedian incision, abdomen full of fresh blood and also large, obviously old, clots. The spleen on removal was found to be severely torn along its

convex border, and the gap was firmly plugged with an organising hæmatoma. There were two or three deep fissures near the hilum which appeared to be responsible for most of the hæmorrhage. On mopping up the blood it was interesting to see that there was a large hæmangioma occupying the retroperitoneal space of the posterior abdominal wall on the right side. This hæmangioma was continuous with the massive venous trunks coming up from the right lower limb. It was difficult to conceive that this patient could have sustained an abdominal injury without rupturing one of these engorged, tortuous, and thin-walled channels.

She made a good recovery, but about the eighth day after the operation developed phlebitis of the right leg. When this subsided she was sent home in a fit condition and remains in good health to-day, nearly five years having elapsed since the operation.

This is a case of the "delayed" type of rupture of the spleen where lacerations of the organ occurred with signs and symptoms of initial shock, with recovery from the shock and a prolonged latent period, i.e. seven days. It is to be presumed that the lacerations became plugged with omentum or blood clot, and that these were detached during some slight exertion, causing bleeding of a serious nature to commence afresh.

*Treatment.* As soon as the condition is recognised operation should be undertaken without delay, and, desirable as a blood-transfusion may be, it is only on the rarest occasion that time permits of a suitable donor being secured at once. During operation an intravenous infusion of Crookes' gum saline, or Ringer's solution to which has been added 5 per cent glucose, is useful in combating shock and in replacing the fluid that has been lost.

As the diagnosis is often in doubt and multiple intra-abdominal lesions may be encountered, it is best to open the abdomen through a mid-line epigastric incision. When the presence of free blood in the peritoneal cavity confirms a diagnosis of serious internal hæmorrhage, the spleen should be palpated to ascertain whether it has suffered any injury. Where there is rupture of the spleen, the surgeon will have the choice of dealing with the condition by one of three methods :

- (1) Tamponade.
- (2) Suture.
- (3) Splenectomy.

The first two methods—plugging the ruptured spleen with gauze, or suturing—can only be recommended for very exceptional cases, and splenectomy should be the procedure of choice.

*Technique of the Operation.* If a mid-line incision has been made a more ready approach to the pedicle of the spleen can be obtained by cutting across the left rectus muscle transversely. The injured spleen will often be found to be mobile and can usually be drawn easily through the incision. If the spleen is fixed by adhesions or has a short pedicle, it should be mobilised in the manner already described (see page 810). The pedicles of the spleen are then grasped by the assistant's fingers or by a rubber-covered intestinal clamp, permitting of a careful inspection of the organ to determine the extent of the injury and to facilitate the process of clamping, cutting, and ligaturing the vascular pedicles.

After the splenic blood-vessels have been securely tied, any blood clots that are present in the peritoneal cavity should be scooped out with the hands and the free blood mopped up as far as possible. A rapid exploration and examination of the other abdominal viscera is then performed in view of the possibility of further injuries, particular attention being paid to the liver, the duodeno-jejunal flexure, and the upper coils of the jejunum.

At the completion of the operation a pint or two of warm saline should be poured into the abdominal cavity. The abdominal wound should be very carefully sutured, as wound sepsis and burst abdomen are common post-operative complications. Arrangements should be made for a blood-transfusion without delay.

The mortality of ruptured spleen of Type 3 is stated to be 7-10 per cent. I have had five cases and all but one survived—a mortality of 20 per cent.



## CHAPTER IX

### EGYPTIAN SPLENOMEGALY

by

H. E. S. STIVEN

EGYPT is the gift of the Nile, as Herodotus well said. The abundance of water provided by engineering works of the first magnitude has had the direful result of propagating parasitic diseases and pests which formerly were kept in check by dry season alternating with flood season.

Of these parasites *Bilharzia mansoni*, *Bilharzia hæmatobium*, *ankylostoma*, and *ascaris* are so common that 75 per cent of the population are infected with one or all of them.

Egyptian splenomegaly appears to be associated with these parasites, and in my opinion especially *Bilharzia mansoni*. The intermediate host of *B. mansoni* is the snail molluscan planorbis: the host of *B. hæmatobium*, *molluscum bullinus*.

The geographical distribution of the snail is under investigation, but there is no doubt that the distribution of the disease is related to the end-canal in the northern parts of the Delta.

The theory that splenomegaly is due to a one-sex infection need only be mentioned to be dismissed, although one snail produces cercariae, which transform into only male or female worms.

The infection is recognised by the fellah (farm worker) and called Baoua Itch, on account of the irritation produced by the passage of the cercariae through the skin, Baoua being the name of the Coptic (Egyptian Christian) month which corresponds to our month of June.

The disease is of insidious onset and has three definite stages.

The first stage consists of a feverish period lasting ten to fifteen days.

The second stage consists of a gradual enlargement of the liver and spleen lasting for a period of three years or more. Wasting and anaemia and irregular pyrexia are notable features, and there is loss of appetite and some pain over the spleen.

The third stage consists of an atrophy of the liver with well-marked cirrhosis and a progressive enlargement of the spleen with ascites and death.

If splenectomy is performed in the second stage or early in the third a regeneration of the liver takes place, and the general health of the subject is improved as soon as the intolerable weight of the spleen is removed.

The blood picture before operation shows a diminished red cell count, i.e. about 2,000,000.

The white cells show :

- (i) A general leucopenia, 3,000.
- (ii) A proportional decrease in polymorphs, 40 per cent (75 per cent normal).
- (iii) A proportional increase in mononuclear elements (lymphocytes, etc.), 40-50 per cent (normal 12 per cent).
- (iv) Some cases have a very high eosinophil percentage, 10-15 per cent (normal  $1\frac{1}{2}$ -2 per cent), but this may be the condition in any worm disease.

*Splenectomy is the only remedy for this disease.* I have performed over eight hundred, and the technique of the operation is becoming well established in Egypt. In the General Government Hospitals in 1933, 658 splenectomies were performed with a mortality of 12 per cent.

That the risks of the operation are worth while may be seen by the photographs of the men taken both before and seven years after operation, and before and four years after operation (figs. 483 and 484).

The result of the examination of these men was :

*Salmi Salama :*

Splenectomy, October, 1927.

Seen and photographed, June, 1934.

Urine . . . . .	Negative
Fæces . . . . .	Ankylostoma
R.B.C. . . . .	2,000,000
W.B.C. . . . .	9,500
Hæmoglobin Index . . . . .	80 per cent
Eosinophils . . . . .	4 "
Basophils . . . . .	0 "
Neutrophils . . . . .	26 "
Lymphocytes . . . . .	64 "
Large Mononuclears . . . . .	6 "



Fig. 483.—SALMI SALEM.  
Before and after operation. Splenectomy, 12/10/27.  
Picture on right taken 27/6/31.



Fig. 484.—MOHAMED SULEMAN.  
Before and after operation. Splenectomy, 30/7/30. Picture on right taken 20/6/31.

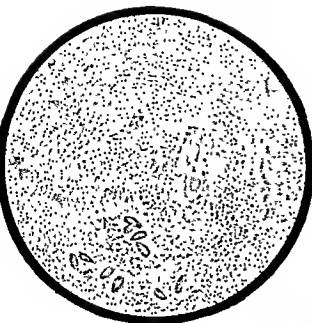
### *Mohamed Suleman :*

Photo and operation, July, 1930.

Seen and photographed, June, 1934.

Urine . . . . .	Negative
Fæces . . . . .	Ascaris
R.B.C. . . . .	2,640,000
W.B.C. . . . .	14,400
Hæmoglobin Index . . . . .	80 per cent
Eosinophils . . . . .	6 "
Basophils . . . . .	0 "
Neutrophils . . . . .	37 "
Lymphocytes . . . . .	50 "
Large Mononuclears . . . . .	7 "

The microscopical picture is well illustrated in the three accompanying micro-photographs.

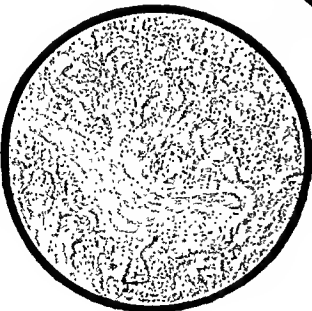


*Fig. 425.—BILHARZIAL TISSUE (XOS-SPLENIC).  
(Low magnification.)*

Plate shows Bilharzial granulation tissue (in the active stage). The outstanding feature is the immense aggregation of the eosinophils round the clumps of Bilharzia eggs in the lower part of the field and the diffuse eosinophilic infiltration all through (small dots).

*Fig. 436.—SPLENOmegaly IN THE ACTIVE STAGE.  
(High magnification.)*

Plate shows a Malpighian body. The central vessel is thickened; there is an apparent and actual decrease in the amount of the surrounding mononuclear elements normally present.



*Fig. 437.—CIRRHOSIS OF THE SPLEEN  
(Low magnification.)*

Last stage after an old Bilharzial infection showing increase of well formed or mature fibrous tissue and detriment of the splenic pulp proper, ending in its disappearance.

## OPERATION

A full dose of carbon tetrachloride is administered on admission to hospital, to clear the intestines of ankylostoma, ascaris, and trichomonas, etc. A full course of tartar emetic is given to kill all the living *Bilharzia hæmatobium* and *mnsoni* and their eggs. During this course six injections of neosalvarsan are given, not only in cases where Wassermann is found positive, but in all cases as a general and most efficient tonic. The positive Wassermann has not been proved to have any connection with the disease.

Anti-pneumococcal vaccine is given in three doses as the time for operation draws near, and the general health of the patient is attended to with good food and tonics.

The operation should always be done under spinal anaesthesia. I use percaine now, whereas formerly I used stovaine with strychnine.

I make an incision varying in length according to the size of the spleen, but always beginning well up to the costal margin and extending down to the level of the umbilicus, or below if necessary. I always divide the rectus muscle into equal halves longitudinally, and although some surgeons advocate a paramedian incision and retract the rectus muscle outwards, I find this procedure involves a loss of time and a definite restriction of the field when manipulating the pedicle of the spleen, which is the crux of the whole operation.

With supporting silkworm-gut sutures I have only had two or three ventral herniae, and these in cases where owing to the general debility of the patient the wound had not healed readily.

On opening the abdomen I have the vacuum evacuator handy and draw off any ascitic fluid that may be present. I insert my right hand to examine the gall-bladder, etc., and ascertain the extent and strength of the adhesions around the spleen. In favourable cases where the adhesions are light they may be broken by the hand. I generally extract the tip of the spleen from the abdomen and cut the tail-like leash of vessels running to this part of the organ after clamping with ordinary artery forceps. I then attack the lienorenal ligament attaching the border of the spleen to the splenic flexure of the colon; this may be broken by the hand, causing only insignificant bleeding, which can be controlled at the end of the operation after removal of the spleen, or if the ligament is strong it can be cut between clamps.

The right hand may now be passed down into the vault of the diaphragm and again adhesions should be broken by the hand. The

whole spleen is then delivered from the wound, and as this is being done the left hand palpates and controls the pedicle of the spleen ready for any emergency should the pedicle tear. As soon as the spleen is delivered I pick up the gastro-splenic omentum, make a hole in it, and put three clamps on the leash of vessels running from the greater curvature of the stomach to the spleen, immediately dividing this, leaving two clamps on the stomach end and one on the spleen. I then clamp and divide the rest of the small omentum and, this being done, it will be found that the pedicle of the spleen is a broad mass lying between the first and the second fingers of the left hand.

I then lift up the lower border of the spleen and free the pedicle where the lienorenal ligament has been partially separated; the tail of the pancreas is also exposed. Bleeding, troublesome to control, may occur here, but a catgut ligature on a round needle, passed into the substance of the pancreas and gently tied, generally succeeds in arresting it. I then put on to the pedicle four very big clamps in juxtaposition; I cut between the third and fourth and the spleen is removed. I then pass a linen thread around the pedicle and removing the clamp nearest the aorta I tie firmly. I then place another ligature in the position of the second clamp, loosening the third clamp during the tightening. I place a third ligature for safety at the end.

(The ligature I use is Barbour's linen thread, No. 18 and No. 36, white and black. They are prepared in lengths of a foot and tacked on to a strip of linen. This is then sterilised in the ordinary way in the drums with the swabs, etc.)

*In the case where the pedicle is too complicated and thick to tie by a simple encircling ligature, I pass a pedicle needle loaded with a white and a black thread through the middle of the pedicle and tie either an interlocking ligature or separate, as suits the circumstances best; here the advantage of a white and a black thread is readily seen. I then put on supporting ligatures according to the requirements of the case.*

A retractor is now placed in the upper end of the wound and the evacuator is placed in the cavity left by the spleen. I then ligature off the vessels held in the clamps on the small omentum with linen thread and also the vessels on the lienorenal ligament. I pick up with big bullet-nosed clamps any bleeding vessels in the bed of the spleen and the vault of the diaphragm, taking particular care at this stage that everything is perfectly dry. On examining the spleen when the

abdomen is first opened, it is sometimes found that the adhesions are very thick and massive and that the spleen is intimately adherent to the abdominal wall and adjacent viscera. The surgeon need not be intimidated by this state of affairs. If the adhesions are really too strong to break and tear, then a species of decortication may be carried out and, with the left hand controlling the pedicle of the spleen, it is found possible to shell the spleen pulp out of its hardened cortex. This cortical shell is left *in situ* and does not bleed. The abdomen is closed with a continuous catgut suture, and then three or four silk-worm-gut sutures are passed through the skin and under the rectus muscle, these being held by artery forceps until the anterior sheath of the rectus is closed with another continuous suture. The skin is closed with Michel clips, and the silkworm-gut sutures are tied, taking care not to strangle the muscle-fibres of the rectus. The long ends are then tied over a round roll of gauze which acts as a splint and support to the wound.

#### AFTER-TREATMENT

The after-care of the patient begins with a pneumonia jacket put on in the theatre, and the placing of the patient's bed in the ward in a position free from draughts. Such patients are very liable to develop a left basal pneumonia, and, in fact, in the early days of the operation the mortality of 90 per cent was caused by this post-operative complication.

I do not advocate the transfusion of blood, glucose, or saline, and I try to prevent the patient from drinking for twenty-four hours.

A feverish period of four or five days is often noticed, and in such cases a quinine tonic is found beneficial. Injections of camphor every four hours is also routine treatment for three days.

The clips are removed on the eighth day, and the supporting silk-worm-gut on the tenth day. Patients are allowed up out of bed on the eleventh day, and go home on the fourteenth. They improve slowly in health, and in three months' time are back at work as healthy labourers.

#### CONCLUSIONS

Splenectomy is the only cure for Egyptian splenomegaly. The risks of the operation are great, but careful preparation of the patient on the lines here laid down considerably diminishes the risk.

The origin of the disease is not established.

I have to thank Dr. Anis Onsy, of the Public Health Laboratories, Cairo, for the use of his micro-photographs, and Dr. Zaki Farag, of the Demerdache Hospital Laboratory, for his help in the examination of material.

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SECTION 10

DISEASES OF THE VERMIFORM APPENDIX

by  
HAMILTON BAILEY

## SECTION 10

### DISEASES OF THE VERMIFORM APPENDIX

#### APPENDICITIS

THE term "appendicitis" was used first in 1886 when Reginald Fitz of Boston published a paper upon this subject. It was Fitz's paper, more than anything else, which resulted in diseases of the vermiform appendix being transferred from the domain of medicine to that of surgery.

During the past 40 years advance in the diagnosis and treatment of appendicitis has been remarkable, but so long as the mortality remains what it is (in England and Wales 3014 individuals died of appendicitis in 1932) all of us have something yet to learn about the subject.

*Surface Anatomy.* McBurney's point has been the guide to the area of greatest tenderness in appendicitis ever since it was first defined by its originator in 1889. Usually the point is described inaccurately. In the original statement it is given as being between  $1\frac{1}{2}$  inches and 2 inches from the right anterior superior iliac spine upon a line joining that spine with the umbilicus (fig. 488). McBurney's point as defined by McBurney is the classical point of greatest tenderness in appendicitis, and also a most useful point to have in mind when an incision to expose the appendix is about to be made. Its erroneous usurper, which unfortunately has been copied from book to book, is a point at the junction of the outer and middle thirds on the same line. The latter is too medial.

*Surgical Anatomy.* The vermiform appendix is present only in man, certain anthropoid apes, and the wombat. It is the only organ in the body which has no normal position. The relative frequency of its more usual positions is depicted in figure 489. The frequency of the



Fig. 488—McBURNLEY'S POINT LIES UPON THE LINE SHOWN, 1 1/2" TO 2" FROM THE ANTERIOR SUPERIOR ILIAC SPINE.

retrocæcal position should be noted. The cæcum of the Mangaby monkey is normally situated beneath the right lobe of the liver; in man, failure of the cæcum to descend is a common anomaly. Extravagant examples of retrocæcal appendices are often associated with a mal-descended cæcum. Complete duplication of the appendix has been described (P. Green), but this is an anomaly of great rarity.

The base of the appendix is always the meeting-place of the tænicæ coli; consequently when the appendix is difficult to locate one of the tænicæ should be traced downwards. If the organ is still not visible and it is certain that it has not been removed, it will probably be found buried in the posterior cæcal wall, and will be discovered by palpation and dissection. A portion only of the appendix may be buried in the cæcal wall, its terminal part being free (fig. 490). Such a condition invites

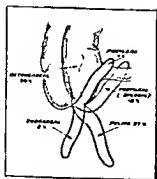


Fig. 489—THE RELATIVE FREQUENCY OF THE VARIOUS POSITIONS OF THE APPENDIX. (From data compiled by Gladsone and Walsley)

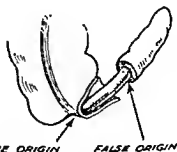


Fig. 490—SHOWING HOW THE PROXIMAL END OF THE APPENDIX MAY BE BURIED IN THE CÆCAL WALL, AND THE CURE METHOD OF ENSURING TOTAL APPENDICECTOMY.

incomplete appendicectomy with recurrence of symptoms and the possibility of legal action.

*Vascular Supply.* The appendix has an abundant blood supply, a vascularity apparently out of proportion to its size.

The appendicular artery is often stated to be the only artery supplying the appendix. In nearly 50 per cent of cases there is an accessory appendicular artery, a branch of the posterior caecal (fig. 491).

The appendicular vein is a radicle of the ileo-colic vein which drains into the portal system.

*Lymphatics.* Four, six or more lymphatic channels traverse the meso-appendix to empty into the ileo-caecal glands.

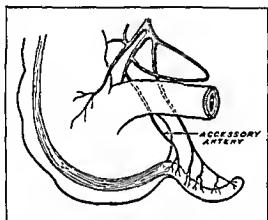


Fig. 491.—THE ACCESSORY APPENDICULAR ARTERY, A BRANCH OF THE POSTERIOR CAECAL, IS PRESENT IN NEARLY FIFTY PER CENT OF CASES. (After Seashachalam.)



Fig. 492.—AN APPENDIX FILLED WITH OXYURIA VERMICULARIS.

*Minute Anatomy.* Interesting features in the structure of the normal appendix are :

1. That the muscular vestment of the appendix is not complete. Where the vessels penetrate, gaps called the *hiatus muscularis* are present ; these gaps afford a possible explanation of the apparent ease with which organisms in appendicitis migrate to the peritoneal cavity.
2. The submucous layer of the appendix contains a heavy deposit of lymphadenoid tissue.

#### ÆTIOLOGY

The riddle of appendicitis—its actual cause and its meteoric rise from an insignificant disease to a menace to the civilised world—has

been a matter for much divergent speculation. So far no satisfying explanation has been forthcoming. The following ætiological factors are important, but for the most part they must be looked upon as purely contributory.

*Worms and other foreign bodies.* That worms (fig. 492) and other intestinal parasites can and do injure the appendicular mucosa and may block its lumen must be allowed. The same will be admitted for a foreign body. Nevertheless, in the majority of instances it is quite clear that parasites and foreign bodies play no part in the production of appendicitis.

*Concretions and strictures.* One of the worst forms of appendicitis, namely appendicular obstruction, is often determined by the presence of a concretion or a stricture preventing the products of inflammation from escaping into the cæcum.

*The role of fats and fatty acids.* Williams of Liverpool believed that fatty acids were excreted by the intestinal glands. Appendicular concretions, he stated, are formed of insoluble soaps and fats. Just as a concretion can block the lumen of the appendix, so, according to his theory, insoluble soaps block the appendicular gland-tubules making them an easy prey to bacterial infection.

*Is appendicitis an endemic disease?* Those who deal with large numbers of cases of acute appendicitis are impressed by the fact that cases occur in groups. Deaver noticed that the numbers increased during the summer. In a Swiss Communal Hospital it was found that the highest incidence was during March and April. We have been impressed by the fact that virulent examples appear to occur in batches so regularly as to defy the vagaries of coincidence.

*Familial susceptibility.* That there is sometimes a familial tendency to the disease cannot be disputed. This generally accepted fact can be accounted for by an hereditary malformation of the organ which predisposes to infection. Thus, the whole family may have a long retrocecal appendix with a comparatively poor blood supply (fig. 493), and many of its members fall victims to appendicitis in one form or another.

*Race and diet.* Appendicitis is particularly common in the highly civilised European, American and Australian countries, while it is

rare in Asiatics, Africans, and Polynesians. If, however, individuals from the latter races migrate to the countries where appendicitis is common they soon acquire the local susceptibility to the disease (Rendle Short). Even apes in captivity appear to acquire the human liability to appendicitis. These significant facts satisfy many that the rise of appendicitis amongst the highly civilised is due to an unbridled departure from a simple diet rich in cellulose. But this cannot be the whole explanation, for acute appendicitis occurs in babes at the breast and in life-long vegetarians, as we have witnessed.



Fig. 403.—A TYPICAL RETROCECAL APPENDIX. AN APPENDIX RUNNING UP TO THE RIGHT KIDNEY AND ALMOST BURIED IN THE CAECAL WALL, A TYPE WHICH IS VERY PRONE TO BECOME DISEASED, IS SOMETIMES COMMON TO MEMBERS OF THE SAME FAMILY.

*The abuse of purgatives.* Here again is another contributory factor of great importance. It is abundantly clear that the common practice of administering purgatives, particularly castor oil, to patients with "stomach ache," and the violent peristaltic action which results, favour, and often determine, perforation of an inflamed appendix.

#### BACTERIOLOGY

Cultures from inflamed appendices usually reveal that the infection is mixed; rarely a pure or almost pure culture is obtained. The following are among the most important organisms associated with acute appendicitis.

*Streptococcus faecalis* is associated especially with cases of severe infection, and is the usual cause of extensive and rapidly fatal peritonitis. H. H. Brown has found it in pure culture in the systemic blood taken from such cases.

*Bacillus coli communis.* Like the foregoing, this organism is, of course, also a normal inhabitant of the large intestine. Again, it has been found in the blood culture of patients moribund from appendicitis with peritonitis. Brown has had prepared a serum of *S. faecalis* and *B. coli* from these cultures.

*B. arogenes capsulatus (B. Welchii).* It was shown by Welch that this organism had an almost universal distribution in the intestinal tract of man. The organism has been found in a number of cases of appendicitis, including fulminating examples. Following the work of B. Williams of St. Thomas's Hospital on the value of anti-gas gangrene serum in intestinal obstruction, anti-gas gangrene serum is used by many as a routine measure in cases of gangrenous and perforated appendicitis, in conjunction with surgical treatment.

*Aschoff's organism.* Ludwig Aschoff has discovered a small Gram positive organism situated within symptomless and normal appendices—always and only, it should be noted, near the tip of the organ. He finds this organism in profusion in smears from acutely inflamed appendices and often within the neighbouring phagocytes. Aschoff considers that this regular and peculiar inhabitant of the distal end of the appendix for some reason becomes virulent and is the actual cause of appendicitis.

#### ACUTE APPENDICITIS

*Pathology.* A practical classification is to divide examples of acute appendicitis into acute appendicitis proper and acute appendicular obstruction.

*Acute appendicitis proper* probably always begins as a simple catarrhal lesion and passes through the various phases of acute inflammation, terminating in some instances in gangrene.

*Acute appendicular obstruction.* The lumen of the appendix is obstructed by a stricture, concretion, parasite or foreign body (fig. 494). When the obstruction is complete it tends to produce early gangrene of the organ. Close examination of gangrenous appendices directly after their removal show that they usually belong to the obstructive group. Perforation often occurs, particularly at the site where a concretion is impacted.

*Macroscopical appearances in early acute appendicitis.* In early cases of acute appendicitis, the organ, when viewed from without, looks normal. What is often more important, when a normal appendix has been bathed in inflammatory exudate from some other intra-peritoneal lesion the exterior of that appendix looks inflamed. So it comes about that when a surgeon is in doubt as to the culpability of an appendix he should remove the organ and at once pass it to someone in the theatre

to slit up. If the appendix is the source of the trouble "it will be worse on the inside than on the outside."

"Dotted" areas of brownish-red pigment within the mucous membrane are often seen towards the tip of the organ. To the uninitiated these may appear to be evidence of inflammation. Scrutiny of the washed surface will reveal their true nature.

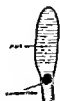


Fig. 494.—ACUTE APPENDICULAR OBSTRUCTION. (After Prof. Wilkie, who first described this important condition)

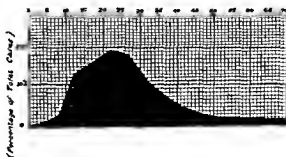


Fig. 495.—AGE INCIDENCE OF ACUTE APPENDICITIS (After Sir George Beatson.)

Every opportunity should be taken to examine the mucosal surface of recently removed appendices. The opinion of a man with experience is often more reliable than a histological examination. Pathologists agree that a doubtfully inflamed appendix is an extremely difficult subject upon which to express a definite opinion.

*Clinical features.* No age is exempt. While it is true that appendicitis is far more common in young adults (fig. 495) it is found in early infancy and even in extreme old age.

*Sex.* Until a decade or so ago males were attacked more often than females. To-day there is very little disparity in the frequency with which the sexes are attacked. The rise in the number of females suffering from appendicitis is but another curious factor in this comparatively new disease.

In a work designed for post-graduates it is unnecessary to reiterate the classical symptoms and signs of acute appendicitis. Typical cases, which constitute about 65 or 70 per cent. of the total, are extremely simple to diagnose. On the other hand, atypical cases can be amongst the most difficult problems with which the surgical clinician is confronted.



## ATYPICAL ACUTE APPENDICITIS

*Acute appendicular obstruction.* The leading symptom is colic; the pain comes and goes. The colic is often severe. The patient may vomit once or twice, but this does not relieve the spasms. Usually the temperature is normal and the pulse between the attacks is not necessarily accelerated. Abdominal tenderness is general, and if the appendix is in the right iliac fossa and not covered by the cæcum this area will be tender. If it is in the pelvis the rectal examination will be significant. If retrocaecal or splenic in position localisation of tenderness may be entirely missing, although deep finger-point tenderness in the loin sometimes suggests a diseased appendix in the former situation. Hyperæsthesia in Sherren's triangle (fig. 496) is often of the utmost value in the diagnosis of these cases. When it is present the appendix should be removed just as soon as possible. We have also found Rovsing's sign of help in not a few instances.

*Rovsing's sign.* Even pressure is exerted over the pelvic colon. This forces gas into the cæcum (fig. 497). If pressure on the left causes pain to be felt in the right iliac fossa the case is probably one of acute appendicitis.

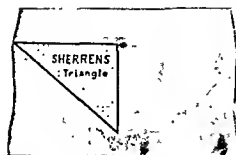


Fig. 496.—SHERRENS'S TRIANGLE FOR APPENDICITIS, FORMED BY (1) THE HIGHEST POINT OF THE ILIAC CREST; (2) THE RIGHT PLEVIC STRIKE; (3) THE UMBILICUS. IN CASES OF ACUTE APPENDICITIS EPICRITIC HYPERÆSTHESIA IN THIS TRIANGLE SUGGESTS THAT THE APPENDIX IS YET UNPERFORATED.



Fig. 497.—ROVSING'S SIGN. WHEN THE SIGN IS POSITIVE PRESSURE ON THE LEFT ILIAC FOSSA CAUSES PAIN IN THE RIGHT ILIAC FOSSA.

*Acute appendicitis with diarrhœa.* Constipation is so usual in the early stages of acute appendicitis that when diarrhœa accompanies it the true diagnosis is liable to be overlooked. If diarrhœa is accompanied by even slight, but constant, tenderness and some rigidity in the iliac fossa, other things being equal, the appendix should be explored.

*Early acute appendicitis with a high temperature.* If the temperature is over 102° F. within the first twelve or twenty-four hours of an attack of abdominal illness, there is a tendency at once to rule out acute appendicitis, even in the presence of some sign pointing to that condition. True, such cases are usually due to pneumonia or right-sided pyelitis, and it is not our object to question this probability. The point we wish to emphasise is that appendicitis cannot be eliminated because the temperature is too high. The examination should be just as thorough and the differential diagnosis weighed, taking the temperature as but a moiety of the factors before us.

#### ACUTE APPENDICITIS WITHOUT ABDOMINAL RIGIDITY

Abdominal rigidity may be entirely absent :

(a) When the appendix is placed deeply in the pelvis. The rectal examination should prove invaluable in this instance.

(b) When the appendix is retrocecal and virtually extra-peritoneal.

(c) In obstructed appendicitis, especially soon after perforation has occurred. Immediately after an obstructed appendix has perforated, the violent abdominal pain disappears. The patient often says he feels better. The hyperæsthesia goes, rigidity may be

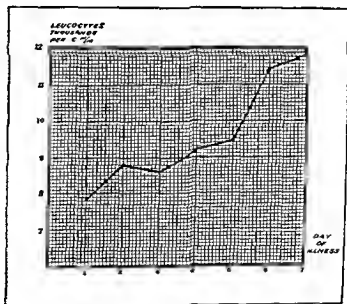


Fig. 498.—AVERAGE TOTAL NUMBER OF LEUCOCYTES PRESENT IN THE BLOOD DURING AN ATTACK OF APPENDICITIS. (R. J. McNeill Love.)

almost, if not entirely absent, but the *pulse-rate soon begins to rise*. But for the last-named sign we would be mistaken more often than we are. Probably this is the most difficult phase of acute appendicitis to diagnose.

It is in problems such as these that a leucocyte count is sometimes of value, a polymorphonuclear leucocytosis being contributory evidence of an inflammatory lesion (fig. 498).

#### DIFFERENTIAL DIAGNOSIS

From a long list of conditions which have often been mistaken for appendicitis the following are among the most important.

1. *Early pneumonia and pleurisy*. Diaphragmatic pleurisy often gives rise to referred abdominal pain and abdominal rigidity. When right-sided, and when percussion and auscultation of the chest are negative, the differential diagnosis can be difficult. Movement of the alae nasi and an increased rate of respiration definitely favour a thoracic lesion. To operate in early pneumonia jeopardises the life of the patient. In all doubtful cases a consultation with a physician should be held. This is the outstanding erroneous diagnosis, and the one which gives greatest cause for regret.

2. *Abdominal influenza* is another condition which is sometimes supremely difficult to distinguish from acute appendicitis. The difficulty, of course, only appertains during epidemics of influenza. We have more than once removed a normal appendix in such circumstances.

3. *Right renal colic or pyelitis*. This is sometimes an extremely difficult differential diagnosis. Urgent cystoscopy with catheterisation of the right ureter and immediate examination of urine from the right kidney will occasionally prove helpful when ordinary methods have failed. Nevertheless, it cannot be emphasised too strongly that pus in the urine does not necessarily exclude appendicitis. We have seen, also, examples of early acute appendicitis with slight hæmaturia. The explanation is that that elusive structure, the appendix, can lie against the right ureter, or rest on the dome of the bladder. Urologists are wont to jeer because a large number of patients with a stone in the right ureter bear the scar of a recent appendicectomy. Be that as it may; when still in doubt early in the attack, after having examined the patient twice within the hour, there can be no question as to the surgeon's duty—it is to remove the appendix.

4. *Ruptured lutein cyst* (syn. apoplectic ovary) occurs particularly during the spring months, and in early womanhood. The patient is usually unmarried, or recently married and childless. The signs are similar to those of very early tubal abortion, but of course the history of a missed period is absent, as also is the sign of a soft cervix. We have encountered a score of these cases and so far have not yet made the correct pre-operative diagnosis with assurance. It is practically impossible to rule out the possibility of a mild acute appendicitis.

5. *Early pregnancy* sometimes commences with abdominal pain and vomiting, and when the pain is right-sided differential diagnosis is difficult, especially in the unmarried.

6. *Ectopic pregnancy*. It is unlikely that a ruptured ectopic pregnancy, with its well-defined signs of hæmo-peritoneum, will be mistaken for acute appendicitis, but the same cannot be said for a right-sided tubal abortion, or more still for a right-sided unruptured tubal pregnancy. In the latter the signs are very similar to acute appendicitis, except the pain *begins* in the right side and there is often a history of a missed period. In tubal abortion signs of intra-peritoneal hæmorrhage are likely to be manifest. When the internal bleeding has not been excessive the differential diagnosis between acute appendicitis and tubal abortion is not always simple, especially when the history of a missed period is lacking. The abdomen moves well on respiration, there is deep tenderness in the iliac fossa, but seldom rigidity. A vaginal examination reveals the cervix softer than usual, and all the fornices are tender; which is of considerable importance since in inflammatory conditions the tenderness is only posterior and lateral.

7. *Salpingitis*. Unlike early acute appendicitis, early salpingitis is far better treated by non-operative measures. The history of a vaginal discharge, of menstrual irregularities and dysmenorrhœa, of burning on passing water, are all helpful differential diagnostic points. The tenderness in salpingitis is usually more medial than that found in acute appendicitis, and it is inclined to be bilateral. A vaginal examination often gives conclusive evidence of acute tubo-ovarian disease. A smear from the cervix uteri examined microscopically sometimes clinches the diagnosis. In a few cases the differential diagnosis is so difficult that in an early case it is wise to explore the lower abdomen.

8. *Suppurating deep iliac glands*. Psoas spasm is a leading feature of the case. Psoas spasm is also seen sometimes in appendicitis.

In three-quarters of all the cases there is a demonstrable infective focus such as a scratch or a sore upon the lower limb of the affected side.

From time to time perforated duodenal ulcer, acute cholecystitis, diverticulitis, twisted ovarian cyst, and torsion of the great omentum also enter the clinical picture and must be excluded.

### THE TREATMENT OF ACUTE APPENDICITIS

There is general agreement that during the first 48 hours following the onset of an attack the appendix must be removed urgently.

#### APPENDICECTOMY

*The incision.* Considerable controversy still centres around the question of the best incision. Some surgeons proclaim that Battle's incision should be used for all cases. Others favour the grid-iron, while a few maintain that a paramedian incision is to be preferred. There are still other incisions which can be employed successfully. An incision is largely a matter of individual preference, and it seems unnecessary to discuss this relatively unimportant matter here. From the practical standpoint it is desirable to point out that the grid-iron approach can, if necessary, be enlarged (fig. 499) and, what is even more important, the extension can be repaired easily and effectively (fig. 500).

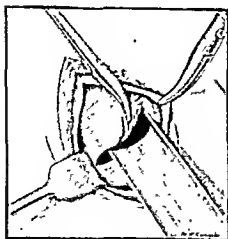


Fig. 499.—ENLARGEMENT OF THE GRID-IRON INCISION. THE INTERNAL OBLIQUE IS DETACHED FROM ITS INSERTION INTO THE RECTUS SHEATH. A PIGMENT'S DEPRESSOR IS USED TO PROTECT UNDERLYING STRUCTURES.

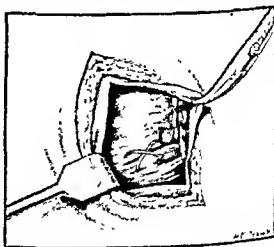


Fig. 500.—THE REPAIR OF THE PROLONGED GRID-IRON INCISION. WHEN THE SUTURING IS COMPLETED THE SCAR LEAVES A REVERSED L.

*Removal of an inflamed appendix.* As soon as the peritoneum has been opened it is a good practice to place a retractor under the medial aspect of the wound, and thereby lift up the neighbouring abdominal wall. This permits the surgeon to peer within before anything has been disturbed. It is often possible to see the cæcum and occasionally the appendix too. An important refinement of urgent appendicectomy is the isolation of the operation field by gauze packs, the better to avoid spreading the infection.

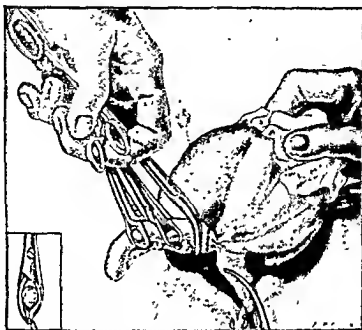
The cæcum is withdrawn. Using a swab, the cæcum is pulled gently, and usually the appendix will come into view. The finger may be inserted into the wound to aid delivery. Once the appendix has been delivered the cæcum is given to an assistant to hold. He should be instructed to take a good grip and hold the slippery structure with



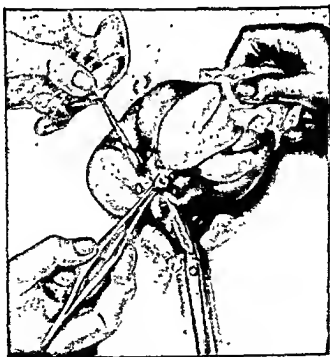
Fig. 501.—MORANT BAKER FORCEPS AND THEIR USE FOR GRASPING THE APPENDIX WITHOUT CRUSHING IT. LANE'S FORCEPS WITH THE ONE TOOTH CANNOT BE EMPLOYED SATISFACTORILY FOR THIS PURPOSE, SINCE WHEN TRACTION IS APPLIED THE MESO-APPENDIX IS TORN BY THE SINGLE TOOTH.

a gauze swab. Morant Baker forceps are applied around the appendix in such a way as to encircle the organ and yet not damage it (fig. 501). Clipping, then cutting, section by section, the meso-appendix is severed, until the base of the organ is reached. A long hæmostat is then applied to the base of the appendix. It is released, and applied again a few millimetres more distally. Around the crushed portion a ligature is applied (fig. 502), tied, and its ends cut short. A purse-string suture is inserted to encircle the caput cæci about half an inch from the appendix. This stitch passes through the muscular coat, particularly at the longitudinal bands. The purse-string having been inserted, it is momentarily left untied while the base of the appendix is severed between the hæmostat and the ligature. The free ends of the purse-string are held moderately taut whilst the appendicular stump is wiped with a gauze swab, which is cast aside promptly. The cut mucous membrane may be touched with pure carbolic. The appendix is

invaginated with a smooth small pair of dissecting forceps (fig. 503) and the purse-string is tied, burying the appendix stump. Attention is now directed to the ligaturing of the meso-appendix. For this purpose transfixion sutures are safe and cannot slip.



*Fig. 502.*—ALL IN READINESS FOR AMPUTATION OF THE APPENDIX.



*Fig. 503.*—INVAGINATION OF THE APPENDIX STUMP.

The almost universal practice of embedding the appendicular stump by a purse-string suture has been criticised by a few operators who maintain that the step is unnecessary and even harmful. We omit the step very rarely; only, in fact, when the surrounding cæcal wall is stiffened by œdema. Under such circumstances instead of the well-known ligature and a purse-string, *two* ligatures are applied to the stump.

*Retrograde appendicectomy* (fig. 504) is an excellent measure in selected cases, and it renders easy what might prove an extremely difficult operation. It is necessary to have the whole course of the appendix under vision before the operation is commenced, otherwise the tip of a gangrenous organ may be overlooked.

*Drainage of the peritoneal cavity.* In the case of operations performed within 48 hours from the onset of the attack a wise axiom is "when in doubt don't drain."

Drainage is *not* employed :

- (1) In unperforated appendicitis, however evil the organ may look.
- (2) In cases where the appendix breaks or hursts during removal.
- (3) In recent perforation with a local serous or sero-purulent exudate. By recent perforation is meant that there was no pre-operative distension or considerable acceleration in pulse-rate.

An important question to settle before closing without drainage is whether the pelvis contains an appreciable amount of purulent fluid. When in doubt a small swab on a holder can be passed downwards into the recto-vesical pouch, withdrawn, and inspected by sight and smell. In general and pelvic peritonitis a suprapubic drainage-tube inserted through a special incision above the pubis is an excellent and life-saving measure.

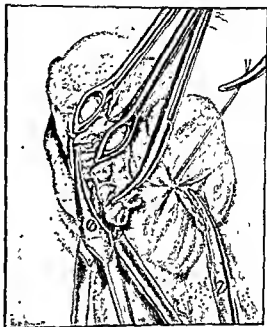


Fig 504.—RETROGRADE APPENDICECTOMY. THE BASE OF THE APPENDIX HAS BEEN DIVIDED AND THE APPENDICULAR STUMP INVAGINATED. THE APPENDIX IS IN THE COURSE OF BEING REMOVED FROM THE BASE TO TIP.



When it is necessary to drain the retrocecal space a stab incision in the flank is useful. The incision is made not too near the crest of the ilium as that area is vascular. Drainage of the retrocecal space is required comparatively rarely. Corrugated rubber is excellent drainage material in this instance.

The foregoing remarks apply to drainage of the peritoneal cavity. We now refer to drainage of the wound. In acute cases of appendicitis it is quite a good practice, as a routine, to insert a strip of glove drainage between stitches at the lower end of the wound. More or less than this is seldom indicated. In the writer's experience elaborate toilet of the wound with drainage of each layer gives no better, if as good, results.

### THE TREATMENT OF ACUTE APPENDICITIS AFTER 48 HOURS' DURATION

*A wise surgeon knows when to operate and also when not to operate.*

If, for one reason or another, the diagnosis has not been made until the third or fourth day of the disease, and there is local peritonitis or an appendix abscess, opinion is divided as to the best course to adopt immediately. At the present time surgeons are divided into two schools.

*The immediate school* advocate removal of the appendix under all conditions just as soon as possible, irrespective of the time since the onset of the attack, although even disciples of this school often stipulate "unless the patient is recovering from the attack."

*The Ochsner-Sherren (delayed) school* teach that after the forty-eighth hour of attack of acute appendicitis considerable thought should be given as to the advisability of an immediate operation. While being prepared to operate immediately, they often institute a rigid non-operative regime and only operate if the signs point to a failure of Nature to combat the infection.

### THE OCHSNER-SHERREN (DELAYED) TREATMENT

The objective of the Ochsner-Sherren school is to help to reduce the serious total mortality of appendicitis, which, at the present time, in spite of better transport, increased hospital facilities, and refined technique, shows no abatement. Not even its ardent supporters would attempt to deny that the delayed treatment of appendicitis is

open to abuse, and if abused would defeat the object of its inception. Evil lies in the abuse of all good things.

The treatment is not merely a postponement of operation; it is not just the old "interval appendicectomy"; it is unfair and inaccurate to duh the treatment as temporising; it is not a substitute for operation, hut a preparation for it—essentially a surgeon's treatment, to be undertaken only in a surgical hospital, or a correspondingly equipped nursing home, with a nursing staff trained in the method. Above all, in a civilised country, the treatment must always be carried out on the very threshold of the operating theatre. As private houses are not thus equipped, it should be regarded as criminal to attempt the treatment in the patient's home. But one can picture circumstances, for instance, in a small ship at sea—where to attempt the treatment would be less dangerous, by reason of these circumstances, than to attempt operation. At home the duty of the practitioner remains as heretofore, to arrange for the admission of the patient to hospital as soon as acute appendicitis is diagnosed—and by his example and teaching to mitigate the ingestion of purgatives in cases of undiagnosed "stomach-ache." With the advent of that Elysium when all cases of acute appendicitis are in the hands of the surgeon while the disease is still limited to the appendix the need for the Ochsner-Sherren treatment will pass.

#### SELECTING CASES FOR THE "DELAYED TREATMENT"

The history is taken, and particular note is made of the number of hours since the onset. The history begins "10, 26, 55 hours ago," not "last Thursday," or "three days ago." The physical signs are then recorded in diagrammatic form. The extent of the rigidity is marked by shading; the presence of a lump is drawn as near as possible to scale. The presence or absence of hyperæsthesia is always recorded, and the findings of a rectal examination are not omitted. It is necessary to include these particulars, for it is impossible to proceed without minute attention to detail, for fear of being misunderstood.

If the diagnosis of acute appendicitis is made and the history is of under forty-eight hours' duration, immediate operation is nearly always advised. If the diagnosis of acute appendicitis is made and the history is of over fifty hours' duration, one should ask oneself the question, "Is there any reason why this appendix should be removed at once?" The answer by one trained in the delayed treatment is "It is safer to postpone operation for the time being, unless . . ."

## EXCEPTIONS TO THE RULE

- (1) Hyperæsthesia is present. Providing that the other signs are consistent this may be taken as good evidence that the appendix is still unperforated.
- (2) Age under 10 years.
- (3) The diagnosis cannot be made between acute appendicitis and some other intra-abdominal catastrophe normally requiring immediate operation—particularly perforated diverticulitis and perforated duodenal ulcer.
- (4) General peritonitis has supervened. Only cases which have obvious general, as opposed to pelvic, peritonitis are excepted.
- (5) The recent ingestion of a powerful purgative may be a justifiable indication for performing an operation which otherwise would be delayed.

## TECHNIQUE OF THE TREATMENT

The patient is placed and nursed in high Fowler's position (fig. 505).

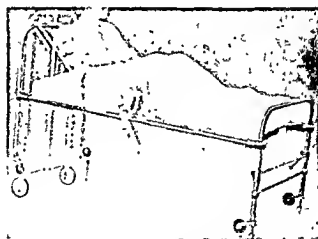


Fig. 505.—HIGH FOWLER'S POSITION. THE HEAD OF THE BED IS RAISED 15 INCHES. HOKIN'S AND SEWELL'S BED-LIFTER IN THE WOODEN BLOCKS CAN BE USED.

*Charts.* As a routine, the pulse is recorded every two hours in graphic form on a special chart (fig. 506). In cases where anxiety is felt as to the advisability of continuing the treatment, an hourly chart is employed. Temperature is relatively unimportant, and it is recorded every four hours. Instructions are given to the nurse to

record any vomiting on a separate piece of paper, known as a "vomit chart." On this is entered the time at which the vomits were ejected, together with the quantity and character of the fluid.

*Diet.* Water only is given for four days, or occasionally longer. This is adhered to very strictly. The amount of plain cold water the patient may have is unlimited, although he is not encouraged to drink more than is necessary to satisfy his thirst.

On the fifth day, if the pulse and temperature are satisfactory and the patient feels hungry, feeding is commenced. Small feeds of Benger's food, alternating with a cup of Bovril, are given. On the sixth day custard and jelly are allowed. After that the diet is gradually increased.



Fig. 500.—ACUTE APPENDICITIS OF FIFTY-TWO HOURS' DURATION, OCHSNER-SHERREY TREATMENT, RESOLUTION.

*Drugs.* All drugs are forbidden. It should be noted particularly that no morphine or its derivatives are given.

Pain, as opposed to tenderness, is very seldom complained of after the first night of the treatment. A hot bottle may be given to the patient to apply to the abdomen; but it is well to repeat that as long as the inflamed appendix remains *in situ* drugs are forbidden, for they may mask those all-important signs which foretell that the delayed treatment is not likely to succeed.

*Bowels.* The bowels are left confined if they are not opened naturally. On the fourth or fifth day a small glycerol enema is given. No purgatives of any kind are given until resolution is complete—that

is, until the temperature and pulse have been normal for a week and pain and physical signs are absent—then liquid paraffin, 2 drachms thrice daily, is prescribed.

#### WATCHING FOR NATURE'S FAILURE TO COMBAT THE INFECTION

Instructions are given for the nurse in charge to watch the patient and report immediately ;

- (1) A rising pulse-rate,
- (2) Vomiting,
- (3) Pain, and in the later stages of the treatment
- (4) Diarrhoea or the passage of mucus in the stools (pelvic abscess).

A rising pulse-rate in the early stages is the most reliable single sign that it is dangerous to proceed with the delayed method. If the pulse-rate has increased even ten points in the first twenty-four hours, operation is often indicated.

Vomiting after the first few hours should always be regarded seriously, and this by itself may be a sufficient indication to abandon delayed treatment.

A patient undergoing delayed treatment should not complain of pain, as opposed to tenderness, after the first six hours of such treatment. If he does, usually there is something wrong, and this is a strong indication for operation.

#### THE OUTCOME

Under the delayed treatment most cases resolve without incident, and the appendix is duly removed after the acute stage has abated. In a few, where the signs point to failure of the delayed treatment, urgent appendicectomy must be undertaken.

#### APPENDIX ABSCESS

"An appendix abscess" signifies a localised abscess connected with a perforated or otherwise inflamed vermiform appendix. A sub-diaphragmatic abscess or a pelvic abscess can arise from the same source, but these conditions are not usually categorised under this heading, and rightly so. The signs of an appendix abscess are those

of acute appendicitis plus the presence of a tender swelling. When a lump is present early in an attack of acute appendicitis it is probable that the bulk of the swelling is due to a mass of great omentum performing its constabulary duties.

*Treatment of appendix abscess.* The same controversy exists in the case of appendix abscess. The immediate school proclaim an old and usually wise surgical axiom, "where there is pus you must let it out." The delayed school state that the rule may be broken in the case of small and moderate-sized appendix abscesses, and the abscess should only be opened if it is getting larger or fails to resolve. They find that the appendix abscesses often resolve completely under the Ochsner-Sherren regime, and when the time comes for the appendix to be removed (in about three months) there is usually a remarkable freedom from adhesions.

In a consecutive series of 75 cases of acute appendicitis with a localised palpable mass treated by the author by the Ochsner-Sherren method, in exactly 60 or 80 per cent the abscess resolved. In one case out of the 75 the abscess burst into the general peritoneal cavity, but because the patient was being treated on the threshold of the operating theatre prompt drainage saved his life. In 9 cases the abscess did not resolve and drainage was carried out. There was but one death in the 75 cases and that was the fourth in the series. This occurred before I realised that it was necessary to allow two and sometimes three months to elapse between the resolution of the abscess and the appendicectomy.

If delayed treatment has been tried the number of appendix abscesses which need drainage will be comparatively small. Those that do require evacuation of pus will be frank abscesses, and the technique may be reduced to the simplest character, for the question of removing the appendix at the time of the evacuation of the pus never arises.

#### DRAINING AN APPENDIX ABSCESS SITUATED IN THE RIGHT ILIAC FOSSA

*Anæsthetic.* Gas and oxygen or evipan usually suffices.

*Technique.* The swelling is palpated under the anæsthetic. A point is chosen about the centre of the swelling, but rather nearer the

lateral than the medial aspect. A small incision is made ; small because it is naturally very prone to become infected, and there will be less of it to break down. The peritoneum is opened. More often than not, opening the peritoneum does not open the abscess. Every care should be taken to avoid breaking adhesions unnecessarily, especially on the medial side. The extremity of a length of gauze may be packed gently into the *mesial* part of the wound. The index finger is passed into the wound, and very, very gently burrows laterally and backwards. In the case of a large abscess it is hardly a moment before the finger is felt to enter a large cavity. The finger is still kept *in situ* and acts as a bung to the flow of stinking pus until a tube can be passed into the abscess cavity. The patient is nursed in high Fowler's position and a pillow placed under his left loin in order to keep him on his right side and thus invoke the aid of gravity. After the first forty-eight hours the tube is turned and shortened. It is usually omitted altogether on the sixth day. There are seldom any complications if a frank abscess has been drained.

#### PELVIC ABSCESS WITH SPECIAL REFERENCE TO APPENDICITIS

Pus can accumulate in this area without serious constitutional disturbance, and unless the patient has been examined carefully from day to day such abscesses may attain considerable proportions before being recognised. The most characteristic symptoms of a pelvic abscess are diarrhoea and the passage of mucus in the stools. It is no exaggeration to say that the passage of mucus occurring for the first time in a patient who has, or is recovering from, peritonitis is pathognomonic of pelvic abscess. Rectal examination reveals a bulging of the anterior rectal wall (fig. 507) which, when the abscess is ripe, becomes softly cystic.

*Rectal drainage.* Left to Nature, a proportion of these abscesses burst into the rectum, after which the patient nearly always recovers rapidly. It is far too risky to wait for this possible happy termination. When the abscess is definitely pointing into the rectum rectal drainage can be employed.

I have found this method exceedingly efficacious in selected cases, but have occasionally first opened the abdomen in order to be quite certain of the diagnosis. Providing the abscess is shut off from the general peritoneal cavity, a point which can be ascertained

undeniably when the abdomen has been opened, rectal drainage of a pelvic abscess is preferable to suprapubic drainage, which in many cases unavoidably breaks down Nature's barriers, and exposes the general peritoneal cavity to the dangers of spreading infection. I have used this method in a large number of cases, and never with regret.

*Technique.* The first thing to do is to empty the bladder with a catheter immediately prior to the anæsthetic. Low spinal anæsthesia is sufficient, or evipan may be used. The patient is placed in an

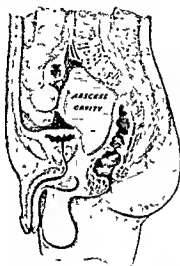


Fig. 507.—A PELVIC ABSCESS ABOUT TO POINT INTO THE RECTUM. MANY OF THESE ABSCESSES ARE VIRTUALLY EXTRA-PERITONEAL.

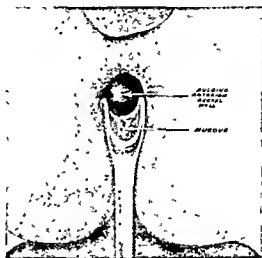


Fig. 508.—ALL IS IN READINESS FOR OPENING A PELVIC ABSCESS INTO THE RECTUM.

exaggerated lithotomy position. The small end of a Sim's duck-billed vaginal speculum, which answers the purpose admirably, is introduced (fig. 508). The passage of some mucus when the speculum has been inserted is very characteristic. I use an ordinary long hæmostat to penetrate the rectal wall. This is not done by a sudden jab, but by even pressure. As soon as the abscess is entered pus streams down the speculum. A suitable piece of drainage-tube is inserted into the abscess cavity and anchored to the anal verge by a single stitch. The tube is removed on the fourth day. As a rule the patient previously had diarrhoea; once the abscess is opened this ceases, and if possible the bowels should remain confined until the tube is removed. The patient is nursed in high Fowler's position for a week.



## ACUTE APPENDICITIS WITH GRAVE GENERAL PERITONITIS

When the pulse is feeble and in the neighbourhood of 140, and the abdomen drum-like, or other combinations of signs make it undeniable that the peritonitis is frankly general, no one will deny

that the interests of the patient are best served by adopting a conservative attitude, for the time being. One of the greatest life-saving measures in such cases is the prompt administration of continuous intravenous saline with glucose (fig. 509), to which may be added anti-gas gangrene serum. If vomiting is in evidence, the contents of the stomach should be aspirated. In all but the moribund some improvement in the general condition takes place, and in a few hours the patient is again examined and the facts of the case

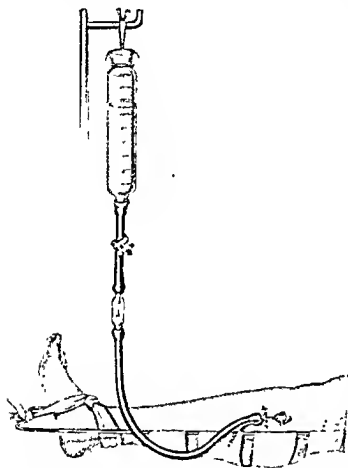


FIG. 509.—CONTINUOUS INTRAVENOUS INFUSION. THE LEG IS CHOSEN IN THE CASE OF A PATIENT WHO IS RESTLESS OR MAY BECOME SO, FOR THE LOWER LIMB IS IMMOBILIZED READILY BY A SPLINT.

are reviewed. In deciding the momentous question what to do for the best the following résumé may be taken into consideration.

The day is not long past when it was considered that the patient's only chance was to operate, above all to drain freely. Close attention to the subject has shown that the problem before us is not nearly so simple as this. It will be recalled that Ochsner himself first framed his method for cases of spreading peritonitis. There is plenty of evidence that certain cases of this type respond to the method. The

treatment aids Nature in transforming the general peritonitis into a localised infra-umbilical or pelvic collection of pus, which can then be treated safely by simple drainage.

Experience teaches us that simple drainage without removal of the appendix *before this localisation has occurred* rarely saves the patient's life. Indeed, in many instances it appears to hasten the end. On the other hand, in most cases the *expeditious* removal of the appendix together with suprapubic drainage is, I believe, the best line of treatment in perforated appendicitis with general peritonitis, but the operation must be expeditious and the anaesthesia as perfect as possible. Should the operation prove to be difficult, necessitating considerable intra-abdominal manipulations, or if the attempt to remove the appendix has to be abandoned in favour of simple drainage, death usually follows. Under these circumstances one must be prepared to state that such a case might have been saved by the delayed method. Each case must be treated on its merits. In two circumstances operation should always be undertaken as soon as the condition of the patient permits.

- (1) *In childhood and early adolescence.* Localisation of a general peritoneal infection does not occur often enough in early life to warrant a trial of the delayed method unless the circumstances are extenuating.
- (2) *If there is good reason to believe that the general peritonitis is due to the recent bursting of an appendix abscess.* Here simple drainage is a life-saving measure.

#### APPENDICITIS IN PREGNANCY

Acute appendicitis is a not infrequent and a supremely important complication of pregnancy, and if only the diagnosis can be established within forty-eight hours from the onset appendicectomy can be undertaken safely. If the intra-abdominal manipulations can be reduced to a minimum the pregnancy is not necessarily interfered with. Abortion occurs in about 20 per cent when operation has to be undertaken during the first three months of pregnancy. After the third month the liability to abort becomes increasingly less.

The chief difficulty in dealing with appendicitis complicated by pregnancy is to establish the diagnosis.

### SOME COMPLICATIONS OF ACUTE APPENDICITIS NOT CONSIDERED ALREADY

*Pylephlebitis.* Acute appendicitis is the commonest cause of pylephlebitis which, even so, is a very rare complication. To have seen an otherwise favourable case of appendicitis slowly succumb to the ravages of portal pyæmia is to realise fully that some systematic effort to prevent its occurrence should be made.

A patient with appendicitis, who has rigors early in the attack and

*before operation*, may be fairly assumed to be in imminent danger of pylephlebitis. It is in such cases that ligation of the ileo-colic vein is indicated. The vein should be ligated before the appendix is removed. The cæcum is drawn well out of the wound and the superior ileo-cæcal angle sought. The vein is located and traced upwards about two inches, where it will be found to be joined by other cæcal branches. Using a Watson Cheyne dissector, the vein is isolated from its peritoneal covering (fig. 510) and, taking care not to include the artery, is ligated.



FIG 510 THE PREVENTION OF PYLEPHLEBITIS. ISOLATING THE ILEO-COLIC VEIN PRELIMINARY TO PLACING THE LIGATURE.

*Internal faecal fistula.* Post-graduates will be familiar with the possibility of an external faecal fistula following acute appendicitis with peritonitis. The patient who develops such a fistula rarely dies, since the faecal fistula acts as an enterostomy. The reverse is the case when the fistula is internal, and J. M. Petty has done a service in calling attention to this condition. Internal faecal fistula should be suspected when the patient suddenly develops signs of general peritonitis about the fourth day after appendicectomy. A large enema is frequently the determining cause and the appendicular stump bursts open.

Once the condition has developed, the only possible treatment is immediate operation and the conversion of the internal fistula into

an external one. This complication, which is probably not as rare as may be thought, could be prevented by sewing a catheter into the cæcum in those occasional cases where the cæcum is much distended and the tissues about the appendix stump œdematous.

*Paralytic ileus.* It is not our intention to discuss this condition fully here. A review of monographs upon the subject shows that a large proportion of cases of paralytic ileus follow operations for acute appendicitis. Furthermore, close observation will reveal that the majority of cases of this complication are a sequel to an operation performed at such a time and upon such a case as the Ochsner-Sherren school eschew. In my own practice, since I adopted the Ochsner-Sherren treatment in selected cases of acute appendicitis, the nightmare of paralytic ileus has been almost banished.

*Problem.* After an operation for acute appendicitis the condition of the patient is unsatisfactory. The temperature is swinging and the pulse is elevated—signs which foretell the pocketing of pus.

*A symposium of methods to be adopted for its elucidation.*

- (1) Examine the scar or wound and the abdominal wall for an abscess of the abdominal wall.
- (2) Consider the possibility of a pelvic abscess.
- (3) Palpate the left iliac fossa for an abscess in this situation.
- (4) Examine the loin for a perinephric abscess.
- (5) Look at the legs—to exclude the possibility of phlebitis.
- (6) Examine the conjunctivæ for an icteric tinge and the liver for enlargement, and inquire if the patient has had rigors—pyelophlebitis.
- (7) Examine the lungs—pneumonia or empyema.
- (8) Examine the urine for organisms (pyelitis), and the feces for blood and pus (proctitis or enteritis).
- (9) Lastly, concentrate diagnostic endeavour upon the possibility of a sub-diaphragmatic abscess.

#### SUB-ACUTE APPENDICITIS

Sub-acute appendicitis is but a mild form of acute appendicitis and requires no detailed consideration.

## RECURRENT APPENDICITIS

Appendicitis is notoriously recurrent. This is perhaps the commonest form of appendicitis—mild sub-acute attacks which are so often attributed to “biliousness” or a “chill on the liver.” The attacks vary in intensity, and the majority of cases ultimately culminate in severe acute appendicitis. If careful histories are taken from patients with acute appendicitis, over two-thirds remember having milder but similar attacks of pain. This bespeaks the importance of recurrent appendicitis as a precursor of the more serious lesion.

## CHRONIC APPENDICITIS

One should be careful to distinguish recurrent from chronic appendicitis. Many cases called “chronic appendicitis” are typical examples of the recurrent form of the disease. Chronic appendicitis is a comparatively rare affection; it certainly exists, and its most typical symptoms are referred to the stomach and duodenum—appendicular dyspepsia.

*Pathology.* Appendices removed from patients suffering from true appendicular dyspepsia usually show a characteristic macroscopical change. There is obliteration of the lumen commencing at the tip and spreading along the organ for a variable distance (fig. 511). The walls of the obliterated portion can be seen to be composed almost entirely of white fibrous tissue. In long-standing cases, the greater part of the organ is attenuated from fibrous contracture. In a few cases, the fibrous changes are seen in the proximal end, but this is more characteristic of the recurrent type of the disease.

*Diagnosis* is difficult. One should remember constantly that in chronic appendicitis there are often no signs in the right iliac fossa—only referred symptoms elsewhere. We should be watchful lest, perchance, we fall into a common error of classing as chronic appendicitis neurotic patients (usually female) with a ptosed, gurgling, and apparently tender cæcum. Removal of the organ brings neither permanent relief to the sufferer nor credit to the surgeon.

*Radiology as an aid to diagnosis.* In the case of the vermiform appendix, radiology is not a great diagnostic aid. If the appendix

cannot be visualised after an opaque meal it suggests that its lumen is obstructed. If it fills and empties, it is indicative that the organ is healthy—but as it is impossible to tell the length of a given appendix until the organ has been displayed, there must always be uncertainty in the radiological diagnosis of appendicitis.

### ACTINOMYCOSIS OF THE RIGHT ILIAC FOSSA

It is not always possible to state definitely if the infection begins in the appendix or in the cæcal wall. That the appendix is often the original focus is suggested strongly by the operative findings in Type 1.

*Type 1.* Appendicitis is diagnosed, and an inflamed appendix is removed. The wound continues to discharge for weeks. A consultation is held; a wise clinician suggests the possibility of actinomycosis. After perhaps many examinations of the pus, sulphur granules are found.

*Type 2.* A patient comes with a hard mass in the right iliac fossa. It is often extremely difficult to differentiate between carcinoma of the cæcum, hypertrophic tuberculosis, and actinomycosis, even after the abdomen has been opened.



Fig. 511.—CHRONIC OBLITERATIVE APPENDICITIS.



Fig. 512.—DIVERTICULOSIS OF THE APPENDIX.

The *treatment* is to persevere with the iodine in milk therapy as in cases of actinomycosis, and to provide drainage when abscess formation occurs. Autogenous vaccines may also be tried. Except when the disease is recognised at an early stage, the prognosis is extremely poor. Multiple sinuses, pelvic abscess, and faecal fistulae are common complications. Sometimes the wounds heal, only to break down later—eventually the infection proves fatal in fully 80 per cent of cases.

## VERMIFORM APPENDICULAR DIVERTICULOSIS

Diverticula of the appendix are not rare, and have been seen in otherwise diseased appendices. The diverticula (fig. 512) may be due to pressure in an obstructed organ, but in specimens we have examined the cause of the diverticulosis has not been apparent.



Fig. 513.—CARCINOID TUMOUR (FALSE CARCINOMA) OF THE APPENDIX.  
(After Lee Wilson.)

## CARCINOMA OF THE APPENDIX

Carcinoma of the vermiform appendix is found in 0.39 per cent of all appendices removed. It is highly important to recognise two forms of disease.

*Carcinoid tumour* (false carcinoma of the appendix) always occurs at the tip, forming a solid bulbous extremity to the organ (fig. 513). This growth is usually found in young females, and when a pathological report of carcinoma is returned there is often much unnecessary anxiety. Carcinoid tumour of the appendix is entirely benign, and although difficult to distinguish from true carcinoma histologically, it does not give rise to metastases.

*True carcinoma of the appendix* usually occurs at the proximal end of the organ, and is intimately related to carcinoma of the caecum. When a carcinomatous mass has formed in the region of the caecum it is often impossible to state precisely where the growth originated, even after a careful pathological examination.

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SECTION 11

COLON

by

CECIL P. G. WAKELEY

## SECTION II

### COLON

THE large howel differs from the small intestine in structure and function, and correspondingly so do its surgical affections and operative technique. The colon in itself is also divisible into two halves—right and left. These have different blood supply, function, and emhryology. The cæcum, ascending colon and hepatic flexure are derived from the mid-gut and are supplied by the superior mesenteric artery, which is the artery to the mid-gut. The splenic flexure, descending and pelvic colon are derived from the hind-gut, and are supplied by the artery to the hind-gut—the inferior mesenteric artery. The right half of the colon is concerned with ahsorption, and the left with storage and excretion (fig. 514).

In contra-distinction to the small intestine, the colon is relatively immohile, has a poorer blood supply, while its contents are swarming with bacteria, all points of importance reflected in the technique of operations. Cancer, rare in the small gut, is common in the colon, nearly 70 per cent of all surgical lesions of the colon being malignant. No satisfactory explanation of this fact is forthcoming, hut it is significant to notice that the reaction of the chemical change is from alkaline in the small intestine to acid in the colon—as in the stomach—the two ends of the gut where carcinoma is common.

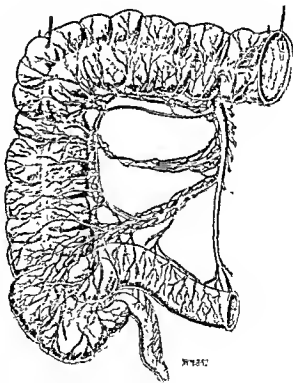


Fig. 514.—THE BLOOD AND LYMPH SUPPLY TO THE RIGHT HALF OF THE COLON.

It may be said that the surgeon will operate on the colon for malignant disease, diverticulitis, tuberculosis, faecal fistula, in that order, interspersed with occasional operation for ulcerative colitis with complications, polyposis, and benign new growths.

In this chapter the various surgical affections will first be detailed from the clinical and operative aspect, while at the end of the chapter the various special operative manoeuvres will be described in detail. No attempt at pathological classification or full description of signs and symptoms has been made. The subject has been approached from the attitude of the practising surgeon. Indications for operation and the various types of operation with their respective application have been stressed.

#### CONGENITAL AFFECTIONS OF THE COLON

These are not very common as a whole, nor when occurring do they generally cause symptoms. From those that are important and may call for surgical intervention the following are selected :

*Congenital Atresia of the Colon.* This is seldom complete and generally affects either the ascending or descending portions. The part affected is present as a fibrous cord. The treatment is urgent laparotomy with the performance of a lateral anastomosis *secundum artem*, rather than colostomy or cecostomy.

*Congenital Mesenteries in the Colon.* These are not uncommon, and may be present for either the ascending colon and caecum (25 per cent of cases), or the descending and iliac colon (35 per cent of cases). They are of surgical interest in that they allow increased mobility to the bowel and increase the risk of volvulus. In fact, volvulus of the caecum is probably never seen apart from this abnormality of its mesentery. The presence of such a mesentery in the iliac colon is of great help to the surgeon when attempting the resection of growths in this region.

*Megacolon.* For a discussion of this congenital affection of the colon see under the section in Sympathetic Nervous System.

#### ULCERATIVE COLITIS

It is not proposed in this work to deal with the diagnosis of colitis. This is usually the work of the physician, while the surgeon is often

only called in to advise treatment. It is important to emphasize, however, that an early attempt to determine the nature of the infecting organism should be made. It is important to exclude members of the dysentery class of bacillus. Sigmoidoscopy is a valuable adjunct in this respect, and is more helpful than simple plating of the faeces. Swabs may be taken directly from the floor of the ulcer, as seen through the sigmoidoscope, and directly plated. Sigmoidoscopy is also of value in estimating response to treatment and in anticipating sequelæ.

The value of surgery in the treatment of ulcerative colitis is much disputed. Out of the various discussions and from the literature the following facts emerge :

(1) Surgery is useless as a final resort when the patient is desperately wasted, anæmic, and dehydrated.

(2) Of the various surgical methods advocated, i.e. short-circuit operations, colostomies, etc., only appendicostomy, cæcostomy, and ileostomy have survived. In England appendicostomy and cæcostomy are preferred, in America ileostomy is favoured.

(3) The value of appendicostomy in some cases is shown by the fact that patients who have improved after the performance of the appendicostomy have relapsed after its injudicious closure.

(4) From this it follows that openings thus made should be kept for at least six months after the subsidence of all symptoms.

American investigators claim to have isolated a diplo-streptococcus as a causative organism in many cases of colitis and have with this prepared an anti-serum. Promising results have been obtained with this serum known as Barger's serum, and with rectal oxygen insufflations. Sufficient oxygen must be given to distend the bowel gently.

If such and other medical measures fail to relieve the condition, appendicostomy is worthy of trial. After its performance we advise daily irrigations with warm saline. There is no advantage in using antiseptic solutions. Appendicostomy is performed in the following manner :

Local anæsthesia is sufficient in many instances. The McBurney incision is the best (see section on "Diseases of the Appendix and Methods of Appendicectomy"). The abdomen is opened exactly as for appendicectomy, and the cæcum and appendix are delivered. The peritoneum is sutured to the cæcum around the base of the appendix. Care is taken to preserve the mesentery of the appendix. The wound is closed in the normal manner. After 72 hours, the appendix is cut

off down to the last inch, which is then split longitudinally and the flaps anchored back (see fig. 515). This stage is performed with a diathermy or electric cauter.

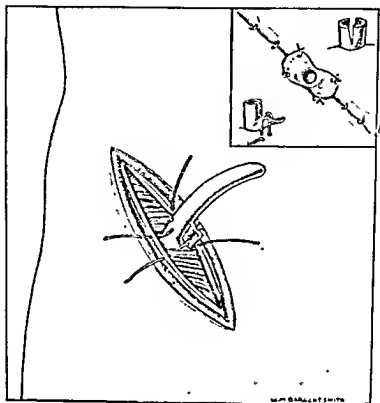


Fig. 515.—APPENDICOSTOMY.

- (a) The base of the Cecum is sutured to the Peritoneum.  
 (b) Special Method of Dividing the Appendix and suturing flaps.

Appendicostomy is preferred to cecostomy or ileostomy because :

- (1) It is easily and quickly performed.
- (2) It has a negligible mortality.
- (3) There is minimal faecal leak and no odour.
- (4) It is a satisfactory method of irrigation of the large bowel.
- (5) It seldom leaves a fistula, and if it does, this fistula can readily be closed. If there is any difficulty in finding the appendix in these cases cecostomy should be done in preference to much intra-abdominal manipulation. (For technique see the end of the chapter.)

Urgent surgery may be called for in cases of perforation of an ulcer. In these cases, as well as suture of the perforation, cecostomy or ileostomy should be performed to rest the colon. If the perforation is large and ragged and difficult to suture, then that segment should be

exteriorised and a local colostomy performed. The prognosis is extremely bad in such cases.

The sequelæ of ulcerative colitis may call for surgical measures. The commonest are pseudo-polypoid—12 per cent of cases, and stricture—10 per cent of cases. Polypi occurring within reach of a sigmoidoscope are best dealt with by diathermy. Those occurring higher in the bowel are less likely to cause symptoms (bleeding) or undergo malignant changes. Stricture commonly occurs in the sigmoid or recto-sigmoid region and is best dealt with by excision where possible. The other forms of colitis and the dysenteries do not call for surgical treatment except for their complications or sequelæ—perforation or stricture.

#### TUBERCULOUS INFECTION OF THE COLON

The tubercle bacillus may affect the colon in miliary tuberculosis, in tuberculous ulcerative colitis, and in ileo-cæcal hyperplastic disease. The first condition is non-surgical. Tuberculous ulcerative colitis may present itself to the surgeon by the formation of peri-colic abscesses or fæcal fistulæ. If possible a short-circuit operation, ileum to sigmoid colon, should be performed, the primary focus being left untouched.

Ileo-cæcal hyperplastic disease affects adults aged 20–40 years (a rather younger age-incidence than carcinoma, a disease it closely simulates) and males rather more frequently than females. The lesion appears to be primary, though search for other tuberculous foci should always be made. The symptoms are protean; pain, diarrhœa, loss of weight, anæmia, hemorrhagmi, are amongst those listed. Patients frequently complain of a palpable tumour which they have discovered. A case may present itself as one of acute intestinal obstruction. Rarely abscess formation and fistulæ occur. A barium enema will reveal a filling defect in the cæcum.

Three operations have been used in the treatment of this condition: Ileostomy, ileo-transverse colostomy, and excision with anastomosis. The last operation is the method of choice. Short-circuit operations are reserved for cases where an abscess or fistula make resection impossible. Ileostomy may be used either as a preliminary or adjuvant measure. Resection with anastomosis is performed exactly as for carcinoma of the cæcum (see page 963).

#### ACTINOMYCOSIS

Three distinct clinical varieties of actinomycotic infection of the cæcum occur. The commonest is the occurrence of a persistent sinus

after appendicectomy, the appendix and caecum at the time being the site of actinomycosis. The condition is not discovered until 'sulphur' granules are found in the pus on the dressings. If feasible, the sinus should be excised together with a portion of the wall of the caecum. The second variety, clinically, is the case where a tumour in the right iliac fossa is discovered. Multiple sinuses may complicate such a case. The third variety is the pyæmic case where an abscess has ruptured into a vein. Surgical treatment consists either in incision, drainage and curettage of abscesses as an adjuvant to medical measures, or in resection. Unfortunately, cases suitable for resection present themselves very rarely. Occasionally, resection in a case diagnosed as carcinoma proves it on section to be actinomycosis. When this occurs the prognosis is more favourable.

### VOLVULUS

Volvulus, a condition of obstruction of the bowel due to torsion of a loop, most frequently affects the sigmoid colon. This is due to its long mesentery with a very short angulated base. The caecum may also be the site of volvulus, particularly when a mesentery exists for the ascending colon. Volvulus in other parts of the colon has also been described. The essential factors for its production are excessive mobility of the bowel with fixation at some particular point. This fixation is usually provided by an abnormal band (inflammatory or congenital) or it may be by the mesentery itself. Volvulus may occur at any age, but it is most usual in the third and fourth decades, and more frequently in the male subject. The condition may be acute or chronic. Chronic volvulus usually presents a clinical picture of repeated attacks of sub-acute intestinal obstruction. It is usually not diagnosed until exploration is performed.

### ACUTE VOLVULUS

Acute volvulus is one of the most urgent surgical conditions in the abdomen. Distension is severe, and vomiting (reflex, not obstructive) is experienced early in the condition. Collapse is marked. The procedure we recommend is as follows:

The anæsthetic of choice is nitrous oxide, oxygen, ether anæsthesia, preferably with an intra-tracheal tube, as respirations are frequently embarrassed. A large left lower paramedian incision is made, the peritoneum is opened, the diagnosis confirmed and the site of the

volvulus noted. If the volvulus is of the usual sigmoid variety, the next step is to get an assistant to pass a flatus tube per rectum, while the operator attempts to guide it into the efferent loop of the howel. Should the manœuvre be successful the loop can be emptied of gas and the operation be thus greatly simplified. If, however, this cannot be done the surgeon should empty the loop by puncturing the bowel with a trocar and cannula. Under no circumstance should the loop be

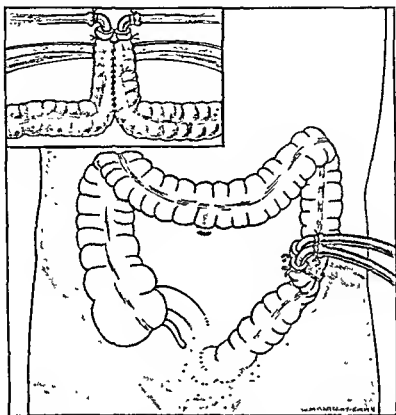


Fig. 516.—DIAGRAM SHOWING METHOD OF JOINING BOWEL AND INSERTION OF PAUL'S TUBES AFTER RESECTION.

delivered in its distended state as fatal collapse may be produced by so doing.

Once the loop is emptied attention is directed to a consideration of its detorsion and viability. If detorsion is easy and the bowel is viable, we recommend that a large catheter be embedded in its lumen, the catheter being brought out through a separate stab wound made from within the abdominal cavity and outwards from the main incision. This has the two-fold advantage of draining the gut of its highly toxic contents and also of anchoring the loop as there is a definite risk of recurrence.

If the howel is gangrenous a rapid resection is performed, the two



ends of the bowel are brought to the surface, and Pnul's tubes tied in them (fig. 516). It may be advisable to bring these out through a separate flank incision to avoid tension on the bowel. Furthermore, this allows better closure of the main incision. The abdomen is drained in all cases. Under no circumstances should an attempt be made at restoration of bowel continuity or plastic operation to prevent recurrence.

#### DIVERTICULITIS

The incidence of sacculations in the colon, usually multiple, is high; probably about 5 per cent of all people over forty years of age.

This condition is, however, termed diverticulosis and is not productive of symptoms. The sacculations most commonly form along the line of entry of the blood-vessels, i.e. at the sides of the mesenteric attachment; they may, however, enter the appendices epiploicae. The sigmoid colon is easily the commonest part of the colon for their occurrence (fig. 517). If the diverticula become the site of inflammatory change, usually due to irritation from stagnation of their contents, then the condition is productive of symptoms, and is termed diverticulitis; this may call for surgical measures.

Diverticulitis may manifest itself clinically in the following ways:

- (1) Acute diverticulitis. (Left-sided appendicitis.)
- (2) Chronic diverticulitis with obstruction.
- (3) Diverticulitis with peri-colic abscess formation and external fistula.
- (4) Diverticulitis with internal fistula.



Fig. 517.—DIVERTICULITIS. PORTION OF BOWEL REMOVED FROM SIGMOID COLON.

In our opinion there is no causal relationship between diverticulitis and malignant disease, the cases of both conditions which have been described being accidental (fig. 518).



*Fig. 518.*—SCLEROGRAM TAKEN AFTER A BARIUM ENEMA SHOWING DIVERTICULITIS OF THE COLON. A WELL-MARKED CASE. THIS CASE WAS KEPT UNDER OBSERVATION FOR FIFTEEN YEARS AND DID NOT DEVELOP CARCINOMA.

## TREATMENT

(1) For acute diverticulitis the treatment is simple, viz. laparotomy, excision of the diverticulum, and suture of the colon. If numerous other diverticulæ are seen it is usually advisable to perform a temporary colostomy (see page 969) as well, and thoroughly irrigate the bowel during recovery.

(2) Diverticulitis may lead to chronic, or acute on chronic, intestinal obstruction. This is best treated by laparotomy—confirmation of diagnosis—cecostomy or transverse colostomy, and bowel lavage, followed two months later by resection of the sigmoid (see page 963).

(3) The abscess should be drained at the first operation, and the various steps as outlined above in (2) performed.

(4) The treatment of internal fistulæ, whether vesico-colic or entero-colic, consists first in a colostomy and later in excision of the fistula and repair of the viscus.

## THE APPENDICES EPIPLOICÆ

The appendices epiploicæ may be the site of torsion. This is one of the uncommon conditions causing an acute abdomen. Sometimes the appendage is completely separated, resulting in a natural cure. This leaves a loose fibro-fatty body free in the general peritoneal cavity. Treatment is simple, consisting in ligation of the pedicle and removal of the appendage.

## NEW GROWTHS OF THE COLON

As has already been stated, about 70 per cent of all surgical procedures for colon disease are performed for new growths, of which the vast majority are malignant. The trend of advance in recent years in colon surgery has been along the lines of early and accurate diagnosis, with a return to the multiple-stage type of operation, and an abandonment of attempts at aseptic intestinal anastomosis in the presence of intestinal obstruction.

*Benign New Growths.* These present themselves, clinically, in one of two groups. They are:

(1) The uncommon benign growths (single adenomata, lipomata, angiomas, and myomata) which present to the surgeon because of the complication of intussusception or obstruction.



Fig. 519.—SPECIMEN SHOWING  
MULTIPLE POLYPI OF THE COLON.  
(By kind permission of  
Mr. L. E. Norbury.)

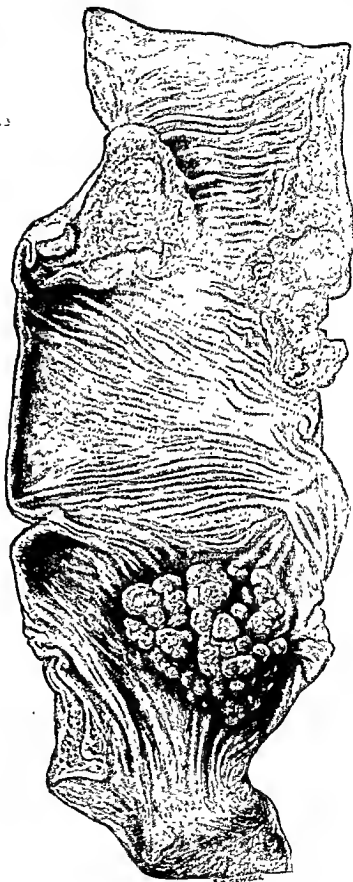


Fig. 520.—CARCINOMA AT THE RECTO-SIGMOID JUNCTION WITH VILLOUS  
PAPILLOMA OF THE RECTUM.  
(By kind permission of Mr. L. E. Norbury)

(2) The condition known as polyposis coli, i.e. multiple pedunculated tumours, which calls for treatment because of pain, bleeding, or diarrhoea, or a combination of these symptoms (fig. 519).

The first group can easily be dismissed. Their treatment is that of the condition which has led to their discovery combined with a local removal of the tumour. If discovered accidentally they should always be removed, as sooner or later they may occasion obstruction or intussusception, while there is always the risk of malignant change. They may readily be removed by transperitoneal colotomy.

The condition of polyposis is more difficult from the point of view of treatment. The multiplicity of the lesions and their proneness to malignant changes (50 per cent of cases), which may occur in more than one tumour in the same case, render attempts at treatment very difficult. Cases, however, do occur where the condition appears to be limited to one portion of the colon (fig. 520).

In these cases resection is the method of election, in the hope that subsequently recurrence higher up in the colon will not take place. Growths in the rectum and lower sigmoid (the most frequent site) can sometimes be dealt with by diathermy through the sigmoidoscope. If one tumour appears to be growing more rapidly than the others, attention should be paid to removal of this portion of the bowel, as some observers have noticed the other growths to regress under these circumstances.

It should be understood that the above remarks are not meant to apply to the condition of polyposis following ulcerative colitis. In this condition malignant changes are less common.

#### CARCINOMA OF THE LARGE BOWEL

Carcinoma of the colon is a condition essentially suited for surgical treatment. In the vast majority of cases the tumours are slow growing and metastasise late. The actual size of the tumour bears, if anything, an inverse relationship to its malignancy, the fact of increase in size locally apparently being evidence of a good resistance on the part of the patient. This has led to increasing courage in operative treatment. Growths which used to be considered "inoperable" because of local attachments or size are now removed with gratifying results. Multiple resections where the growth in the colon has become attached to the stomach, small intestine, uterus, or even the bladder, are reported with a reasonable survival-rate and freedom from recurrence. Adherence to the parietes, particularly the anterior or lateral abdominal wall,



Fig. 522.—CARCINOMA OF THE SIGMOID COLON. THERE IS A DEFINITE INFLAMMATORY ZONE AROUND THE GROWTH.  $\times 100$ .

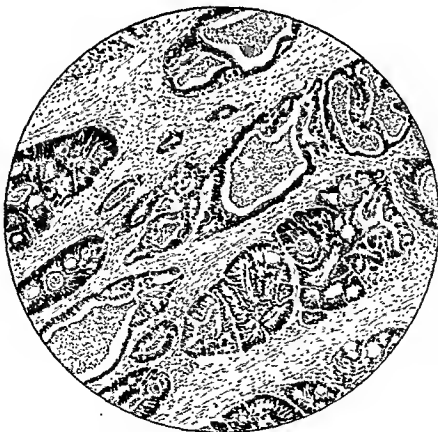


Fig. 521.—MICROSCOPICAL DRAWING OF A CARCINOMA OF THE CECUM.  
A TYPICAL APPENDICEAL CARCINOMA.  $\times 100$ .

is no bar to surgical removal. The only definite contra-indications to removal are the presence of secondaries in the liver, the presence of ascites, or the finding of growth in the pelvic floor, due to trans-cælotomic spread. We may conclude that the results of surgery are encouraging



Fig. 521.—SKILLGRAM OF CARCINOMA OF THE RECTO-SIGMOID JUNCTION AFTER A BARIUM PRIMA. A.P. VIEW. NO GROWTH CAN BE DEMONSTRATED DUE TO THE BALLOONED BOWEL.

and offer greater security than treatment by radium or deep X-ray therapy. Histologically the growths are adeno-carcinomata (figs. 521 and 522).

Carcinoma affects all parts of the colon, but most commonly the sigmoid colon. In a large series reported by Judd, of the Mayo Clinic,

the incidence was as follows: Sigmoid colon 292; cæcum and ascending colon 159; transverse colon 75; descending colon 46; hepatic flexure 29; splenic flexure 24: total 625. The frequency rate in the occurrence of growths of the cæcum varies with different authors, as



Fig. 524.—SCLEROGRAM OF CARCINOMA OF THE RECTO SIGMOID JUNCTION AFTER BARIUM ENEMA. OBLIQUE VIEW SHOWING CARCINOMATOUS STRICTURE. SAME CASE AS FIGURE 523. IT IS VERY IMPORTANT TO TAKE AN OBLIQUE VIEW IN THESE CASES AS AN ORDINARY ANTERO-POSTERIOR VIEW FREQUENTLY DOES NOT REVEAL THE STRICTURE.

the common site for such growths is at the junction of the cæcum and ascending colon, away from the ileo-cæcal valve, and hence in some series they are considered as growths of the ascending colon. An important generalization is that growths of the left side of the colon tend to be obstructive, while the right-sided growths, though often much larger, only rarely lead to obstruction. This is due to two



factors, the nature of the tumour, and the consistency of the intestinal contents. On the left side, the tendency of the growth to encircle the bowel, together with the solid nature of the faeces, leads to obstruction relatively early. On the right side, the carcinoma does not usually encircle the bowel, rather protruding into its lumen, while the liquid intestinal contents can readily pass it. The same two factors account for the fact that anæmic and septic intoxication are more common with the large ulcerating right-sided growths.

As an aphorism we may say that the diagnosis of carcinoma of the

colon is best made by a continual remembrance of its possibility. While there is no single reliable test for the condition, a pre-operative diagnosis can usually be made by a consideration of all the findings. It cannot be too strongly emphasised that in a middle-aged person any alteration in the bowel habit, whether constipation, diarrhoea, or an alternation of the two, would strongly suggest the presence of carcinoma in the colon or rectum. Loss of weight, anorexia, cachexia, jaundice, etc., are rather signs of a speedy fatal termination than of the growth itself.

A barium enema with oblique as well as A.P. views is a valuable aid,



Fig. 525.—SCLAGRAM TAKEN AFTER BARIUM ENEMA SHOWING "FILLING DEFECT" IN THE CECUM. AN ADVANCED CASE OF CARCINOMA OF THE CECUM.

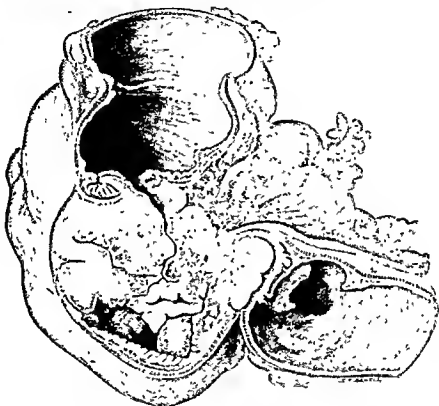
while the actual screen visualisation of the filling of the bowel with the enema is very useful. Sigmoidoscopy should always be performed in a doubtful case.

#### CARCINOMA OF THE CECUM AND ASCENDING COLON

The onset of this condition is insidious, the duration of symptoms before diagnosis being usually about eighteen months. The growth is usually on the outer wall of the cecum and ascending colon, though occasionally a large fungating tumour may fill the cecum below the ileo-caecal valve entrance and thus fail to cause obstruction (see fig. 526). Cases present themselves complaining of one or other of the following symptoms or some combination of them:

- (1) Mild dyspepsia, pain in the right flank, constipation, borborygmi, (often diagnosed as chronic appendicitis).
- (2) A mass in the right iliac fossa.
- (3) Anæmia and debility.

Blood may occur in the stools and there may be attacks of diarrhœa. On further investigation some degree of anæmia and leucocytosis is usually found. A tumour is frequently palpable.



*Fig. 526.—SPECIMEN OF CARCINOMA OF THE CÆCUM REMOVED AT OPERATION.*

*(King's College Hospital Museum.)*

The treatment is straightforward; namely, a two-stage excision of the growth (operative details will be described later). The first operation consists of laparotomy, exploration of the abdomen, confirmation of the diagnosis, and the performance of ileo-transverse colostomy (end-to-side). If there is any suspicion of obstruction we advise an additional ileostomy. The second operation consists of removal of the growth.

# LEFT-SIDED CARCINOMA OF THE COLON, PARTICULARLY SIGMOID GROWTHS

Cases present themselves clinically under the following groups:

(1) *The chronic obstructive groups*, suffering from increasing difficulty in obtaining a bowel movement, exemplified by increasing use of purgatives, borborygmi, and occasional attacks of fermentative diarrhoea.

(2) *The ulcerative groups*, complaining of bleeding, diarrhoea, pain in the left iliac fossa and tenesmus where the growth is low down.

(3) *Acute intestinal obstruction*. These patients on close questioning will usually give a history of chronic obstruction preceding the condition, though of course acute obstruction may be caused by intussusception or volvulus of the growth. This condition is highly dangerous—the patient is already devitalised by a period of chronic obstruction, stercoral ulcers may form readily, and perforation is likely. The condition of sigmoid carcinoma can always be diagnosed by careful examination combined with radiology and sigmoidoscopy. Not infrequently a lump can be felt; this is due less to the growth than to fecal retention above it (fig. 327).

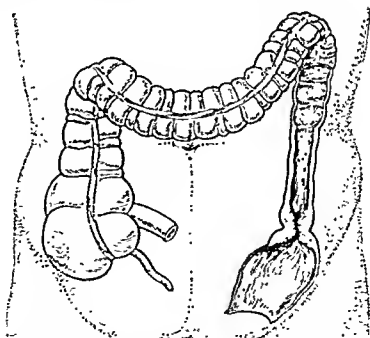


Fig. 327.—Diagram of constricting carcinoma of the sigmoid colon. Note dilatation of the cecum, hypertrophy of the descending colon and dilatation below the growth due to paralysis of the bowel.

*The Treatment of the Condition* varies with the condition of the patient at the time of examination and the findings at the first laparotomy. If *acute intestinal obstruction* is present the following is recommended :

A left lower paramedian incision is made with confirmation of the condition and rapid examination for operability. If the growth is operable, a cæcostomy is performed through a separate right iliac fossa stab wound (made from within outwards) and the abdomen is closed. The patient is then left to recover from the obstruction, the colon being cleansed by enemata before the resection is attempted some weeks later. The details of resection will be described fully later. If the growth is inoperable and low down, then a colostomy above it is preferable to a cæcostomy. If the growth is locally movable but inoperable because of liver or pelvic floor metastases, a Mikulicz-Paul colostomy is advised. Here the growth is lifted outside the abdomen, when it and the protruding colon can then be removed with a diathermy knife and the obstruction relieved. This colostomy can then subsequently be closed and the secondaries in the pelvic floor treated by radium in the pouch of Douglas or by deep X-ray therapy. If there is sufficient normal colon below the growth which is fixed and also inoperable, then cæcostomy should be performed, to be followed later by a short-circuit operation as being more tolerable for the patient. It cannot be over-emphasised that to attempt aseptic anastomosis in the presence of obstruction is to court disaster.

*Operative Treatment in the Absence of Acute Obstruction.* It should be remembered that in all cases of sigmoid growths some mild degree of obstruction is present which means œdema and toxic changes in the bowel wall above the growth. We therefore recommend multiple stage operations. The first operation is necessarily an exploratory laparotomy. The growth is found, its degree of mobility determined, and the best type of short-circuit considered for future performance, e.g. end-to-end junction of pelvic and descending colon (for growths in the middle of the sigmoid), transverso-sigmoid anastomosis (for high sigmoid or descending colon growths), etc. A cæcostomy is performed and the abdomen closed. A month later the abdomen is reopened and the growth extirpated with performance of the anastomosis. As an alternative, at the second operation the end of the bowel may be brought out of the incision for treatment by the Mikulicz-Paul method (see page 966).

Much ingenuity may be called for in cases where the growth has spread to neighbouring viscera. It can be stated that as the tumours spread only slowly no urgency obtains in the matter of resection, and preliminary operations and short-circuits may be performed before the final excision.



*Fig. 224*—SIGMOIDOM SHOWING DIVERTICULOSIS OF TRANSVERSE COLON PROXIMAL TO AN ANASTOMOTIC DIVERTICULITIS OF THE DESCENDING AND SIGMOID COLON CAN BE EASILY SEEN.

OPERATIONS ON THE COLON

The various operative procedures described below must not be taken as representing the only methods of satisfactorily performing the operation described. Most surgeons have their own individual preferences and methods in regard to colon surgery, their methods giving satisfactory results in their own hands. We have not yet arrived at the stage when the various operations can be standardised. Methods of anastomosis, whether side-to-side, end-to-end, or end-to-side, the number of stages taken, etc., are all matters open to discussion. The methods described are those the writer has personally tried and found satisfactory.

Two axioms may be laid down :

(1) Aseptic suture of the bowel must never be attempted in the presence of intestinal obstruction.

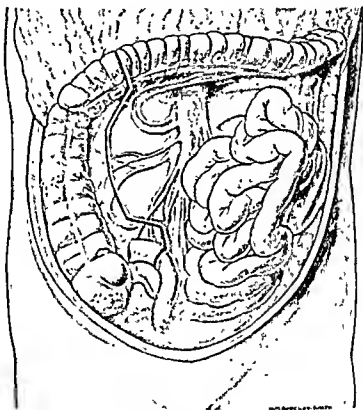
(2) Anastomosis must never be made between loops under tension. Thus it is safer to perform end-to-end anastomosis, than to attempt side-to-side anastomosis where this involves extra pulling on the bowel ends.

A consideration of these two points leads in general either to the performance of two- or three-stage operations, or of extra-peritoneal anastomosis (Mikulicz-Paul), both operations which we can confidently recommend. Naturally all the various operations on the colon cannot be described, but the following are taken as examples of the different techniques :

(1) *Excision of the Cæcum.* This is usually a straightforward procedure. The parts to be removed are : the last six inches of the ileum (as its blood supply is endangered) (see fig. 514) and the cæcum, ascending colon, hepatic flexure, and proximal sixth of the transverse colon, with the attached portion of the omentum (fig. 529).

This operation may be performed in one stage as there is seldom any obstruction, but, if the patient is unfit or the cæcum very fixed, a two-stage operation may well be performed. An ileo-transverse anastomosis is performed at the first operation, the actual resection being carried out at a later stage. The best incision is a free muscle-cutting oblique incision in the right iliac fossa. The incision is retracted well inwards and packs are inserted to keep the small intestine out of the field, only the bowel to be removed being kept in view. Clamps are

applied to the ileum and it is divided. The operator then working from below upwards, and from the outer side of the colon where he incises the peritoneum, proceeds to mobilise the ascending colon with its blood and lymph supply. Care is taken to avoid injury to the ureter and spermatic vessels. The hepatic flexure is freed by blunt dissection and snips with scissors, but care must be taken not to damage the retroperitoneal portion of the duodenum. The transverse colon is now divided between clamps and the portion of the omentum



*Fig. 529.*—LINE OF INCISION OF THE BOWEL AND PERITONEUM IN RESECTION OF THE CÆCUM FOR CARCINOMA.

on the proximal part ligatured ready for removal. The blood-vessels to the cæcum and colon can now be clamped and the bowel cut away portion by portion. The divided ends of the bowel are next invaginated by a series of purse-string sutures and the end of the ileum drawn alongside the transverse colon. A side-to-side anastomosis is then made between them, the sutures being inserted as in a gastro-enterostomy. The suture line is reinforced with the omentum (*fig. 530*).

The final stage is to reperitonise the raw area left by the incision made on the outer aspect of the colon and the section through its

vessels. This is done *secundum artem*. The abdomen is closed with drainage which should never be omitted because of the exudate

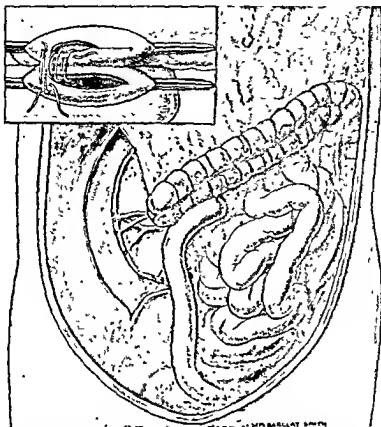


Fig. 530.—COMPLETED OPERATION AFTER EXCISION OF THE CECUM FOR CARCINOMA. THE INSET SHOWS HOW THE LATERAL ANASTOMOSIS IS PERFORMED.

from the raw area of the posterior abdominal wall. The tube may be brought out through a separate stab wound. The same operation is employed for growths in the ascending colon and hepatic flexure.

(2) *Resection of the Sigmoid Colon.* The conditions which call for resection of the left half of the colon are always obstructive in nature and hence, as a routine, a two-stage operation is advised. At the first operation, performed through a left lower paramedian incision, the abdomen is explored and a cæcostomy performed. At the second operation, performed through an oblique muscle-cutting incision, the growth is mobilised, working from the outer side towards the mid-line, and a sufficiency of bowel is freed on either side to allow restoration of continuity (fig. 531). Four clamps are then applied to the colon, the area is suitably packed off, the bowel



divided, and the growth removed together with a wedge of mesentery. The next stage is that of restoration of continuity. Usually an end-to-end junction is made as this is possible without tension (see fig. 332). The suture line is reinforced with omental tags.

The peritoneal covering on the postero-lateral abdominal wall is now restored as completely as possible. The abdomen is closed in layers with drainage.

An alternative method which has much to recommend it, especially

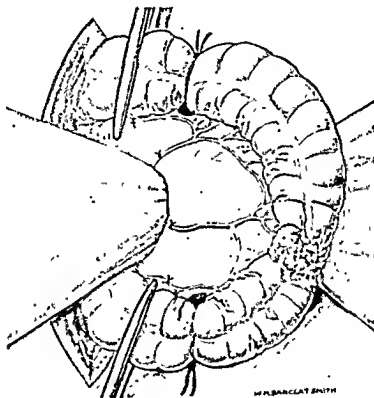


Fig. 331. Drawing showing how the clamps are applied after mobilising a growth in the sigmoid colon.

to those inexperienced at aseptic anastomosis, is the operation associated with the names of Paul and Mikulicz. This type of operation can be performed in the presence of moderate degrees of obstruction. A free incision is made over the tumour, which is then mobilised. The bowel is then divided between clamps and the growth removed. The ends of the divided bowel are lifted out of the wound and their adjacent sides united by suture, the clamps being left *in situ*. The wound is now closed and dressings applied round the clamps. At the end of twenty-four hours the upper clamp is removed and the bowel functions through this colostomy. By the third day the second clamp may be

removed and repair of fistula commenced. On the spur between the two loops of the bowel, an enterotome (figs. 533 and 534) is placed and

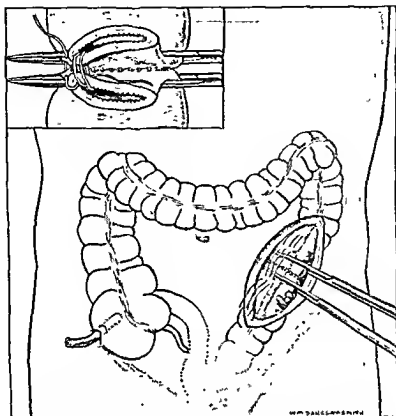


Fig 532.—DRAWING SHOWING THE COMPLETION OF THE OPERATION FOR RESECTION OF THE SIGMOID COLON WITH END-TO-END UNION. A CACOTOMY HAS BEEN PERFORMED SOME DAYS PREVIOUSLY. THE INSET DEMONSTRATES THE METHOD OF SUTURE.

the pressure gradually increased. Sutures hastening the closure may be inserted at the bedside without any anæsthetic. In about four weeks' time, in a favourable case, the fistula will be quite healed and under a local novocaine injection the parietes can be incised and the bowel allowed to drop back into place (fig. 535).

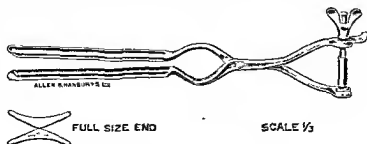


Fig 533.—A USEFUL ENTEROTOME (DEVINE'S).

Devine of Melbourne has perfected a technique for partial colectomy which has given very satisfactory results. The segments of bowel lying adjacent to each other which are to form the anastomosis are sutured

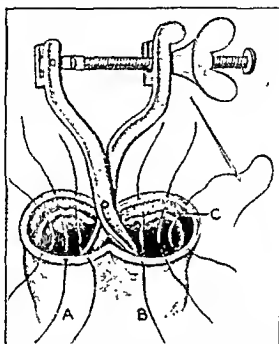


Fig 531.—METHOD OF APPLYING DEVINE'S ENTEROTOME. A—DISTAL SEGMENT. B—PROXIMAL SEGMENT. SUTURES INTRODUCED READY TO BE TIED. C—AREA INSIDE BOWEL DIVIDED OF MUCOUS MEMBRANE.

(After Devine.)

together and clamps applied and the growth removed. In removing the segment containing the growth the sero-muscular layer is turned back as a cuff. The enterotome is applied (fig. 534) and the ends of the bowel



Fig 532.—ENDS OF BOWEL AFTER REMOVAL OF ENTEROTOME.

(After Devine.)

so sutured that there is just room to remove the instrument. When the enterotome is removed the bowel shrinks and soon closes. A small plastic operation will close the defect in the anterior abdominal wall.

(3) *Colostomy*. Colostomy may be required either as a temporary measure, e.g. in diverticulitis with fistula formation in acute obstruction, or as a permanent measure, e.g. in excision of the rectum. While colostomy is a simple procedure, there are various points in its performance which make for great comfort to the patient. The afferent loop of the colostomy should always be as short as possible as this limits the tendency to prolapse. The colostomy opening is usually either made through the upper end of a left paramedian or transrectus incision, or through a grid-iron (muscle-splitting) incision in the left iliac fossa. Both these sites have their respective advantages. For a permanent end colostomy a small stab wound made from within outwards in the left iliac fossa is the best. For a temporary colostomy no glass rod or rubber tube is necessary. The colon is lifted to the incision (it may be necessary to mobilise it by dividing the peritoneum on its outer aspect), and the parietal peritoneum is sutured to the side of the colon. The skin and muscle layers are sutured above and below it. A few days later (twenty-four hours should elapse if possible) the colon may be opened by incising it in its long axis. When the colostomy is to be permanent a spur must be made, and this is best done by passing a glass rod under the exposed loop as well as above and below it. When opened, the bowel is cut across transversely with a cautery.

*After-care of Colostomy Openings*. The best method of care for the skin round a colostomy opening is scrupulous cleanliness. A daily bath is advisable with careful drying of the skin and powdering. Greasy ointments should not be used. The intestinal contents can be deodorised by a daily capsule of kerol (3 minims) before breakfast. The patient will learn for himself what articles of diet are likely to cause accidents with the colostomy (tomatoes, onions, fruit, cream, spinach and beer) and will avoid them.

The old-fashioned colostomy belt should be avoided. It is messy and leads to prolapse. A colostomy, well made and well cared for, works only once a day, and the only covering it needs is a wool pad and a well-fitting abdominal belt. The colostomy should be washed out through its upper opening first thing each morning. It will not act again during the day unless there has been a dietary indiscretion.

(4) *Cæcostomy*. This simple operation is performed through the same incision as that used for appendicectomy. The cæcum is drawn into the wound and a purse-string suture inserted but not tied. An incision is made in the centre of the area enclosed and a tube passed into the cæcum.

The purse-string suture is then tied and invaginated by pushing the tube farther into the bowel when a further purse-string is inserted and tied, thus inverting the first. The incision is then closed in layers.

A cœcostomy needs considerable after-care as the contents of the cæcum are irritating to the skin. The skin should be covered with lanoline to protect it.

SECTION 12

ACUTE INTESTINAL OBSTRUCTION  
AND PERITONITIS

by

A. J. COKKINIS

CHAPTER I

General Pathology—Diagnosis and Treatment

CHAPTER II

Strangulated Hernia

CHAPTER III

Post-Operative and Paralytic Obstruction

CHAPTER IV

Peritonitis

CHAPTER V

Peritoneal Abscesses

## SECTION 12

# ACUTE INTESTINAL OBSTRUCTION AND PERITONITIS

## CHAPTER I

### ACUTE INTESTINAL OBSTRUCTION

#### INTRODUCTION

ACUTE intestinal obstruction, or ileus, is a convenient clinical term which groups together a number of pathological conditions associated with complete stoppage of the intestinal canal. This stoppage is responsible for morbid changes (e.g. proximal distension and distal contraction), and for clinical phenomena (pain, vomiting, coprostasis, and distended abdomen), which are common to all obstructive lesions. But each lesion has also its own distinctive morbid and clinical features ; indeed, a comparison of some types reveals a quite unrelated causation, a widely differing pathology, and a contrasting clinical picture. This lack of uniformity inevitably complicates any discussion on the subject, but our difficulties cannot be solved by divorcing the various lesions from the whole group, or by dealing with them as separate entities. A broad understanding of the comprehensive picture of intestinal obstruction is essential, and the grouping of similar types and the contrasting of dissimilar types must be made within the loose confines of the general idea.

The treatment of acute obstruction is one of the outstanding failures of present-day surgery. While the mortality of appendicitis, perforated ulcers, and other abdominal emergencies has fallen to a small fraction of what it was thirty years ago (Burgess, 1), that of acute obstruction still remains at about 40 per cent. This lack of progress is reflected very strikingly in the statistics given by Souttar (2) in 1925, and by Vick (3) in 1932 (see Table 1).

TABLE 1. MORTALITY OF MAIN TYPES OF ACUTE OBSTRUCTION

<i>Souttar</i> (1920-1924). (Mortality in 3064 cases from 7 London hospitals.)			<i>Pick</i> (1925-1930). (Mortality in 6892 cases from 21 British hospitals.)		
	501	per cent.		70	per cent.
Gall stones . . . . .	43.5	"		42.5	"
Carcinoma . . . . .	31	"		33	"
Adhesions . . . . .	33	"		31.3	"
Internal strangulation . . . . .	51	"		52	"
Volvulus . . . . .	22	"		17.6	"
Intussusception . . . . .	16	"		11.5	"
Strang. Ing. Hernia . . . . .	20	"		16	"
Strang. Fem. Hernia . . . . .	35	"		40	"
Strang. Umb. Hernia . . . . .					

It has become customary to attribute the high mortality of obstruction to *late diagnosis*. This may be true, but the explanation, as Wilkie (4) points out, lies in faulty hospital teaching rather than in the procrastination or ignorance of practitioners. Clinical teachers show a curious partiality for the complete or "classical" picture of disease, and the classical picture of any abdominal emergency is unfortunately the herald of impending death. Timely diagnosis cannot be made by men who leave hospital with the impression that acute obstruction means complete coprostasis, a distended abdomen, and faecal vomiting. Teachers must stress the importance of early symptoms and signs, and endeavour to impart to the student that priceless attribute of "clinical sense" which tells us when serious mischief is brewing, even in the absence of dramatic symptoms.

Fundamental errors in our conception of the essential pathology and morbidity of obstruction are also, in no small measure, responsible for fatal results. Such errors often lead to hurried and meddlesome surgery, intent on mechanical relief of the obstruction, without adequate thought, preparation, or after-treatment. Rushed "exploratory" laparotomies under general anaesthesia are legacies of a "classical" mentality which reaches no further than the operating table.

Of recent years efforts have been made to attack the problems of obstruction from a different angle. These efforts represent the third and culminating stage in the battle against the disease. The first stage, ending with the beginning of this century, was concerned with the morbid anatomy of obstruction, with the elucidation of its various types and causes, the gross pathological changes and mechanisms, and the correlation of these with the symptomatology and diagnosis. Thus was built up the essential scaffolding, without which further progress would be unthinkable. The names of Brinton, Leichtenstern, and



Treves stand out of this period, and their masterly description of the gross pathology of obstruction is the foundation on which present-day knowledge rests.

The second stage covers the first quarter of this century, and represents an attempt to explain the rapid collapse and fatal results of obstruction, which have never been satisfactorily accounted for by the gross morbid changes. The discovery of an abundant bacterial flora in the intestine, and its increase in conditions of stasis, not unnaturally led to the belief that the cause of death was bacterial. With internal strangulations, so often followed by perforation or gangrene, the fatal effect of a complicating bacterial peritonitis has never been in doubt. Also, it was soon appreciated that a similar peritonitis was a common termination of simple obstruction, the bacteria making their way through the damaged intestinal wall, even in the absence of perforation or gangrene. But the collapse of obstruction makes its appearance *long before* peritonitis can occur, and to explain this the "toxæmia" theory was propounded, the absorption of bacterial toxins from the distended gut being blamed for the so-called "toxic" state of the patient. Although this may occur under special conditions, there is a remarkable lack of experimental proof that it is a constant, or even common event. More recently, search has been made for some toxic factor, other than bacterial, the absorption of which might account for the rapid collapse of obstruction. Toxic proteoses and amino-acids have been found in the obstructed intestine, probably produced by a breakdown in the normal mechanism of protein digestion. Recent research suggests that at least one or two of these toxic substances can find their way into the blood or lymph leaving the intestine, when its walls have been grossly damaged by the effects of prolonged distension.

The third stage had its birth in the heyday of the "toxæmia" period, i.e. about twenty years ago, but it is only just beginning to make itself felt. A great deal of experimental work has been done which suggests a re-orientation of thought on the essential morbidity of obstruction, and with this have come new ideas of fundamental importance and new lines of treatment of the greatest possible promise. Very briefly, the present stage consists of a study of the chemical and physical changes in the blood and body fluids, secondary to acute obstruction, but intimately concerned with its morbidity. It has been found that dehydration and loss of essential electrolytes, by the vomiting of digestive juices, deplete the plasma and body fluids, and lead to over-concentration of the blood. The replacement of the lost fluid and salts and the rehabilitation of the blood-plasma have become essential

parts of the treatment of many cases. Furthermore, the lethal effect of distension of the intestine has been proved, and measures of decompression have been devised which occasionally do away with the need for operative intervention, and always minimise the risk when such intervention is necessary. The stimulating property of hypertonic saline, acetyl-choline, and other substances, has been demonstrated quite recently, and the value of spinal and local anæsthesia is now definitely established.

There is at least some hope that when the new ideas and methods have had time to become disseminated and adopted, a real drop may occur in the terrible mortality of acute obstruction.

### ÆTIOLOGY AND CLASSIFICATION

THE old classification of acute obstruction into *mechanical* and *functional* (paralytic) is not without some value, but a more rational one is into (a) *simple obstruction*, and (b) *strangulation*. In simple obstruction we are dealing only with a blocked lumen. In strangulation we are confronted, in addition, with grave vascular changes in an isolated loop or segment of intestine.

#### SIMPLE OBSTRUCTION

Variations in this large class are mainly determined by the anatomical level of the block. For this reason it will be subdivided into (a) *high small-gut*, (b) *low small-gut*, and (c) *large-gut obstruction*.

*High small-gut obstruction* is mostly a temporary blockage, but it is characterised by an enormous loss of fluid and salts, with rapidly fatal dehydration and blood changes. For practical purposes it may be said to include cases of congenital and acquired pyloric or duodenal obstruction, acute dilatation of the stomach, and high jejunal kinks and stenoses following operations on the stomach.

*Low small-gut obstruction* is a much larger sub-class, the outstanding feature of which is rapid and extreme distension of the jejunum and ileum. Loss of fluids and salts also occurs, but not to the same degree as in the previous group. Here are included the bulk of adhesive obstructions (post-inflammatory, post-operative, or post-traumatic), gall-stone ileus, cicatricial stenosis, and most cases of functional ileus.

*Acute large-gut obstruction* is rarely of sudden onset, being mostly the culmination of a long-standing chronic block. There is thus time for the gut to adapt itself to a condition of stasis, and the ileus tends

to run a sub-acute course. The urgent results of dehydration are absent, while the greater size and distensibility of the colon render the dangers of an obstruction less immediate. Moreover, the factor which transforms the chronic block into an acute one is usually of a temporary nature (œdema, spasm, or obturation of the narrowed lumen). Among the common causes are carcinoma (70 per cent), diverticulitis (10 per cent), and faecal impaction (10 per cent); less frequent causes are adhesions, spasm, Hirschsprung's disease, congenital malformations, and fibrous strictures.

#### STRANGULATION OBSTRUCTION

This is almost limited to the small gut, and is mostly encountered in the distal part of the ileum. The chief morbid factor is the fate of the strangulated coil, the blood supply of which is inevitably interfered with. The mesentery affords no protection to the vessels which it carries to the intestine, and the damage is done through compression of the mesentery by the obstructing agent. The morbidity of the lesion is proportional to the degree of vascular interference.

It is convenient to sub-classify strangulation into external and internal.

*External strangulations* include all forms of external strangulated hernia, which of course constitutes the most common single cause of acute obstruction (see page 1034).

*Internal strangulation* is mostly produced by constriction or snaring of a loop of gut and its mesentery by a peritoneal, omental, or visceral band. Less often the cause is an internal hernial aperture, or a hole in the omentum or mesentery. A *volvulus*, or twist, is a definitely rare cause of strangulation. *Intussusception* is also potentially a strangulation, but serious vascular damage is rarely seen; nearly all cases are operated upon while the lesion is still in the stage of a partial simple obstruction. *Mesenteric vascular occlusion* causes a paralytic ileus, but the fatal vascular changes are the result of infarction and not of true strangulation.

The above classification is summarised in Table 2, while the incidence of the various types (based on Vick's statistics) is given in Table 3.

Table 3 clearly shows that there are only five common causes of obstruction, viz. strangulated external hernia, intussusception, internal strangulation, carcinoma, and adhesions. Between them they account for no less than 94 per cent of Vick's 6,892 cases.

parts of the treatment of many cases. Furthermore, the lethal effect of distension of the intestine has been proved, and measures of decompression have been devised which occasionally do away with the need for operative intervention, and always minimise the risk when such intervention is necessary. The stimulating property of hypertonic saline, acetyl-choline, and other substances, has been demonstrated quite recently, and the value of spinal and local anæsthesia is now definitely established.

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to run a sub-acute course. The urgent results of dehydration are absent, while the greater size and distensibility of the colon render the dangers of an obstruction less immediate. Moreover, the factor which transforms the chronic block into an acute one is usually of a temporary nature (œdema, spasm, or obturation of the narrowed lumen). Among the common causes are carcinoma (70 per cent), diverticulitis (10 per cent), and faecal impaction (10 per cent); less frequent causes are adhesions, spasm, Hirschsprung's disease, congenital malformations, and fibrous strictures.

#### STRANGULATION OBSTRUCTION

This is almost limited to the small gut, and is mostly encountered in the distal part of the ileum. The chief morbid factor is the fate of the strangulated coil, the blood supply of which is inevitably interfered with. The mesentery affords no protection to the vessels which it carries to the intestine, and the damage is done through compression of the mesentery by the obstructing agent. The morbidity of the lesion is proportional to the degree of vascular interference.

It is convenient to sub-classify strangulation into external and internal.

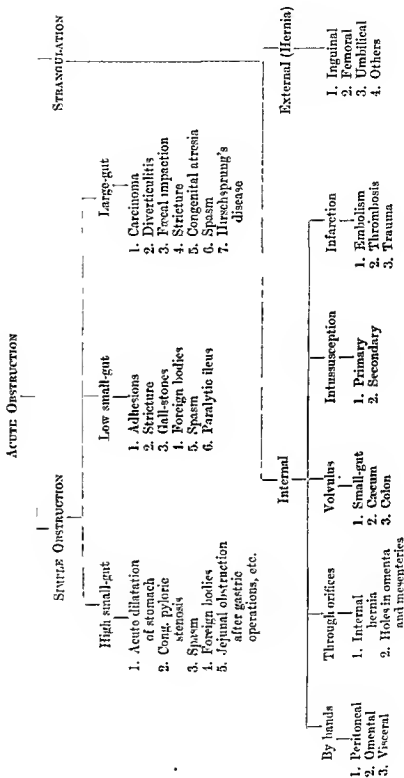
*External strangulations* include all forms of external strangulated hernia, which of course constitutes the most common single cause of acute obstruction (see page 1034).

*Internal strangulation* is mostly produced by constriction or snaring of a loop of gut and its mesentery by a peritoneal, omental, or visceral band. Less often the cause is an internal hernial aperture, or a hole in the omentum or mesentery. A *volvulus*, or twist, is a definitely rare cause of strangulation. *Intussusception* is also potentially a strangulation, but serious vascular damage is rarely seen; nearly all cases are operated upon while the lesion is still in the stage of a partial simple obstruction. *Mesenteric vascular occlusion* causes a paralytic ileus, but the fatal vascular changes are the result of infarction and not of true strangulation.

The above classification is summarised in Table 2, while the incidence of the various types (based on Vick's statistics) is given in Table 3.

Table 3 clearly shows that there are only five common causes of obstruction, viz. strangulated external hernia, intussusception, internal strangulation, carcinoma, and adhesions. Between them they account for no less than 94 per cent of Vick's 6,892 cases.

TABLE 2. CLASSIFICATION OF TYPES AND CAUSES OF ACUTE OBSTRUCTION



# GENERAL PATHOLOGY

Before embarking on a discussion of the several types and causes of obstruction, it is necessary to consider the main anatomical and physiological changes which constitute its general pathology. Almost at once major differences become apparent between the principal groups, e.g. between simple obstruction and strangulation, or between high and low obstruction. It is proper that these differences, as well as the broader similarities of obstructive lesions, should receive consideration here.

The general pathology of acute obstruction may be conveniently divided into its morbid anatomy and morbid physiology.

TABLE 3. INCIDENCE OF VARIOUS TYPES OF ORGANIC OBSTRUCTION (based on Vick's 6,892 cases).

## Simple Obstructions.

	No. of Cases.	Per-centage of Total.
1. Adhesions . . .	505	7.35
2. Intussusception . .	1034	15
3. Carcinoma . . .	895	13
4. Stricture . . .	36	.5
5. Gall-stones . . .	47	.7
6. Other Obturations . .	24	.35
7. Congenital Atresias, etc. . . . .	40	.6
8. External Compression . .	29	.4
Total . . .	2610	37.9

## Strangulations.

	No. of Cases	Per-centage of Total
1. Str. Ing. Hernia . .	1378	20
2. Str. Fem. Hernia . .	1318	19.5
3. Str. Umb. Hernia . .	371	5.4
4. Other Hernias. . .	170	2.5
5. Internal Strangulation . . .	790	11.4
6. Volvulus . . .	176	2.6
7. Embolism and Thrombosis. . .	49	.7
Total . . .	4282	62.1

# MORBID ANATOMY

1. *Changes in the Intestine.* These differ very materially with the presence or absence of strangulation.

(a) *Simple Obstruction.* Here the outstanding morbid change is *distension of the intestine above the block* (fig. 536). This distension is progressive, and its degree and rate of increase vary with the level, duration, and cause of the obstruction. In general, it is most marked in low small-gut and in large-gut stoppage; in the latter, the cæcum may be blown up to the size of a small toy balloon.

The wall of the distended gut is stretched to a paper-like thinness and becomes very friable, the least violence sufficing to tear it. At

first pale, the distending intestine soon becomes congested, and finally cyanotic, from compression of its veins by the tension of the accumulated fluid and gas. Edema and hæmorrhagic areas develop in the mucosa, while in late stages patches of actual gangrene ("distension necrosis") appear at the anti-mesenteric border of the last two or three feet of the distended intestine. Perforation of these patches is a likely termination in neglected cases, but peritonitis can occur without visible rupture or perforation, the organisms passing through the thinned-out and devitalised wall in its necrotic areas.

The intestine *below the block* is emptied by a strong peristaltic wave at the onset of the obstruction, and is pale, firm, and contracted. The abrupt transition from the enormously distended and cyanosed proximal

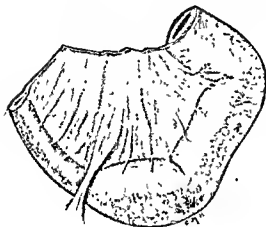


Fig. 536.—SIMPLE OBSTRUCTION BY A BAND.

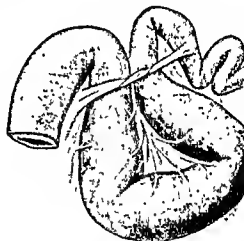


Fig. 537.—STRANGULATION BY A BAND

gut to the contracted and pale distal gut is a striking phenomenon in all mechanical obstructions, and serves as an unerring guide to the actual obstructing lesion. In paralytic ileus the transition is much less abrupt.

(b) *Strangulation.* The intestine above and below the strangulated segment shows the same changes as in a simple obstruction, but owing to the shorter duration of life the proximal distension tends to be less marked.

The vital changes are those which involve the strangulated segment itself (fig. 537). They are seen typically in an external strangulated hernia, and will be dealt with more fully in the article devoted to this condition. Briefly, it should be noted that the isolated coil becomes very distended and tense. The distension is extremely rapid owing to complete inability of the veins to absorb the carbon dioxide and other gases given off in the decomposition of the contents.



The strangulated coil is congested and cyanosed, and rapidly passes through the stage of hæmorrhagic infarction to non-viability and, finally, to gangrene. The hæmorrhagic infiltration of the gut-wall is associated with bleeding into the lumen, and also into the peritoneum.

These vascular changes are more rapidly fatal in internal strangulation than in external herniæ. In the former the strangulated loop is in the peritoneal cavity, and peritonitis, with or without perforation, is likely to terminate life very quickly.

Before leaving this section, attention may be drawn to an important if uncommon anomaly, i.e. the occurrence of strangulation of the intestine without complete obstruction of its lumen. This is the case in *Richter's hernia* (see fig. 538), where a portion only of the circumference of the gut passes through a hernial orifice and is strangulated by its edges. The constriction is intense and gangrene or perforation occur very early. Since this may happen without obstructive symptoms the danger of a fatal issue is very real.

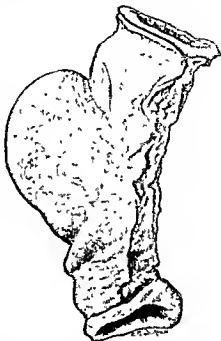


Fig. 538.—RICHTER'S HERNIA.  
(From St Mary's Hospital Museum.)

2. *Changes outside the Intestine.* A fluid exudate accumulates in the peritoneal cavity. In simple obstruction the exudate is serous, but in internal strangulation it soon becomes blood-stained and abundant. In the terminal stages the purulent exudate of *peritonitis* makes its appearance.

The lungs, in fatal cases, show *broncho-pneumonia* and *acid digestion*, caused by inhalation of vomited matter. Pulmonary abscess or gangrene are occasionally seen.

The tissues generally show the changes of *dehydration*. *Toxæmia* is not prominent in simple obstructions, but some cases of strangulation show changes which suggest that the action of toxius is in part responsible for the fatal result.

#### MORBID PHYSIOLOGY

The early collapse and fatal outcome of acute obstruction cannot be accounted for on anatomical grounds, nor has the "bacterial toxæmia"

theory provided an acceptable explanation. It was only when the physiology of the disease began to be investigated that the factors responsible for its morbidity became apparent.

The first conclusion we reach from a study of this work is that the physiological changes of obstruction are many and variable, and that no single factor can be picked out as affording a constant explanation of its mortality. The next conclusion is that the level and type of the obstruction exert a determining influence on the operation of physiological factors. In other words, the morbid physiology varies with the anatomical level and type of obstruction. This must not be taken to mean that there is no physiological overlap between various levels and types; such an overlap, of course, exists, but it does not detract from the broad truth of this conclusion.

On an anatomical basis, the physiological changes may be classified into those which occur (i) in simple obstruction, and (ii) in strangulation; the former being sub-classified into high small-gut, low small-gut, and large-gut.

#### MORBID PHYSIOLOGY OF SIMPLE OBSTRUCTION

(a) *Morbid physiology of high small-gut obstruction.* The clinical picture of profuse vomiting, severe shock, and early and fatal collapse, at once suggests a physiological disturbance of the utmost gravity. Modern research has shown that three fundamental changes are responsible for this disturbance; they are: (i) dehydration of the blood and tissues; (ii) profound loss of inorganic electrolytes from the plasma, and other chemical and physical blood changes; (iii) failure of renal function.

(i) *Dehydration.* This is the most obvious of the physiological changes, and its effect on the blood and tissues is reflected by the intense thirst, dry skin, sunken eyes, hoarse voice, and oliguria which so constantly occur in high obstructions. The *extreme loss of water* from the body can be attributed to two factors: (a) profuse vomiting, and (b) complete failure of intestinal absorption owing to the high level of the block.

The deadly effect of dehydration was proved conclusively by Hartwell and Hognet (5) (1912), who prolonged life in dogs with a high obstruction for several weeks, by introducing large amounts of saline subcutaneously. Control animals, not so treated, died in a few days. Similarly, Armour (6), of Edinburgh, kept dogs with a high obstruction

alive for five weeks, by introducing fluids into the intestine *below* the experimental block, thus proving the lethal effect of the loss of absorption.

(ii) *Blood changes.* At first it was thought that the beneficial action of saline treatment lay in the maintenance of the *water content* of the blood and tissues, and for a time the "dehydration" theory held the field. Very soon, however, it was realised that other substances besides water are lost to the body. In 1923, Haden and Orr (7) demonstrated conclusively a *fall in the blood chlorides* in experimental high obstruction. Five years later, Gamble and McIver (8) showed a loss of other ions besides *chlorine*, namely *sodium* and *bicarbonate*, and proved beyond doubt that this fall of essential plasma salts can only be explained by the *loss of the digestive secretions* of the stomach, pancreas, liver and intestine. The same workers also showed that the fixed base (mainly sodium) and the ions (chlorine, bicarbonate, etc.) in the combined digestive juices exist in proportions closely approximating to the ionic content of the plasma (see fig. 539).

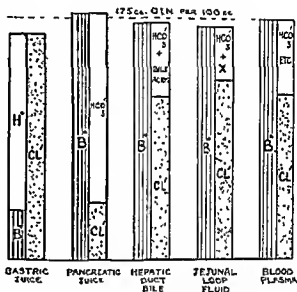


Fig. 539.—COMPOSITION OF DIGESTIVE JUICES COMPARED WITH BLOOD PLASMA. (after McIver and Gamble)

Normally, some five litres of digestive secretions are poured daily into the stomach and upper intestine, i.e. about twice the total volume of the blood-plasma, which is, of course, their source. It is easy to see how essential to life is the re-absorption of these juices by the lower intestine. Their loss to the body, by vomiting and absence of absorption, practically represents so much plasma depleted; this loss, and the associated dehydration, unquestionably provide the true explanation of the morhidity of high obstructions.

With the depletion of its plasma, the blood shows the physical changes of *anhydraemia*, i.e. it becomes concentrated and *its viscosity is increased*; there is also a rise in the red count. The total volume of the plasma may actually shrink by one-third, but even this big loss represents only a small fraction of the total withdrawal of fluid and

salts from the body. In the later stages of obstruction a mild *alkalosis* manifests itself (see below); also the *plasma protein* rises from the normal 7 per cent to as much as 10 or 11 per cent, and there is a definite *increase of the blood urea* and a rise in the total *non-protein nitrogen* of the blood (see below). This *anhydraemia* is responsible for progressive interference with the respiratory and renal exchanges of the blood.

It is of more than passing interest to observe that the physiological changes of high obstruction approximate closely to those which occur in cholera or dysentery, and in gastric, duodenal or pancreatic fistulae. In high obstruction, duodenal fistula, and the diarrhoeas, the digestive juices are lost in equal proportions and, therefore, the ionic contents of the plasma are depleted in roughly equivalent quantities. Thus, any gross disturbance of the acid-alkali balance of the blood is avoided, although actually there is a slightly greater loss of chlorides than bicarbonate, and so a consequent tendency to a moderate but increasing *alkalosis*. With a gastric fistula there is a great depletion of chlorine ions and thus a relative heaping-up of bicarbonate, with marked *alkalosis*; the same change occurs in pyloric obstruction, in which the *alkalosis* may reach a degree sufficient to produce tetany. On the other hand a pancreatic fistula leads to a great loss of bicarbonate ions, and so to a relative increase of chloride, with marked *acidosis*. These chemical changes will be better understood by reference to figure 539.

(iii) *Renal failure*. The association of renal failure with intestinal obstruction was known to the workers of the last century, but it was attributed mainly to reflex causes. It is only in the last twenty years that this association has been fully investigated. The outstanding discovery is a *great rise in the blood urea* and in the *non-protein nitrogen* of the blood. There is also a diminished output of urine and a *fall* in the excretion of *urea* and *salts*.

For a time this failure of kidney function was attributed to the action of toxins on the renal epithelium, but a far more likely explanation is provided by the physical changes in the concentrated blood. In consequence of the *anhydraemia* and increased protein content, the blood becomes too viscous to pass through the renal capillaries at a normal rate; alternatively, the renal epithelium fails to separate a normal urine from the anhydrous blood. Oliguria results, and the case may progress to complete anuria, with *uraemic* manifestations.

(b) *Morbid physiology of low small-gut obstruction*. It was at first hoped that the promising results of research on high obstruction would

also find an application in the more common obstructions lower down. Unfortunately, these expectations have been only partly realised. Dehydration and the blood changes consequent upon the loss of digestive juices occur with diminishing force as the level of the block descends. They are still important factors of morbidity in jejunal and high ileal blockage, but by the time the lower end of the ileum is reached they become relatively insignificant. Hartmann and Elman (9), investigating the blood chemistry of ileo-cæcal obstruction, found that only slight disturbances occur, certainly not sufficient to cause death, and that the administration of parenteral solutions does not delay appreciably the fatal issue.

The most striking difference between high and low obstruction, however, appears in their clinical course. In the former this is rapidly and progressively downwards, corresponding with the progressive dehydration and blood changes; whereas in low obstruction the patient's condition remains more or less stationary for a number of days, and then suddenly shock and collapse appear with fatal results in a few hours (Elman, 10). This sudden and unexpected termination is particularly distressing when it occurs, as not infrequently it does, after a successful operation.

Recent research suggests that two physiological changes, with associated disturbances, are mainly concerned in the morbidity of low small-gut obstruction, namely (i) distension, and (ii) sudden decompression of the distended gut.

(i) *Proximal distension.* This is the outstanding pathological change in all low obstructions and is produced by an accumulation and local production of fluids and gases.

The *fluids* are mainly brought down from above by forced waves of peristalsis, attempting to make a way through but failing to do so. In addition to swallowed fluids, the secretions of the stomach, liver, and pancreas are poured in. There is also an increased secretion of intestinal juice and mucus on the part of the obstructed and congested intestine. Furthermore, the tension of the accumulated fluids and gases compresses the capillaries of the intestinal wall, and thus hinders the re-absorption of these substances. A vicious circle is set up and the distension becomes extreme, involving greater and greater lengths of the proximal intestine.

The *gases* in the obstructed gut include carbon dioxide, nitrogen, oxygen, methane, and hydrogen sulphide; of these carbon dioxide is usually present in greater volume than the others. The amount of gas

varies: extreme gaseous distension is more often seen in the colon than in the small gut, while the presence of partially digested food greatly increases the volume of gas formed by bacterial action. Finally, vascular factors play an important part in this connection, as will be explained below.

There are four possible sources of intestinal gas. The first is *swallowed air*, which is especially likely to accumulate in post-operative and peritonitic ileus. The second source is *bacterial decomposition* of the contents of obstructed intestine; this is the usual origin of meteorism of the colon. Thirdly, gases (especially  $\text{CO}_2$ ) diffuse into the intestine *from the blood* (McIver, 11). The fourth and most important cause of gas-distension is *failure of the normal method of elimination* of intestinal gas; ordinarily gas is partly expelled as flatus, and partly absorbed into the blood and eliminated by the lungs. In obstruction both mechanisms fail, the first for obvious reasons, and the second because the circulation in the gut-wall is interfered with by the distension.

The part which *distension* plays in the *causation of death* is obscure, but its importance cannot be doubted. The contents of obstructed coils are, of course, highly toxic, consisting of trypsin, peptones, proteoses, amino-acids, bacterial toxins, and other poisons. It is extremely doubtful, however, if they are absorbed in lethal amounts while the gut-wall remains intact and alive. What evidence we have is certainly against the likelihood of a bacterial toxæmia, unless of course peritonitis is present. On the other hand, Elman (10) and others recently found an increase of guanidine and other highly toxic amino-acids in the blood of experimental obstructions, but much work remains to be done before we can decide what bearing this has on the cause of death.

In an article just published, Holt (12) brings forward convincing evidence of the part played by "distension necrosis" in the morbidity of simple obstruction. The patches of devitalised mucosa at the anti-mesenteric border of the distended gut come into direct contact with its highly toxic contents, and are unable to prevent the absorption of toxic substances (probably proteoses or amino-acids) which may contribute to the fatal result. By a series of experiments on dogs, Holt shows that the toxin of *B. Welchii* is *not* absorbed from the intestine, and also that the administration of *anti-Welchii serum* does not influence to any degree the course and result of simple obstructions. It seems probable, however, that the enormous increase of *B. Welchii* in obstructed intestine (Williams, 13) does play some part in the pathology of obstruction; the role which may be assigned to it is

that of increasing the toxic proteoses and amino-acids in the distended intestine, in virtue of its proteolytic properties.

One possibility which seems to have been overlooked is that the morbid effect of increasing distension may be *nervous*. It is well known that trauma to the mesentery causes shock. After a certain degree of distension is reached it is possible that shock begins to be produced by stretching of the peritoneum, or by actual tension on the mesentery. Once initiated, this shock would quickly increase with further distension and, I submit, at least provides a reasonable explanation for the rapid collapse which often occurs.

(ii) *Sudden decompression*. The lethal effect of sudden release of an obstruction has been recognised for many years. Death within a few hours may follow rapid emptying of the distended gut by operation, or by rupture into the peritoneal cavity. It has been usual to ascribe these rapid deaths to an overwhelming toxæmia, the toxins being absorbed by the healthy intestine beyond the obstruction, or by the peritoneum. That this is not the sole explanation is proved by the occurrence of equally "sudden" deaths after evacuation of the distended coils outside the body. It has been suggested by Elman (10) that the release of tension opens up the compressed capillaries and veins in the gut-wall, which, through lack of tone, now become engorged, and that sufficient blood may be lost to the general circulation in this manner to prove fatal to the already enfeebled patient. To these views I would again add the suggestion that this sudden decompression may operate through the nervous system, death being caused, in part at least, by mesenteric shock.

Whatever explanation is favoured, there can be no doubt that sudden release of distension is a real danger to the patient, and that a gradual decompression of the intestine should always be aimed at.

(c) *Morbid physiology of large-gut obstruction*. Acute obstruction of the large intestine in more than 90 per cent of cases occurs as a gradual culmination of a chronic obstructive lesion. The acute stage is never as urgent as in small-gut cases, partly because the colon has had time to adapt itself to the stasis, and also because the block is of a temporary nature and rarely complete. Dehydration and blood changes are never prominent, and although extreme distension may occur, the colon appears to tolerate it far better than does the ileum. The danger of sudden decompression is also present in a lesser degree.

In spite of these favourable conditions the mortality of major operations for acute colonic obstruction is extremely high. The

explanation lies in a lowering of the patient's resistance by the previous debilitating illness. If this be kept in mind, and the more radical procedures avoided, there is a fair prospect of tiding the patient over the acute attack, and thus of converting the case back to a chronic obstruction.

#### MORBID PHYSIOLOGY OF STRANGULATION

Strangulation of the intestine is associated with more urgent changes than those discussed under simple obstruction. In fact, so lethal is its effect that other considerations, such as the level of the block or the degree of proximal distension, become relatively unimportant. The root of the matter is the speed with which strangulation causes death. Although the physiological changes of dehydration, blood-depletion, renal disturbances, distension, etc., may all play a part in the fatal issue, there is rarely sufficient time available for their contribution to be really effective.

The principal morbid factor in internal strangulation (particularly when a short coil is involved) is *necrosis* of the isolated segment, *peritonitis*, with or without *perforation*, being the immediate cause of death. Were this an invariable sequence the morbidity of strangulation would present no problem for investigation. But many cases of strangulation die before gangrene or peritonitis have had time to appear, while others do so after removal of the devascularised segment.

A closer examination of the morbid physiology of strangulation demonstrates that an outstanding feature, both in experimental animals and in actual patients, is *shock*; this is shown by unmistakable signs of impaired circulation and depressed metabolism. It is not toxic shock, for it appears suddenly at the very onset of the strangulation. In the first place it must be *traumatic*, and the obvious explanation is injury of the strangulated *mesentery*: the larger the amount of gut and mesentery constricted, the greater the shock. After this *initial shock* the circulation gradually recovers, mostly by a peripheral vasoconstriction, and the blood-pressure may remain at a normal level for some hours. This improvement is a snare, for the shock is only *latent* and liable to return at any moment. Zachary Cope (14) refers to a valuable sign by which the presence of latent shock can be established, i.e. instability of the blood-pressure; according to him, the pulse-pressure gives a better indication of the condition of the patient, and of his fitness to withstand a major operation.

The nervous origin of initial shock cannot be doubted, but the factors responsible for the *terminal shock* and fatal collapse (in the



absence of peritonitis) are less clear. Wangensteen (15) and others have shown that a dog with a ligature tied round a loop of gut, tightly enough to compress the veins only, dies in 6-12 hours from bleeding. Arguing from these experiments, they maintain that in actual cases of strangulation the *loss of blood* caused by extravasation into the intestine and peritoneum may be sufficient to determine a fatal exacerbatation of the latent shock; this is almost certainly true when a long loop of bowel (two feet or more) is strangulated (Foster and Hausler, 16).

*Sudden decompression* of the strangulated coil, or of the distended gut above it, appears to be the immediate cause of death in some cases. We have already shown that this may be explained by engorgement with blood of the hitherto collapsed and atonic capillaries and veins in the intestinal wall, or by shock through stimulation of the mesenteric nerves by the sudden release of tension.

In the absence of peritonitis, it is unlikely that the *absorption of bacterial toxins* plays more than a subsidiary part in the causation of death. But it has been shown (16) that when segments of moderate length (one to two feet) are strangulated, the fluid exuding from the devitalised loop after about sixteen hours becomes highly toxic (non-bacterial), and that absorption of this fluid by the peritoneum may be a direct cause of death.

## DIAGNOSIS

The fate of a patient with acute intestinal obstruction depends more on early and accurate diagnosis than on anything else. The importance of the *time factor* has already been stressed; every anatomical and physiological change associated with obstruction is cumulative, and the prolonged operation of these changes diminishes the chance of recovery in something like a geometrical progression.

*Accuracy* of diagnosis is equally important. The knowledge that a patient has an obstruction is not sufficient in itself. Individual types and lesions differ so widely in their course, morbidity and treatment, that the patient's life frequently depends on recognition of the exact kind of obstruction he is suffering from.

Accurate clinical diagnosis involves three distinct steps:

- (1) The discovery of the *presence* of acute obstruction.
- (2) The recognition of the *type* (simple or strangulated) and *level* of the obstruction.
- (3) The diagnosis of the *cause* of the obstruction.

## I. THE DISCOVERY OF ACUTE OBSTRUCTION

The "classical" picture of an advanced obstruction is unmistakable. There may or may not be a history of sudden onset with pain, shock, and initial vomiting (the peritonism "triad"). The patient is suffering from *gripping pains*, which come and go, and which are obviously "colicky" in nature. The *vomiting* returns and becomes persistent and "regurgitant"; at first the vomitus is gastric, but soon

becomes bilious, and lastly the foul-smelling brownish contents of the small intestine are gulped up ("faecal" vomiting). Usually there is *absolute constipation*, the patient having passed neither faeces nor flatus for some hours or days. The abdomen becomes *distended* and tympanitic, and may enlarge to an enormous size. Finally, the symptoms of terminal *collapse* manifest themselves.

This complete picture is only seen at a late and usually hopeless stage. Unfortunately the recognition of obstruction in its early and curable stages is more difficult. The fundamental symptoms and signs (pain, vomiting, coprostasis and distension) are actually the same, but they are far less obvious; if we are to recognise

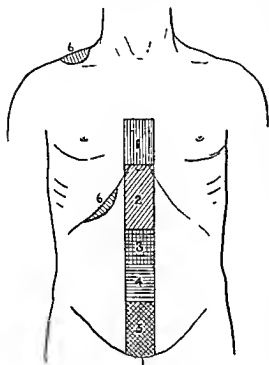


Fig. 540.—SURFACE AREAS OF REFERRED PAIN. (Modified from Mackenzie.)

1. ESOPHAGUS.
2. STOMACH AND DUODENUM.
3. JEJUNUM.
4. ILEUM.
5. COLON.
6. GALL-BLADDER AND DIAPHRAGM.

them in their incipient stage, we must first understand the physiological mechanisms responsible for them, and then learn how to look for and properly assess them.

*The Symptoms and their Mechanism.* (a) *Pain.* According to Mackenzie all pain of visceral origin is referred: i.e. the spinal segment which supplies the diseased viscus becomes "irritable," and normal sensory stimuli are interpreted as pain and referred to that part of the abdominal wall which is supplied by the same segment (fig. 540).

Although this mechanism explains inflammatory pain it does not account for all the painful sensations of obstruction. The intermittent colic associated with forced peristalsis is almost certainly a true intestinal pain, produced by internal pressure. Similarly, the dull and wearing ache of paralytic ileus, and the continuous pain of mechanical obstruction between the attacks of colic, are probably caused by increase of the tension on the muscle-fibres of the distended intestine (Ryle, 17). Further, sensory nerves run in the areolar tissue outside the peritoneum and within the mesenteries, and there is little doubt that pain can be produced by traction, pressure or inflammation of these structures. The final pain is, of course, caused by peritonitis, but at this late stage the patient's sensibility is mercifully very low.

A knowledge of these mechanisms enables us to explain, with some accuracy, the painful sensations of the patient, and also to correlate them with the various stages of the disease.

The more urgent cases have an acute onset, with sudden agonising pain and shock, which can only be attributed to injury to the mesentery. Then comes the dull and wearing ache of intestinal distension, soon to be submerged in the gripping "colicky" pains of forced peristalsis, which recur with a dreaded rhythm until the obstruction is relieved or the intestine becomes paralysed. These *gripping pains* are of the greatest possible diagnostic importance, since they appear quite early and constitute the outstanding symptom of all mechanical obstructions; they are not relieved by measures which cure or alleviate simple intestinal colic.

At a later stage, the paroxysmal pain diminishes and the continuous ache increases, indicating a change-over from the period of intense peristaltic activity to that of extreme distension and commencing paralysis.

(b) *Vomiting*. The initial vomiting of urgent obstructions is reflex, and is produced by stimulation of the vagal nucleus. The *persistent regurgitant vomiting* which soon follows is one of the most constant symptoms of obstruction. The earliest explanation of the mechanism by which intestinal contents reach the stomach was that of "reverse peristalsis." In the last century this view was replaced by the theory of the "reversed central stream," according to which some of the contents of the distended intestine are squirted back towards the stomach by forced peristalsis against an obstacle, this reverse movement taking place in the central part of the column of fluid.

Neither peristalsis nor anti-peristalsis account for the equally

persistent vomiting of paralytic ileus and of the last stage of mechanical obstruction. The usual explanation that such vomiting is "toxic" is valid only when peritonitis is present. In other cases the act of vomiting is itself probably sufficient to bring the intestinal contents to the stomach: in this act the diaphragm and abdominal muscles compress the column of fluid in the distended intestine, forcing it upwards.

*Persistent vomiting* is a symptom of the utmost diagnostic value, particularly when it fails to relieve pain. True vomiting of feces never occurs in obstruction, but the regurgitation of the foul contents of distended small gut, which passes under the name of "fecal vomiting," should be dreaded as a herald of death, rather than regarded as an aid to diagnosis.

There are three conditions in which persistent vomiting may be absent: (i) previous starvation (e.g. in early post-operative ileus); (ii) low large-gut obstruction (e.g. carcinoma or volvulus of the sigmoid); (iii) partial obstruction (e.g. intussusception and Richter's hernia).

(c) *Coprostasis*. Strange though it may seem, constipation is the least reliable of the symptoms of obstruction. A patient with an urgent strangulation may have had a free action of the bowels a few hours previously; another may be constipated for more than a week and yet suffer from no obstruction whatever. Further fallacies are: (i) the stimulus of a sudden stoppage may empty the intestine distal to it; (ii) an enema may wash out colonic contents days after the onset of obstruction; (iii) in partial blockage of the lumen (e.g. Richter's hernia and intussusception) there may be no cessation of normal bowel action.

Diagnosis of acute obstruction should be made within a few hours of its onset: therefore we cannot wait for absolute constipation to become established. Nevertheless, we can always discover whether or not the patient has passed *flatus* since the onset of his illness. Peristaltic pain, in the absence of obstruction, is always accompanied by the passage of flatus: therefore, a patient with colicky pains who is unable to pass wind is almost certainly obstructed.

Nothing establishes the presence of a complete intestinal block with more certainty than the administration of *two diagnostic enemas*, at an hour's interval. Feces and a little flatus may be evacuated with the first enema, but the second is either quite clear or contains only one or two small scybala, and—this is crucial—no flatus at all is passed with it. The value of this test was recently emphasised by Newland (18), while

Yates (19) rightly insists that the doctor or a reliable nurse should be with the patient, to make certain if flatus is passed or not.

*The Examination and Physical Signs.* A careful examination of the abdomen should never be omitted. *Inspection* in an advanced case usually reveals *distension of the abdomen*, but this is a sign which *must not be waited for*. In the early stage of obstruction the belly is *flat* and *flaccid*, and it is in this stage that diagnosis must be made. Considerable distension of the intestine may be present without obvious swelling of the abdomen. It is only in large-gut and paralytic ileus that abdominal distension is of real diagnostic value.

An earlier and more significant sign is *distension of individual coils*. In thin people a distended coil may sometimes be seen through the abdominal wall, particularly when it is rendered prominent by peristalsis. This phenomenon of *visible peristalsis*, when accompanied by colicky pain, is practically pathognomonic; unfortunately it is not often seen, except in thin people, or in acute obstruction supervening on chronic, when it is exaggerated by the hypertrophied musculature.

*Palpation* may reveal localised distension or peristalsis when these do not appear on inspection. A distended and gurgling *cæcum* can be palpated in most large-gut obstructions, and occasionally a loop of small gut may be felt enlarged or contracting in obstructions of the ileum. The discovery of a *mass* or *tumour* is of value in the search for a cause rather than in the diagnosis of obstruction. A *rectal examination* must never be omitted; apart from the discovery of local growths and strictures, this enables us to feel the pelvic viscera and perhaps to find distended loops or tumours of the lower ileum and pelvic colon.

Palpation is also of value through the negative evidence it may afford. The *absence of abdominal tenderness and rigidity* is an important confirmatory sign of obstruction, since it at once excludes the inflammatory emergencies.

*Abdominal auscultation.* Auscultation through a stethoscope should be regarded as an essential step in the examination of any acute abdomen; it is indispensable in the early diagnosis of obstruction. The student should first familiarise himself with the distribution, tone, and intensity of the normal sounds produced by the intestine; after this, it is not difficult to detect abnormalities of intestinal function. Almost from the onset of a mechanical obstruction the intestinal sounds are *louder and more frequent than normal*. Peristaltic rushes can be heard, increasing in intensity as the site of the obstruction is reached,

where an actual explosion may appear to occur. We are thus sometimes able not only to diagnose an obstruction, but also to locate its site. Finally, nothing can be more striking than the contrast between the turbulence of a mechanical obstruction and the dead silence of peritonitis or paralytic ileus.

*X-rays in diagnosis.* Of recent years radiography has been used in the diagnosis of acute obstruction, particularly in America. It may show gas in the small intestine (normally only present in infants), or a distended cæcum and colon ending abruptly at the site of obstruction. In the erect position gas may be seen over fluid levels. Without denying its usefulness in special instances, we cannot see any real justification for its general adoption. It is of little assistance in early cases, where help is most needed, and it is bound to waste time and to add to the patient's exhaustion.

## II. RECOGNITION OF THE TYPE AND LEVEL OF OBSTRUCTION

(a) *The Type.* Having diagnosed the presence of an acute obstruction it is essential that we proceed farther. Our first and most onerous duty is to *distinguish between simple obstruction and strangulation*; fortunately, this distinction can be made in most cases.

*Strangulation* is nearly always of sudden onset, with agonising pain and severe shock. The pulse becomes rapid and there may be some fever. The diagnosis of *external strangulation* is made easy by the presence of a tense, tender, and irreducible swelling over a hernial orifice, with no impulse on coughing. *Internal strangulations* are more difficult to recognise, but the tense strangulated loop may be felt, and there is usually some abdominal tenderness and rigidity owing to the presence of blood in the peritoneum. Moreover, recovery from the initial shock is never complete, the blood-pressure remains unstable, while the patient is obviously ill and becomes *rapidly* worse.

*Simple obstruction*, on the other hand, is more gradual in onset and there may be no evidence of shock for several days. The pulse is slow and the blood-pressure is not affected till the later stages. The temperature is normal. There is no abdominal tenderness or rigidity. The course is relatively slow, and for some days the patient may not appear to be seriously ill.

The clinical differentiation between *mechanical* and *paralytic ileus* is of obvious importance, and will be considered at length elsewhere. The main distinguishing features of paralytic obstruction may,

however, he briefly summarised here. They are: (i) presence of an adequate cause, e.g. previous operation or peritonitis; (ii) complete absence of paroxysmal "colicky" pains; (iii) rapid tympanitic distension; (iv) absolute silence of the abdomen on auscultation; (v) reaction to spinal anaesthesia, also to hypertonic saline, acetyl-choline, and other substances.

(h) *The Level.* Determination of the level of obstruction is the next step in diagnosis, and it is particularly necessary in cases of simple mechanical ileus.

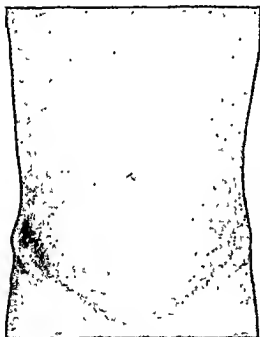


Fig. 541.—THE ABDOMEN IN LOW SMALL-GUT OBSTRUCTION.



Fig. 542.—THE ABDOMEN IN LOW LARGE-GUT OBSTRUCTION.

*High small-gut obstruction* is characterised by little pain, *very profuse and frequent vomiting*, and rapid dehydration. Diminution of urinary output, a rising blood urea, *intense thirst*, and early collapse are prominent features. Distension is limited to the epigastrium, and there is little or no evidence of increased peristalsis. Faeces and flatus may be passed with or without an enema.

*Low small-gut obstruction* has a less urgent course. Its main features are severe and frequent *colicky pains*, persistent but less profuse vomiting, absolute constipation, and very obvious *peristalsis* on auscultation. At first, one or more dilated coils may be seen or felt; later the *distension* spreads, but even in advanced cases it limits itself to the central part of the abdomen (fig. 541). In thin patients, and with

preceding chronic obstruction, the well-known "ladder" or "organ-pipe" pattern of distended coils may be seen. Dehydration, and the blood and renal changes associated with it, do not appear until late.

*Large-gut acute obstruction* is a much less urgent condition as a rule. There is little vomiting or shock and no dehydration, but absolute constipation is almost invariable. Blockage of the sigmoid is characterised by tenesmus, ballooning of the rectum, and inability to pass an enema. Abdominal *distension* is a marked feature; in the early stages a *dilated cæcum* is the keynote to diagnosis, but later the whole colon is distended and stands out (fig. 542), extending well into the flanks. A mass or tumour may be felt in the abdomen or per rectum. Finally, the small intestine dilates as well, and the abdomen becomes barrel-shaped.

A previous history of intestinal symptoms is always in favour of large-gut obstruction. Owing to hypertrophy of the muscular wall, *increased peristalsis* is a marked feature; it can always be heard on auscultation, and can often be seen and felt as well. The distended cæcum, which may dilate to an enormous size, is usually very conspicuous, and loud peristaltic splashes can be heard over it.

### III. DIAGNOSIS OF THE CAUSE OF OBSTRUCTION

The discovery of the actual cause of obstruction is the last step in diagnosis. It is very obvious that surgical treatment is more likely to be successful if one knows exactly what to look for. Unfortunately it is not possible to diagnose the actual lesion in all cases, and occasional errors are bound to occur. But an accurate diagnosis is more likely to be made if one carries in one's mind a rational clinical classification of the causes of obstruction.

I have found the following *classification*, based on the age, history, and clinical findings, of the greatest possible assistance:

- (a) *Congenital causes*—imperforate anus or rectum, congenital pyloric stenosis, congenital bands (rare).
- (b) *In early life*—strangulated hernia, intussusception, bands (appendix, tuberculous peritonitis), Meckel's diverticulum.
- (c) *In middle life*—strangulated hernia, adhesions and bands (post-operative or post-inflammatory), cancer, gall-stones.
- (d) *In late life*—cancer, strangulated hernia, faecal impaction, diverticulitis, volvulus, gall-stones, adhesions and bands.



- (e) *With previous history of operation or inflammation*—adhesions and bands near appendix, gall-bladder, in pelvis, or under abdominal scar; paralytic ileus.
- (f) *Cases with special symptoms and signs*—strangulated hernia, intussusception, carcinoma, gall-stones, volvulus, mesenteric embolism and thrombosis, paralytic ileus.

These individual causes of obstruction and their diagnosis will be considered more fully, either later in this article or in other sections of this work.

### TREATMENT

We must repeat that acute obstruction is not, in itself, a clinical or even a pathological entity. It follows that we cannot promulgate general lines of treatment to meet the indications of all, or even of a majority of obstructive lesions.

Unfortunately, it is not always possible to determine with certainty the exact lesion which may be present, and we are therefore forced to resort to some form of grouping of cases from the point of view of treatment. No better classification exists for this purpose than the one we have employed already in the sections on pathology and diagnosis, grouping the cases into main types and levels. Our general headings will thus be: (i) simple high small-gut obstruction; (ii) simple low small-gut obstruction; (iii) large-gut obstruction; (iv) internal strangulation. The treatment of strangulated hernia and paralytic ileus is not included, these conditions being discussed elsewhere.

Of course, only general principles and procedures will be dealt with in this section. Therapeutic measures of more specific application will be considered under individual obstructive lesions.

#### I. TREATMENT OF HIGH SMALL-GUT OBSTRUCTION

The treatment of high obstruction is governed by the physiological changes which determine its morbidity. The risk of strangulation is negligible and the stoppage is usually temporary. There is thus no indication for immediate operation; in fact, in most cases the necessity for operation never arises. This is certainly the case in acute dilatation of the stomach, in pyloric spasm and in nearly all obstructions of the proximal jejunum (mostly post-operative). Even when operation is indicated (e.g. in the rare cases of persistent obstruction of the

proximal jejunum) this should always be preceded and accompanied by the treatment now to be described.

The essential measure is the replacement of the fluid and plasma salts lost by the profuse vomiting and non-absorption of the digestive juices; this replacement automatically restores the plasma volume, dilutes the blood back to its usual concentration, corrects the dehydration of the tissues, and re-establishes normal renal function. These life-saving objects are best attained by the *intravenous* administration of some modification of Ringer's solution, which contains the essential

plasma salts in isotonic concentration; Hartmann's solution (sodium, potassium, and calcium chloride, with sodium lactate) is probably the best to use. To this, glucose (5 per cent) should be added periodically, as it provides badly-needed calories; glucose must never be given alone, since it stimulates the urinary excretion of salts and thus depletes the blood still further. In the absence of Hartmann's solution, normal glucose-saline will have to be employed, but this is not quite so effective as it does not restore the depleted plasma qualitatively.

The secret of success lies in

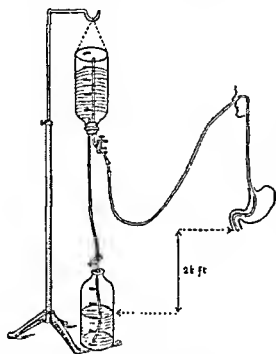


Fig 543.—DUODENAL DRAINAGE BY SUCTION SIPHONAGE, THROUGH NASAL ROUTE. (After Wangensteen.)

giving the intravenous fluid *slowly* (e.g. by the continuous drip method); if this cannot be guaranteed it is better to resort to continuous *subcutaneous* saline, and to punctuate this with four- or six-hourly intravenous feeds of glucose-saline (it is remarkable how quickly patients with a high obstruction absorb the subcutaneous saline). When the intravenous route is employed, great care must be taken to see that the amount of fluid given does not exceed the amount lost by vomiting and drainage.

The second essential in the treatment of high obstruction is *drainage* of the distended stomach and intestine above the block. This is best effected by the method of *suction-siphonage* with a duodenal catheter employed so successfully by Wangensteen (15); he passes the duodenal

tube by the nasal route (the oral route may be used instead), and insists on the importance of getting the tube into the duodenum. The advantages of duodenal drainage will be discussed later, while the actual management of the siphonage is shown in sufficient detail in the accompanying illustration (fig. 543). As soon as the obstruction is overcome the catheter can be employed to administer liquids and food; it should not be withdrawn while there is any likelihood of recurrence of the obstruction. In nearly all high cases complete relief of the obstruction may be expected in a few days. A record should be kept of the amount of fluid withdrawn, to provide a control on the quantity of fluid introduced intravenously or subcutaneously.

As an alternative to suction-siphonage, the duodenal catheter may be employed for intermittent drainage. The catheter is introduced through the mouth or nose and left *in situ*, the gastric and duodenal contents being withdrawn at regular intervals (e.g. hourly) by suction with a strong syringe.

## II. TREATMENT OF SIMPLE LOW SMALL-GUT OBSTRUCTION

The lower ileum is the commonest site of acute obstruction, and the treatment is here governed by the frequency with which *strangulation* occurs. If we exclude recent post-operative cases, something like 70 per cent of ileal obstructions are associated with strangulation. Unless, therefore, we can be certain that the obstruction is simple, it is far safer to treat the case as a strangulation, i.e. by *immediate operation*.

There remain, however, a number of cases in which strangulation can be safely excluded on clinical grounds. In these it may be wise to give conservative measures a short trial before resorting to operation, particularly in late cases. With a really early obstruction it may be difficult, indeed it is often wrong, to resist the urge to open the abdomen and restore the blocked lumen by a simple procedure, such as division of a band. But in late cases operation is so fatal, whether the cause of the ileus is removed or not, that the patient's best chance probably lies in conservative treatment.

In the section on morbid physiology it was shown that the morbidity of simple ileal obstruction mainly depends on distension of the proximal intestine, and also on sudden decompression of the dilated gut. The object of conservative treatment is, therefore, a slow *decompression*. It is here that duodenal drainage, by continuous or intermittent suction through a stomach tube, finds its most successful application. Wangenstein (15) employed it in twenty-four cases of mechanical obstruction,

with twenty-two recoveries and only two deaths; in four of them he had to perform an enterostomy ultimately. He states that pain is relieved at once and that the distension progressively subsides. The diminution of the distension *straightens out the kinks* which are usually responsible for the blockage, and so re-establishes continuity. The advantages of duodenal drainage over enterostomy are that it avoids operation and an intestinal fistula, and also effectively prevents air-swallowing (an important consideration).

If duodenal drainage, after a short trial, fails to relieve the obstruction, *operation* becomes imperative. What is done must depend on the condition of the patient. When this is good an attempt should be made to *find and deal with the cause*. If gross distension is present, an enterostomy should also be performed a little proximally to the obstruction, to avoid a sudden decompression of the released intestine.

In late cases, with a desperately ill patient, it is often wise to content oneself with a "blind" enterostomy, *provided strangulation can be definitely excluded*. The duodenal tube should be left undisturbed and suction continued after operation, whether enterostomy has been performed or not.

In all except the earliest cases, intravenous or subcutaneous administration of fluid is as necessary as in the high obstructions. Sufficient depletion of water or plasma salts will result from vomiting, siphonage, or enterostomy, to demand replacement. The benefits may not be so dramatic as in the high obstructions, but they are none the less real and life-saving. The use of *hypertonic intravenous saline*, which is still recommended by many authorities, is definitely contraindicated in *mechanical* obstructions, unless an outlet exists. Its action is to stimulate peristalsis, and although it may be useful in encouraging a sluggish enterostomy, without this outlet it can only result in increased pain and distension.

### III. TREATMENT OF ACUTE LARGE-GUT OBSTRUCTION

Internal strangulation of the colon is definitely rare; volvulus of the sigmoid is the chief cause, and it can usually be diagnosed before operation. No less than 98 per cent of large-gut obstructions are simple (carcinoma, diverticulitis, fecal impaction, etc.), and in a large majority of cases chronic obstruction precedes the acute. The danger of overlooking a strangulation of the large intestine is not more than 1 per cent (Lockhart Munimery, 20).

In view of this negligible risk, and also of the very high mortality

attending radical operations on acute colonic obstruction, there can be no question whatever that the treatment must be palliative. An attempt should first be made to convert the obstruction from an acute back to a chronic one, by withholding all food and drink and by repeated enemata. Duodenal suction-drainage may be of real service, particularly if the occurrence of vomiting shows that the distension has extended up the small gut, and the patient's strength must be maintained by the judicious use of intravenous glucose-saline. Peristaltic stimulants and aperients must be strictly forbidden. The factors which are responsible for the conversion of the partial chronic obstruction into a complete acute block are nearly always temporary (closure of narrowed lumen by faeces, or by spasm and congestion caused by an aperient); it is therefore not surprising that these conservative measures are frequently rewarded by re-opening of the lumen and recovery of the patient.

When the above treatment fails to give relief within a reasonable period (up to forty-eight hours), or the patient is too ill to justify the adoption of time-consuming measures, the only chance of survival lies in a *blind cæcostomy*, performed under local anaesthesia. The danger of an exploratory laparotomy in such cases, even when the diagnosis is uncertain, is many times greater than the danger of leaving behind a large-gut strangulation. After the cæcostomy, the continuity of the narrowed lumen is soon re-established, and then the actual lesion can be attacked at leisure.

#### IV. TREATMENT OF INTERNAL STRANGULATION

Ninety-five per cent of internal strangulations affect the small intestine, the lower part of the ileum being the common site. The primary factor of morbidity is *shock*, at first caused by trauma to the mesentery, and later aggravated by loss of blood and perhaps by sudden release of tension. Even greater is the lethal effect of *peritonitis*, from necrotic changes or perforation of the strangulated segment. These dangers are urgent and the patient's only chance of survival lies in *early diagnosis* and *timely operative intervention*.

*Anti-shock treatment* is started while preparations are made for operation: the patient is kept warm and 500-1000 cc. of glucose-saline are given intravenously, while a small injection of morphine supplies much-needed rest and prepares him for local or spinal anaesthesia. In cases operated on after the first day a *blood-transfusion* might make all the difference between success and failure. A catheter

should be passed into the stomach or duodenum before operation, and duodenal suction may be instituted with benefit after it (especially in high strangulations and in cases with much proximal distension). *Anti-Welchii serum* should be given in adequate doses (20 cc. of concentrated serum) if at all, but much doubt has recently been expressed (Wheeler 21, Holt 12, and others) as to its efficacy. The anti-shock measures must be continued after operation.

The operation resolves itself into four essential steps :

- (i) *Release the constricting mechanism*—this may mean dividing a band, widening an orifice, undoing a twist, or reducing an intussusception.
- (ii) *Decide whether the strangulated segment is viable, doubtful, or non-viable.*
- (iii) *If non-viable, deal with it.*
- (iv) *Provide for slow decompression* by duodenal drainage or enterostomy.

The treatment of doubtful or non-viable intestine will be discussed in the article on strangulated hernia, while further details of the operative treatment of internal strangulations will be given in a later section of this article.

### OPERATIVE TREATMENT

The following operations are employed in the treatment of acute obstruction :

- (a) *Exploratory laparotomy*, for obstruction of uncertain origin.
- (b) *Planned laparotomy*, for obstruction of known type and origin.
- (c) *Operations for drainage of intestine*—enterostomy and cecostomy.

The actual steps of operative treatment are four : (i) the preparation, (ii) the anæsthetic, (iii) the operation, (iv) the after-treatment.

#### I. PREPARATION

Most therapeutic procedures which come under this heading have already been dealt with in sufficient detail, and can be summarised as follows : (i) one or two enemata, which serve to clear the lower bowel as well as to establish diagnosis ; (ii) insertion of a tube through the

mouth or nose into the stomach or duodenum, with provision for suction ; (iii) anti-shock measures, such as rest, warmth, and a small dose of morphia ; (iv) intravenous glucose-saline, subcutaneous saline, or Hartmann's solution, according to the indications given above ; (v) blood-transfusion in certain cases, e.g. large strangulations ; (vi) anti-gas gangrene serum, if thought advisable ; (vii) skin preparation.

## II. THE ANÆSTHETIC

Inhalation anæsthesia is definitely contra-indicated in acute obstruction. Apart from its toxic effects, there is a grave danger of asphyxia, broncho-pneumonia, and acid digestion of the lungs, from the inhalation of vomit. A stomach tube largely obviates this risk, nevertheless general anæsthesia is a very real danger to an obstructed patient. Chloroform and ether are particularly objectionable, as they definitely predispose to the subsequent occurrence of paralytic ileus. Gas and oxygen is fortunately free from this objection.

Unquestionably the ideal for major operations is *spinal anæsthesia*, its only contra-indication being a low blood-pressure. It provides excellent relaxation, and so facilitates the search for a cause, and minimises the escape and handling of coils of distended intestine ; these are advantages of profound importance. The abdominal wall can be lifted up for the insertion of a surgical light when delicate or difficult intra-abdominal manipulations are necessary. Vomiting is much less likely to occur, and the danger of inhalation of vomitus is abolished. Finally, it throws the sympathetic out of action and thus, by stimulating peristalsis, rapidly restores the intestinal stream after the removal of a mechanical obstacle ; moreover, in cases of paralytic ileus, a spinal anæsthetic may be followed in a few minutes by the passage of flatus and fæces, and thus might avoid what is bound to prove an unnecessary and harmful operation. If the blood-pressure falls to a dangerous level, an intravenous glucose-saline should be administered without delay ; up to half an ounce of brandy may be added to this with benefit. Ephedrine is best avoided in obstruction.

In minor procedures, e.g. cæcostomy or enterostomy, *local anæsthesia* should have the first preference. A thorough infiltration of the abdominal wall in the region of the proposed incision, with 1 per cent novocaine, is all that is needed ; the actual manipulating, opening, and suturing of the intestine cause no pain whatever, provided gentleness is observed.

In *shocked patients* with a low blood-pressure, and in *small children*, we must also rely on local anaesthesia, even for major procedures. During the intra-abdominal part of the operation a little gas and oxygen will usually be required.

### III. THE OPERATION

Every operation on an acutely obstructed patient should be undertaken with the sole object of saving life; removal of the obstructing cause or lesion must not be attempted unless it is essential to the attainment of this object. Two rules must govern our procedure: (i) "to get in quick and get out quicker" (Murphy); (ii) to do the minimum consistent with ultimate recovery.

(a) *Exploratory Laparotomy.* With our realisation of the importance of accurate clinical diagnosis "exploratory" operations are becoming less common than they were. Of necessity, a large incision and prolonged search add enormously to the risks of operative treatment; every attempt should therefore be made to avoid unnecessary explorations by narrowing the diagnosis, if possible to the actual cause, if not, at least to the type and level of the obstruction.

There remain, unfortunately, a number of cases in which exploratory operations are unavoidable; they are mostly low small-gut obstructions and internal strangulations of uncertain origin. Large-gut acute obstructions, if diagnosed, should under no circumstances be "explored."

*Technique.* The best general incision is a right paramedian, one-third above and two-thirds below the umbilicus. In the first place it should be made large enough only to admit two fingers; the peritoneum must be opened with great care to avoid injury of the distended coils, which have a habit of presenting first. The fingers are immediately passed to the caecum. *If the caecum is distended* the obstruction is in the large gut and the operation must be stopped: the laparotomy wound is closed at once, and a caecostomy established through a stab wound over this organ.

*If the caecum is collapsed* the obstruction is in the small intestine and the exploration must be proceeded with. The incision is carefully enlarged until it admits the hand (about  $4\frac{1}{2}$  inches), and the *hernial orifices* are palpated from within for a possible "hidden" strangulated hernia or a *Richter's* hernia. Should a loop of distended gut be felt passing into a hernial orifice, the abdominal wound is packed with a



saline swab, and the hernia exposed and dealt with by a suitable incision over it.

If the bernal sites are clear we must be prepared for a difficult operation. The chief troubles are caused by distended coils which keep presenting into and out of the wound, by blood-stained fluid which may well up from the abdominal cavity in large quantities, and by fat-laden fringes of omentum which do the same. If necessary, the incision should be further enlarged, but the only certain way of finding the lesion quickly is by a *methodical and intelligent search*. Starting with the ileo-cæcal junction, the surgeon should run the collapsed gut through his fingers (and return it into the abdomen) loop by loop, until it meets the distended gut; this junction marks the site of obstruction, and the actual lesion (a kink, twist, or obturation; a constricting band or aperture, etc.) is now seen and dealt with. Very often the obstruction is found in the last few feet of the ileum, either in the right iliac fossa or in the pelvis; in the last case it may be wise to put the patient in the Trendelenburg position and pack the intestine out of the way, as this will greatly facilitate the necessary manipulations.

Having removed the cause of obstruction, we now turn our attention to the intestine. When this is healthy and only moderately distended, nothing further need be done; having satisfied ourselves that peristalsis is now occurring through the recent block, and that the collapsed intestine is receiving contents, we thankfully close the abdomen. But if the intestine is grossly distended, unquestionably the wise thing to do is to perform an *enterostomy*; the distended intestine is drained as near the obstruction as possible, the catheter being brought out through a separate stab wound. When *non-viable* strangulated gut is present the surgeon must choose between primary resection and anastomosis, and exteriorisation with secondary resection.

(b) *Planned Laparotomy*. Operations for individual obstructive lesions will be dealt with in the second part of this article.

(c) *Drainage or Decompression Operations*. The essential object of these operations is a slow decompression of the distended intestine, and not a diversion of the intestinal contents from their normal channel. Of the two procedures available, enterostomy is employed for small-gut obstruction only, and cæcostomy for large-gut obstruction.

(i) *Enterostomy*. Whenever possible this should be performed *after* the obstructing cause has been found and dealt with. The

only indication for a "blind" enterostomy is a late small-gut obstruction in a very ill patient. *if strangulation can be excluded with certainty.*

When preceded by a laparotomy, the enterostomy should be performed through a stab incision clear of the main wound. A "blind" enterostomy is best made through a small oblique incision at the edge of the left rectus opposite the navel; only local anaesthesia should be used. A distended loop is chosen conveniently near the obstruction. The loop is brought out, emptied by milking or aspiration, and

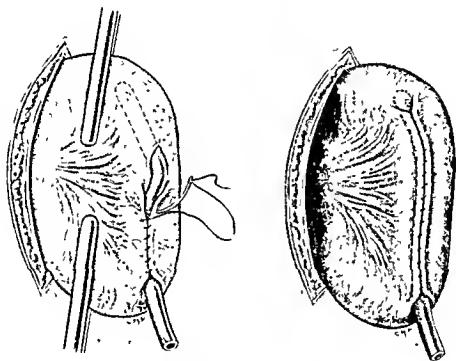


Fig. 544.—ENTEROSTOMY BY WITZEL'S METHOD.

rubber-covered clamps are applied at each end. It is surprising how the thinned-out wall of the distended gut thickens after it is emptied. Great care must be taken to see that no contents escape into the wound, as this might easily lead to a fatal peritonitis.

Drainage is instituted with a self-retaining catheter or an ordinary soft rubber catheter, 14 to 16 French, which should be buried in the gut-wall, preferably by the *Witzel method* (see fig. 544). The burying suture starts  $1\frac{1}{2}$  inches proximal to the puncture for the catheter, and continues for another  $1\frac{1}{2}$  inches beyond it. The puncture itself should be only just large enough to admit the tube and should be made with a fine scalpel. The loop is then returned into the abdomen and the

catheter may be surrounded with a sleeve of omentum if desired. Great care must be taken after the operation to keep the lumen of the catheter clear. Every few hours, and at the first sign of blockage, a little saline should be forced through with a syringe. This is particularly necessary when the opening is in the low ileum.

The catheter is removed when no longer required, and the small peritoneal tunnel heals, usually without leakage. A persistent fistula is likely to result when the lumen has been narrowed too much by the invaginating suture, or when too large a catheter has been employed.

(ii) *Cæcostomy.* A blind cæcostomy, under local anaesthesia, is the one life-saving operation for acute large-gut obstructions. The distended cæcum is exposed by a small oblique incision outside the rectus, and brought up into the wound; it is very friable and must be handled with great care. A purse-string suture is inserted round the area chosen for incision, which is made with a fine scalpel, a medium-sized Paul's tube is pushed in and the suture drawn tight. I have given up catheter-decompression for the cæcum; the contents are too solid and much trouble may be experienced from blocking of the catheter. The cæcum is fixed with two catgut sutures, which also serve to close the peritoneum, and one or two through-and-through silkworm-gut sutures suffice to close the wound.

The cæcostomy may heal spontaneously after the obstructing cause has been removed at a later operation, or after a permanent colostomy has been performed for irremovable obstruction. If it does not so heal, a third operation will be needed to close it.

The mortality of simple enterostomy or cæcostomy under local anaesthesia is extremely low.

#### IV. THE AFTER-TREATMENT

Many lives are lost by relaxation on our part after operation. For the first few days the patient's condition remains critical, and life-saving measures must be continued, if anything with greater enthusiasm and energy than before. The stomach tube should be left in while anything can be sucked up, and may, in fact, be used for the first day or two after this as a vehicle for oral feeding. Rest, sleep, and warmth are very necessary, and intravenous or subcutaneous saline and glucose must be continued until fluids can be given by mouth (12-24 hours after operation).

Sluggish return of peristalsis should be countered by intestinal stimulants, such as intravenous hypertonic saline or acetyl-choline. An ox-bile enema may also be employed for the same purpose. These measures are particularly useful in late cases, in which some degree of paralytic ileus is almost inevitable.

### THE VARIETIES OF OBSTRUCTION

IN this section will be considered the special problems associated with individual types of obstruction. Strangulated external hernia, and paralytic and early post-operative ileus are dealt with in separate articles, thus leaving adhesive obstruction, internal strangulation, obturation, intussusception, volvulus, and mesenteric vascular occlusion for consideration here.

#### ADHESIVE OBSTRUCTION

*Pathology.* Abdominal adhesions are occasionally congenital, but more often they follow injury to the peritoneum (actual trauma or inflammation). As a cause of obstruction, they are almost always preceded by abdominal operation or by a local or general peritoneal inflammation; in both cases they are at first soft and fibrinous, lightly glueing adjacent coils of intestine to each other, or to their surroundings. These fibrinous adhesions are in great part absorbed, but occasionally a proportion of them organise into fibrous folds, sheets, or bands.

Individual patients vary in their response to peritoneal injury; some develop extensive adhesions after a mild peritoneal inflammation or trauma, while others may get over a severe peritonitis without any adhesions whatever.

Most frequently adhesions are *local*, and follow localised inflammations (e.g. appendicitis, tuberculous glands, salpingitis, etc.), or operative trauma. Post-operative adhesions particularly occur after appendix operations (especially when complicated by abscess), pelvic operations, and operations on the stomach, colon, and gall-bladder. Their likelihood is greatly increased when raw areas are created by denuding them of peritoneum, and also when prolonged drainage has been employed.

Local adhesions attach coils of intestine to each other, to an inflamed organ (e.g. the appendix, a mesenteric gland, the omentum), to a

denuded area on the abdominal wall, or to an operation scar. They occur as fine filaments, thin membranous sheets, or short dense fibrous bridges, and in the course of time they may be stretched and moulded into actual *bands*.

Occasionally adhesions are *generalised*, when they mostly follow tuberculous or chronic pyococcal peritonitis. In such cases the adhesions are short and extensive, and may obliterate a large part of the peritoneal cavity; cicatricial contraction of the mesentery also occurs,

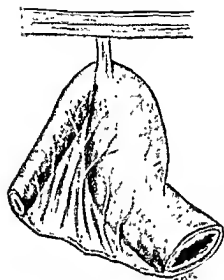


Fig. 545.—KINKING BY AN ADHESION.  
(INCREASED BY PROXIMAL DISTENSION.)

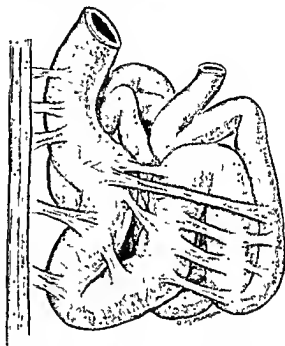


Fig. 546.—FIXATION BY MULTIPLE ADHESIONS.

the intestine being pulled back to the posterior parietes. This general fibrosis is mainly responsible for *chronic* obstruction, and it is unusual for such cases to terminate acutely.

Acute obstruction caused by adhesions (as distinct from actual bands) is rarely complicated by strangulation. The mechanism of its production is simple, the most common effect being a local *kinking* of the gut (fig. 545); less often the adhesion *compresses* or flattens a segment of intestine against a solid structure, such as the posterior abdominal wall (see fig. 536). In either case the blockage is incomplete in the first place, but tends to become complete as the effect of the kink or compression is *aggravated by distension* of the intestine. With relief of the distension the intestine straightens out and the obstruction may again become incomplete. Another way in which adhesions cause obstruction is by fixing coils to the parietes or by attaching them to

each other, thus distorting their lumen (see fig. 546), or arresting peristalsis. In rare cases the adhesive process is responsible for the formation of a *volvulus*.

*Diagnosis.* In this section only post-inflammatory and late post-operative adhesions are discussed, early post-operative adhesions being dealt with in a separate article.

Adhesive obstruction is rarely of sudden onset, and its course is usually sub-urgent. There may be a past history of abdominal operation (months or years previously), or of an inflammatory lesion. Very often, several attacks of threatening obstruction (colicky pains, constipation and vomiting) have occurred over a long period preceding the acute obstruction, but they have been of short duration, and thus far relieved by an enema or aperient. The severity of the attacks increases until complete blockage develops, but even this is fairly gradual in its onset.

The *symptoms and signs* are typically those of a simple low small-gut obstruction. In over half the cases there is a previous history of appendicitis, with or without operation, while in 95 per cent the small gut is involved (in nine-tenths it is the ileum). Severe colicky pains, vomiting, marked increase of peristalsis (particularly on auscultation), and central distension of the abdomen are the outstanding clinical features, but constipation may not be absolute. An abdominal scar is frequently present, and peristalsis may be loudest near it.

These features often combine to produce an unmistakable picture. The frequency of early diagnosis is proved by Vick's figures, which show that of 505 cases of acute adhesive obstruction 60 per cent were submitted to operation within forty-eight hours of the onset of symptoms.

In some cases there is a pronounced tendency to recurrence of the acute obstruction. I have treated one patient whose abdomen had been opened six times for complete stoppage by adhesions, while Wangenstein mentions cases which have been operated on more than twenty times!

*Treatment.* In spite of relatively early diagnosis the mortality of acute adhesive obstruction is over 30 per cent. One cannot help wondering whether the orthodox treatment—i.e. immediate laparotomy and division or freeing of the adhesions—is not in some measure responsible for this death-rate, especially in advanced cases.

With an early case rapid operation is undoubtedly the safest procedure. We generally know in advance where the obstruction is and

can attack it directly, through an appropriate incision. The liberation of the kinked or compressed intestine may be a very simple matter, merely requiring division of a short fibrous bridge or band, or dissection and removal of a thin sheet of fibrous tissue. Sometimes a more tedious dissection is needed to free perhaps several adherent coils from an abdominal scar or some other structure. Occasionally, the adhesions are so extensive that the attempt to liberate the intestine may prove lethal to the patient; moreover, such an attempt is followed by an inevitable recurrence of the adhesions, often in an aggravated degree. In these cases, it is far better to short-circuit the obstruction by a lateral anastomosis, provided the distension is moderate.

No operation for adhesive obstruction is complete unless all scar tissue in the abdominal wall is excised and all raw areas are covered by peritoneum. Only thus can recurrence be avoided.

In late cases, with a grossly distended intestine and a dehydrated patient, a laparotomy directed at the cause of the obstruction is usually fatal, and the best chance of saving life lies in conservative treatment. Slow decompression with a duodenal tube should be instituted at once, and *dehydration* and *collapse* treated by intravenous glucose-saline or Hartmann's solution. In some cases these measures relieve distension completely, and so overcome the obstruction; in others, they diminish the distension and minimise the danger of subsequent operation. If the relief is inadequate, an *enterostomy* should be performed under local anaesthesia, thus completing the decompression. This is not entirely a "blind" enterostomy, as the risk of overlooking a strangulation is greatly minimised by the abdominal incision (a strangulation nearly always reveals itself by the escape of blood-stained fluid).

Should obstructive symptoms recur when drainage is discontinued, one can pass on to a laparotomy, aimed at release of the intestine, with a better prospect of survival of the patient.

### INTERNAL STRANGULATION

*Pathology.* Under this heading are included strangulations by bands and internal apertures.

*Bands.* These are mostly seen in the form of arches, under which a loop of small gut passes and becomes strangulated (see fig. 537). Such bands are *fixed*, being attached at both ends to fixed points, such as the

posterior abdominal wall, mesentery, or pelvic organs. Less often the bands are *free*, forming snares or knots which catch a loop of intestine and strangle it.

Bands are of three varieties: (i) *Peritoneal* (fig. 547)—formed of adhesions, stretched and moulded by movements of the viscera into

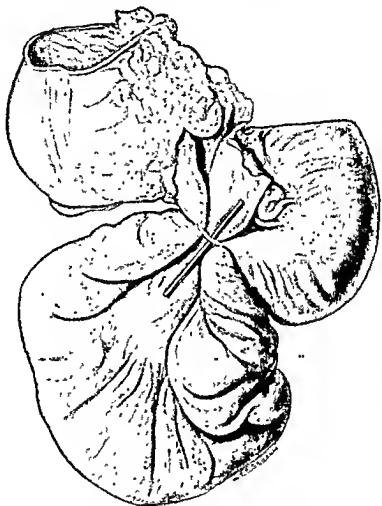


Fig. 547.—STRANGULATION BY PERITONEAL BAND.  
(From the Museum of the Royal College of Surgeons.)

strong fibrous cords or ribbons, of varying thickness and length; they are mostly seen at sites of previous inflammation or under an operation scar. (ii) *Omental*—the edge of the omentum attaches itself to an inflamed structure (e.g. the appendix or a tuberculous gland) and is gradually stretched and moulded into a band; the omentum being connected above to the mobile stomach and transverse colon, it follows that strangulation produced by it tends to be sub-acute. (iii) *Visceral bands*—these are mostly formed by an adherent appendix or Meckel's



diverticulum (fig. 548), less often by the Fallopian tube, an appendix epiploica, the pedicle of an ovarian or uterine tumour, and even by a loop of the intestine itself.

*Internal Apertures.* Holes in the *mesentery*, *mesocolon*, *omentum*, or *broad ligament* are occasionally congenital, but more often result from trauma or operation. A loop of intestine passing through such a hole is liable to be strangulated by its edges.

Strangulation may also result if a loop of gut enters one of the retroperitoneal fossæ produced during embryonic development and

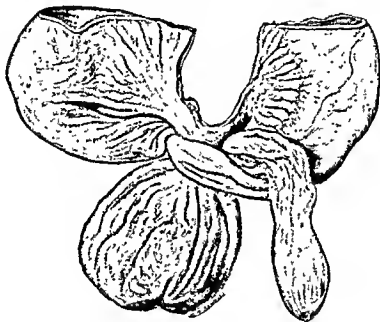


Fig. 549.—STRANGULATION BY MECKEL'S DIVERTICULUM.  
(From the Museum of the Royal College of Surgeons.)

mainly associated with flexures of the intestinal canal. The more important of these fossæ are: (i) the *paraduodenal fossa*, to the left of the ascending limb of the duodenum; (ii) the *mesenterico-parietal fossa*, below the duodenum and immediately behind the root of the mesentery and superior mesenteric vessels; (iii) the *ileo-cæcal fossa*, between the appendicular mesentery and the ileo-cæcal fold of Treves; (iv) the *retrocæcal fossa*, behind a partially anchored cæcum; and (v) the *retrosigmoid fossa*, behind the pelvic mesocolon with its sigmoid vessels, and in front of the bifurcation of the left common iliac artery.

Finally, strangulation may occur through congenital or acquired openings in the *diaphragm* or *pelvic floor*, and through the *obturator foramen*, while rarely the *foramen of Winslow* may act as an agent of

strangulation. All these openings must be regarded as potential hernial orifices, and occasionally *internal herniæ* may occur through them without strangulation.

*Mechanism of Strangulation.* Once a loop has passed through, the band or the edges of the aperture exert a constricting, valve-like action, and prevent the escape of contents. Distension of the loop occurs, and this draws more intestine into the "one-way-trap," until strangulation finally supervenes.

The *morbid changes* in the strangulated loop and the *physiological factors* underlying the morbidity of strangulation have been discussed already, but may be briefly summarised as follows: The first disturbance is *shock*, caused by injury to the mesentery; when a *small loop* (under a foot) is strangulated, early *necrosis* and *perforation* occur and cause a fatal *peritonitis*; with *moderate loops* (one to two feet) the lethal factor appears to be *absorption* by the peritoneum of the blood-stained *exudate* from the engorged intestine, which after some sixteen hours becomes intensely *toxic*; in strangulation of *long loops* (two feet or more) death may be caused by loss of blood from hæmorrhage into the engorged coil and into the peritoneal cavity.

*Diagnosis.* The clinical features of strangulation have been considered at sufficient length in the section on general diagnosis. We may repeat that the *onset* is nearly always *sudden*, with *severe pain* and initial *shock*, and that the *course* is usually extremely *acute*. The pulse tends to be rapid, and there may be some fever. The patient is obviously ill and becomes rapidly worse. There is often some abdominal tenderness and rigidity, owing to extravasation of blood into the peritoneum, and occasionally blood may be passed *per rectum*. Sometimes the tense strangulated loop can be palpated.

These symptoms and signs are all produced by the strangulation, and are absent in cases of simple blockage. Obstructive symptoms are present as well, their intensity varying somewhat with the level of the block, but owing to the short duration of life they are rarely very pronounced.

An exact diagnosis of the *cause* of internal strangulation is not often possible. The presence of an operation scar or the history of previous abdominal disease may lead us to the actual site, while the palpation of the tense strangulated loop occasionally acts as a guide (I once correctly suspected a strangulated paraduodenal hernia by finding a combination of acute high obstruction with a tense globular lump

above and to the left of the umbilicus). The discovery, on auscultation, of the point of maximal peristaltic noises may also help in this direction.

*Treatment.* This is governed by the urgency of the lesion and by the fatal risk of peritonitis from necrotic changes in the strangulated loop. The need for *anti-shock measures* and the advantages of *blood-transfusion* have already been referred to; likewise the administration of *anti-Welchii serum* has been discussed.

*Operation* is imperative and must be performed without delay; it provides the only protection against the otherwise inevitable fatal termination. In most cases the operation starts as an *exploration*. Either spinal or local anæsthesia is employed, with the addition of gas and oxygen when necessary, a stomach tube having been previously passed. Escape of *blood-stained fluid* immediately confirms the presence of a strangulation, and the actual site is now sought for by the methodical procedure described on page 1004.

The next step consists in *release of the constricting mechanism*. When this is a peritoneal or omental *band*, it should be divided between forceps and excised. Care must be taken to ensure that the band is not visceral; a segment of intestine, acting as a constricting agent, may be so stretched and twisted that it could be easily mistaken for a band, and divided. When the bowel is strangulated by an aperture, special care is needed to avoid injury to the vessels which so often run in its boundaries; it is always safer to stretch the opening with the fingers or with forceps, than to divide the edge with a scalpel or scissors. After withdrawal of the strangulated loop the aperture should be carefully sutured, to prevent recurrence of the internal hernia.

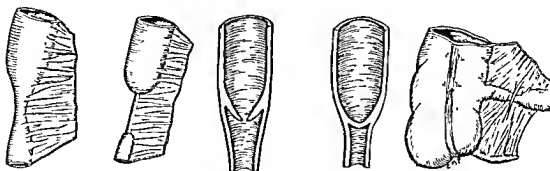
The treatment of the strangulated loop and the after-treatment of the patient have been considered already, and will be discussed in greater detail in the article on strangulated hernia.

### CONGENITAL OBSTRUCTION

Congenital anomalies may cause obstruction at or soon after birth, or they may be determining factors of obstruction later in life. The first or direct causes are rare; the second, or indirect, are more common.

*Direct causes* come under the heading of mal-development, and are mostly seen near the sphincters, i.e. in the duodenum, lower ileum, and

rectum. The various types are sufficiently indicated in the accompanying diagram (fig. 549). Congenital bands, and incomplete atresias



PARTIAL ATRESIA    COMPLETE ATRESIA    PARTIAL SEPTUM    COMPLETE SEPTUM    CONGENITAL BAND

Fig. 549.—TYPES OF DIRECT CONGENITAL OBSTRUCTION.

(stricture) and septa tend to cause partial obstruction at first, while complete atresias and septa will, of course, result in complete stoppage from birth (fig. 550).

*Indirect congenital causes* may produce obstruction at any age, but there is naturally a tendency for it to affect adolescents and young adults, rather than old people. Among these causes are: (i) *Meckel's diverticulum* (acting as a band, or initiating an intussusception or volvulus)—obstruction by Meckel's diverticulum might be suspected



Fig. 550.—CONGENITAL ATRESIA OF RECTUM.  
(St. Mary's Hospital Museum.)

if a young person develops a low small-gut obstruction, for which no obvious cause exists; (ii) *defects of rotation and fixation of the embryonic gut*, which may determine the development of volvulus of the small intestine or caecum; (iii) *megacolon* (Hirschsprung's disease), in which increasing chronic obstruction may terminate acutely, either in childhood or in adult life.

## OBTURATION

The intestinal lumen may be blocked by gall-stones, faecal impaction, or enteroliths.

## I. GALL-STONE ILEUS

Gall-stones constitute the commonest cause of acute obturation; in Vick's series of 6,892 cases there are 47 gall-stone obstructions (0·7 per cent of the total). Only stones ulcerating from the gall-bladder into the duodenum are likely to cause obstruction (when one inch or more in diameter). They pass through the wide and relaxed jejunum safely, but tend to be arrested in the lower part of the narrow and spastic ileum; complete obstruction is caused by spasm of the irritated circular muscle round the impacted stone. Stones coming down the bile-duct are too small to obstruct a normal lumen, while stones ulcerating into the colon find the latter too large and accommodative.

*Diagnosis* is made difficult by the sub-acute course of the obstruction, and by the tendency, both on the patient's and doctor's part, to mistake its early stages for "another attack of gall-stones." Late diagnosis is thus usual; of Vick's 47 cases only 14 were operated on within 48 hours of the onset. It is scarcely surprising that the mortality is about 70 per cent, although it must be admitted that the patients are usually had subjects for operation.

Actually, it should be possible to recognise the presence of acute obstruction by the second day. The patient, generally a fat woman of fifty or over, gives a long-standing history suggestive of gall-stones, with a recent exacerbation due to the ulceration of the stone into the duodenum. After a short interval of quiescence, the characteristic symptoms of a low small-gut obstruction gradually set in. Vomiting is very profuse (at first dark and later "faecal") and there are intermittent colicky pains. Shock is slight or absent, constipation may not be absolute for a day or two, and distension is masked by the obesity which is usual in these cases. Occasionally, the stone may be felt by rectal or vaginal examination. On auscultation there is marked increase of intestinal sounds, usually near the umbilicus, but in late cases peristalsis may actually be less than normal owing to the onset of paralysis. Sometimes the stone may be seen by X-rays.

*Treatment* consists in immediate laparotomy, preceded and followed by duodenal drainage and replacement of lost fluids. Under spinal

anæsthesia a low right paramedian incision is made, and the stone is usually felt at once in the lower foot or so of the ileum. The loop is withdrawn and emptied by milking. After clamps have been applied, a longitudinal incision is made along the anti-mesenteric border a little proximal to the stone, and this is removed. Some of the contents are allowed to escape and the incision is closed transversely with Lembert sutures, so as not to narrow the lumen. In late cases with much distension it is wise to institute enterostomy, through a stab incision near the laparotomy wound.

## II. OBSTRUCTION BY FÆCAL IMPACTION

Considering the frequency of obstinate constipation it is amazing that complete obstruction by fæcal masses is so rare (Viek's 6,892 cases include only 17 obstructions from this cause). The pelvic colon and rectum form the usual site, the patients are mostly elderly women, and the symptoms of complete blockage supervene gradually after a long period of chronic obstruction. The acute attack starts with colicky pains and is followed by extreme distension and collapse; a tender mass can be felt along the left colon, which may pit on pressure. The rectum is often full.

*Treatment* should start with two or more enemata, in the attempt to avoid operation. Unless relief is rapidly afforded a *cæcostomy* should be performed. When the acute symptoms have subsided, the mass is softened and removed by injections through the cæcostomy and anus.

## III. ENTEROLITHS

These are mostly produced from accumulations of food with a high cellulose residue (husks of corn, bran, and green vegetables). Less often they consist of mineral matter (e.g. calcium carbonate) which may have been taken medicinally. Bismuth and barium given for radiography have been known to complete an obstruction caused by carcinoma. Sometimes these "stones" form in a sacculus and cause blockage when they are displaced into the lumen.

Acute obstruction by enteroliths is extremely rare, while the treatment is identical with that of gall-stone ileus.

## ACUTE INTUSSUSCEPTION

*Incidence.* Acute intussusception is essentially a disease of infants and small children. Perrin and Lindsay (22) state that 78.5 per cent occur in the first year, the sixth and seventh months showing the highest incidence (fig. 551). There is a definite seasonal variation, with peaks in March and December. The condition is more than twice as common in boys as in girls, and the children afflicted with it are otherwise usually healthy.

*Mechanism.* This has been the subject of much speculation. The modern view, basing its arguments on the known production of intussusception by Meckel's diverticulum and polypoid tumours, inclines to an *organic* explanation, even for the common cases in which no such causes exist. In these it is thought that swollen lymphoid patches (perhaps from a mild enteritis) play the part of an irritant. The predominance of the ileo-cæcal variety is thus attributed to the presence of large Peyer's patches in this region; but it can also be explained by the powerful detrusor action of the terminal ileum, and by the greater size of the colon, which is ready to act as a sheath.

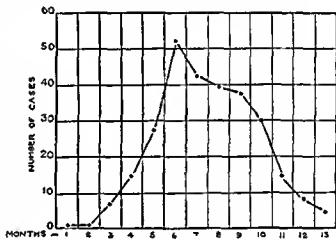


Fig 551.—INCIDENCE OF ACUTE INTUSSUSCEPTION DURING FIRST YEAR OF LIFE. (After Perrin and Lindsay)

The usual theory is that the polyp or lymphoid patches irritate the gut into strong peristalsis, which drags the "foreign body" and its point of attachment distally and so starts the invagination. Wardill (23), however, has shown that the polyp is not always found at the apex of the intussusception, as would be the case if this theory were true; he suggests that the invagination is started by a *fixed* spasm.

When no local cause exists, it is probable that the true explanation is *functional*. Fitzwilliams (24) supports Treves' suggestion that *local* spasm is a sufficient cause, contraction of the longitudinal muscle

dragging the relaxed distal gut *backwards* over the spastic segment. According to this view, the sheath plays the active part, and the intussusception grows at its expense.

### MORBID ANATOMY

Typically, an intussusception forms a firm, curved, sausage-shaped swelling, composed of three concentrically arranged tubes or layers:

the *entering* layer, the *returning* layer, and the *ensheathing* layer. The former two constitute the *intussusceptum* and meet at the *aper*, or most distal part of the invagination (see fig. 553). The ensheathing layer forms the *intussusciens*, and joins the returning layer at the *neck* of the intussusception. As the invagination grows in length, the *mesentery* is dragged into the intussusceptum, where it is wedged in between the entering and returning layers on the concave side. The tension on the mesentery

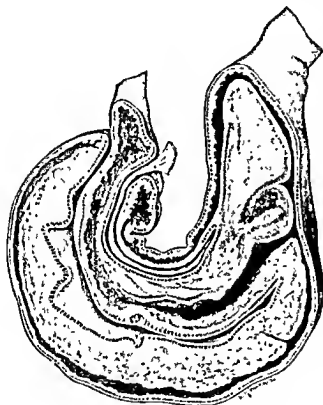


Fig 552. ENTERIC INTUSSUSCEPTION.  
(From St. Mary's Hospital Museum.)

produces the curved shape of the intussusception, the concavity facing the mesenteric attachment, and at the same time drags it back towards the posterior abdominal wall.

The constriction and twisting of the mesentery sets up a congestion from pressure on the veins, which is most marked at the apex and in the adjacent part of the returning layer (see fig. 552). As a result, an ooze of blood and mucus occurs into the bowel, and the apical part swells, sometimes enormously, thus obstructing the lumen. Later on, adhesions tend to form between the apposed serous surfaces of the entering and returning layers, and these adhesions, together with the



apical swelling, may seriously hinder reduction. Finally, the oedematous and congested intussusceptum is attacked by bacteria and gangrene may supervene. The sheath is not implicated and acts as a protection; indeed, very rarely the entire intussusceptum is cast off as a slough and a spontaneous cure results. More often the necrosis terminates in peritonitis.

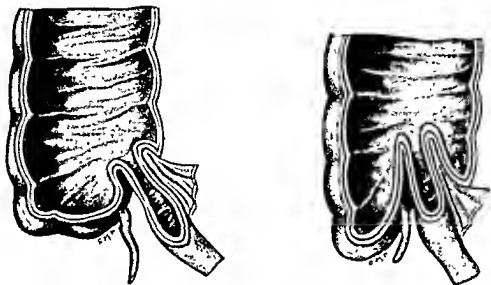


Fig. 553 and 554.—STAGES IN THE FORMATION OF ILEO-CAECAL INTUSSUSCEPTION.

*Classification.* Of recent years the anatomical varieties of intussusception have been multiplied unnecessarily. Actually there are three main forms :

(a) *Enteric*—small-gut into small-gut. These account for about 10 per cent of cases and occur in older children and adults. Most of them show an organic cause, e.g. an inverted Meckel's diverticulum, an adenomatous polyp (see fig. 556), a tuberculoma, or a carcinoma.

(b) *Colic*—colon into colon. This type occurs in oldish people (the usual cause is a polypoid carcinoma), and accounts for some 5 per cent of intussusceptions.

(c) *Entero-colic*—ileum into colon : the common variety in infants and embracing 85 per cent of cases. There are three distinct sub-types of this variety : (i) *ileo-caecal*, the ileum passing into the colon with the ileo-caecal valve as the fixed apex ; (ii) *ileo-colic*, starting in the terminal ileum (apex short of the valve), and then proceeding into the colon (fig. 555) ; (iii) *enteric ileo-caecal*, starting as an enteric intussusception, becomes wedged in the ileo-caecal valve, pushes this before it, and then proceeds as an ileo-caecal intussusception, with the valve at its apex. The so-called *caput caeci* type is an ordinary ileo-caecal

invagination in which the loose outer wall of the cæcum slips beyond the apex, and shows as a dimple after reduction (Fitzwilliams).

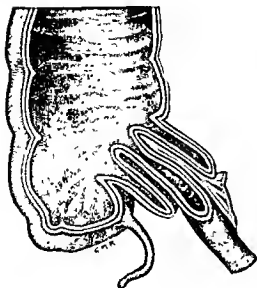


Fig. 555.—ILEO COLIC INTUSSUSCEPTION.

The common entero-colic intussusception of infants tends to traverse the greater part of the colon. Swinging round the attachment of the mesentery, clock-wise, the intussusceptum passes up the right colon, along the transverse, and down the left colon, and may actually reach the rectum and even protrude through the anus. Complete obstruction of the lumen is a secondary, and usually a late result, being caused by oedematous swelling of the most advanced part of the invagination, perhaps assisted by spasm.

### DIAGNOSIS

In the common intussusception of *infants* the clinical picture is usually very characteristic and errors of diagnosis are uncommon. Of all the causes of obstruction it is the earliest diagnosed: thus of 989 cases collected by Vick no less than 727 (73·5 per cent) were operated upon within 24 hours of the onset. The beneficial result of this early diagnosis is reflected in the relatively low mortality (17·6 per cent in Vick's cases).

The *onset* is usually sudden and typical, with *severe pain, shock, and initial vomiting*. The child, mostly a healthy male infant, suddenly screams with pain, draws up its legs and vomits. Its face is *pale* and its eyes widely dilated with fear. After a time the symptoms abate and the child seems better, but it is *listless* and may remain pale and shocked. This "interval" pallor is very suggestive. Very soon the *colicky pains* return and the child looks ill again. There may be several such attacks with free intervals. Vomiting tends to occur with each attack of colic, but it is rarely a pronounced symptom.



Fig. 556.—INTUSSUSCEPTION CAUSED BY ADENOMA. (From St. Mary's Hospital Museum.)

The stools are very variable. At any period, and especially soon after the onset, the child may pass a *normal stool*. Subsequently faecal matter may be passed with blood, or only blood and mucus may be evacuated. Tenesmus is usual in the later stages. In the absence of bloody motions, a finger passed into the rectum is often withdrawn covered with blood and mucus. In neglected cases the pain becomes continuous, the abdomen distends, and fatal collapse sets in.

Careful examination of the abdomen reveals a lump, firm and sausage-shaped, in about three-quarters of the cases. More often than not this is felt in the transverse or descending colon, and it hardens during an attack of colic. Gentle palpation and a warm hand are essential. In late cases the lump may be palpated on rectal examination. Occasionally, however, the intussusception cannot be felt, either because it is inaccessible (e.g. in the splenic flexure) or because the abdomen is rigid through crying. In such cases it is often palpable under anaesthesia. The "signe de Dance" is probably a myth.

*Differential diagnosis.* The only conditions liable to be mistaken for intussusception are: (i) *Henoch's purpura*, which incidentally may be a cause; (ii) *Acute enterocolitis*, which occasionally presents real difficulty; (iii) *Simple colic*; (iv) *Tuberculous mesenteric glands*, which may cause pain, diarrhoea, and a lump. They are all of more gradual onset and less acute course. If any doubt exists, the presence or absence of an intussusception can be definitely established by a barium enema and X-rays.

It should be strongly emphasised that the intussusception of infants is *not* primarily an obstruction, and that the true obstructive symptoms (absolute constipation, persistent vomiting, and distension) are late developments. The early picture is one of recurrent colic and mesenteric shock. Acute intussusception of *older children* and *adults*, on the other hand, behaves more like an ordinary obstruction, and the typical symptoms and signs of intussusception (e.g. bloody motions, shock, and lump) are usually absent.

#### TREATMENT

Everything depends on *early* and *speedy* operation; every hour before operation and every minute at operation diminish the chances of recovery. By devoting special attention to these points, Cluhbe (quoted by Newland, 18) was able to operate on 100 consecutive cases with a mortality of only 3 per cent.

Immediate laparotomy is indicated with very few exceptions (see below). Chloroform or ether anæsthesia are responsible for most fatal results in early cases and should *never* be employed. For several years I have used infiltration of the abdominal wall with  $\frac{1}{4}$  or  $\frac{1}{2}$  per cent novocaine, with *gas and oxygen* for the intra-abdominal manipulations; relaxation has proved perfect and the post-operative shock which so often occurred after general anæsthesia has been conspicuously absent.

*Technique of operation.* Every instrument, ligature and suture should be ready before commencing to operate. The infant is placed on a heated pad with its legs and chest amply covered. An area to the right of the navel is infiltrated with novocaine and five minutes later the administration of gas and oxygen is commenced. The value of an expert anæsthetist in this connection cannot be exaggerated.

With the child lightly under, a right paramedian incision is rapidly made opposite the navel, and long enough to admit *two fingers*. These are introduced and the intussusception felt for; usually it is found with great ease. The fingers are now made to meet beyond the apex and the "tumour" is gently "milked" back; in an early case the apex positively runs away from the milking fingers, and all except the last inch or two of the intussusception is reduced in a few seconds. In several cases I have been able to complete the reduction without withdrawing any intestine from the abdomen, but as a rule a little difficulty is encountered with the last few inches. This small segment should be brought out of the wound and inspected; usually it looks congested and feels œdematous. It should be gently compressed for a moment or two with a hot saline swab, and in nearly all cases it will then yield to a little persuasion in the form of a squeeze. I can see no objection to a little traction on the entering gut, provided it is *really gentle* and coincides with a "squeeze" on the apex.

After reduction, the gut is inspected and any injured or devitalised patch is segregated by a non-narrowing sero-muscular suture (this is rarely needed). The abdomen is then closed without waste of time, the gas and oxygen being stopped while this is done. The entire operation should not take more than 10 or 12 minutes, unless difficulty is encountered in the reduction. With good team-work and favourable conditions it can sometimes be completed in five minutes.

*Procedure in difficult cases.* Sooner or later, every surgeon will encounter an intussusception, the last few inches of which resist reduction. The urgent need for hurry makes such a case a serious

problem, which can only be solved by a previously planned procedure. If two minutes' squeezing with a hot pack fails to effect reduction, then either the apex is too swollen to come through, or adhesions of some firmness have formed between the entering and returning layers.

In either case, the simplest procedure is to insert a finger into the neck of the intussusception, between the entering and returning layers, and sweep it round in this space, thus breaking down adhesions and facilitating the reduction (Cope). If this fails, a rubber-covered sinus forceps is pushed into the neck as far as it will go, and the blades opened in several planes; this procedure serves the double purpose of dilating the neck and of breaking down recent adhesions. When neither of these methods succeed, the neck should be divided for an inch or more along its anti-mesenteric border, with a pair of scissors. This is almost bound to release the intussusception, and the cut, which is now straightened out, should be closed transversely to avoid narrowing of the lumen. I have only employed the last procedure once, and the forceps twice, but in all three cases a probably fatal resection was avoided.

It only remains to consider the rare cases in which sloughing of the intussusceptum has commenced, and the even rarer instances (in infants) when the above procedures have failed to obtain reduction. In small feeble infants probably the only chance of survival lies in leaving the intussusception alone (in the remote hope that it will slough and be passed), and suturing the margin of the neck to the entering layer to increase the protection afforded by the sheath. In older and stronger children a better chance is offered by resection, although the mortality of this is bound to be very high. Probably the best resection procedure in small children is *Maunsell's operation*, which imitates natural cure and can be performed quickly; it is, of course, only applicable when the sheath is healthy. The sheath and the entering layer are sewn together at the neck, a clamp is applied proximally, and the intussusceptum is brought out through a longitudinal incision in the sheath, and amputated by degrees, its two layers being sutured together as this is done. In adults and older children a complete resection with anastomosis is preferable.

*After-treatment.* After operation the child is returned to its cot and a radiant-heat cradle placed over it. Rectal or subcutaneous saline should be given if there is any evidence of shock, while a blood-transfusion may prove a life-saving measure in the worst cases. Breast feeding can be started as soon as the child is able to take it.

*Non-operative treatment.* Under exceptional circumstances one might be justified in resorting to conservative treatment (e.g. when expert surgical help is not available). An early intussusception can sometimes be completely reduced with an enema, run in under pressure, or by a rectal injection of air, again under pressure. I have seen more than one case in which the intussusception was reduced with a barium enema, but only to recur when the enema was run off.

In one case I deliberately refrained from operation.

The infant was three weeks old and weakly, with an intussusception of twelve hours. The doctor attending the case assured me that he had felt the lump in the left half of the transverse colon, but when I saw the infant, six hours later, the intussusceptum had moved back into the ascending colon. Moreover, colic and vomiting were subsiding, the infant was not shocked, and he had passed a normal and only slightly blood-stained motion an hour previously. I massaged the swelling down towards the cæcum, and distinctly felt it move some distance in this direction. We decided to wait for six hours, by which time all symptoms had disappeared, while the lump had diminished markedly and was now definitely in the ileo-cæcal region. By the next day the lump had gone, and the child made an uninterrupted recovery.

This was obviously a spontaneous reduction. It is probable that such an event is not very uncommon, but the occasions in which one can postpone operation in the hope of its occurrence must be very rare indeed.

### VOLVULUS

Twisting of the intestine mostly occurs round its mesenteric axis and, therefore, involves "free" portions of the gut. An isolated loop forms, shut off at both ends, and vascular changes result from compression of its mesentery. The vascular changes are aggravated by the rapid and often enormous distension of the rotated loop; the gut becomes extremely congested, and finally perforation or gangrene may supervene and lead to fatal peritonitis. In other cases the fatal result is accelerated by hæmorrhage into the engorged loop and peritoneal cavity.

Volvulus is practically always an extremely acute emergency, with a sudden onset, a rapid course, and a very high mortality (over 50 per cent). Although a rare disease, it is undoubtedly more common than was once thought; Vick collected 176 cases from 21 British hospitals

in five years (2·6 per cent of all obstructions). Several factors are concerned in its *etiology*. Among them are *congenital anomalies*, such as faulty rotation of the embryonic gut, a mobile cæcum, and megacolon; *acquired factors* also play a part, and include mesenteric and other adhesions, narrowing the base of attachment of a loop (e.g. the sigmoid), and sudden effort (such as straining at stool).

Volvulus occurs in three situations—the *sigmoid* (omega loop), the *cæcum*, and the *small gut*. It is usually stated that the sigmoid accounts for 75 per cent of cases (Miles, 25), and that the small intestine is the least common site. This is not the case nowadays; of Vick's 176 cases 85 occurred in the small intestine, 56 in the sigmoid, and 35 in the cæcum. It is probable that volvulus of the sigmoid is less frequent than it was, while the condition is becoming more common in the small intestine.

#### VOLVULUS OF THE SIGMOID

*Pathology.* Mostly seen in elderly people with chronic constipation volvulus of the sigmoid is predisposed to by—(i) elongation and loading of the omega loop, and (ii) narrowing of the already narrow attachment of the pelvic mesocolon, by inflammatory adhesions or bands. The immediate cause of the twist is often a strain (e.g. at stool). The upper limb of the loop as a rule comes down in front of the lower (i.e. anti-clockwise), twisting on its mesenteric axis from half to two turns. The twisted loop becomes rapidly and enormously distended, chiefly by unabsorbed carbon dioxide; also it soon becomes intensely congested and œdematous, and ultimately shows patches of gangrene. Hæmorrhage occurs into the wall, the lumen and the peritoneal cavity; sufficient blood may be lost to terminate life very quickly. Perforation may affect the isolated loop, or the distended intestine proximal to it.

*Clinical features.* The patient is usually elderly and gives a history of constipation; men are affected more often than women. The onset is sudden, with colicky pains and *complete obstruction*, soon followed by *enormous tympanitic distension*. Vomiting is not a prominent feature, but *tenesmus* is usual and distressing. The huge sigmoid rises as far as the diaphragm, pushes out the parietes, and may compress the thoracic viscera, causing respiratory embarrassment.

Diagnosis is confirmed by *inability to pass an enema*. The course is very acute and the patient may die in 24–48 hours.

*Treatment.* If a case is seen within a few hours of the onset, a long rectal tube should be inserted with the patient in the genu-pectoral position, and an attempt made to get into the loop and deflate it; this procedure may succeed in reducing the volvulus. If it fails, the abdomen must be opened by a long incision, and an attempt made to guide a rectal tube past the twist, which will then usually straighten out;

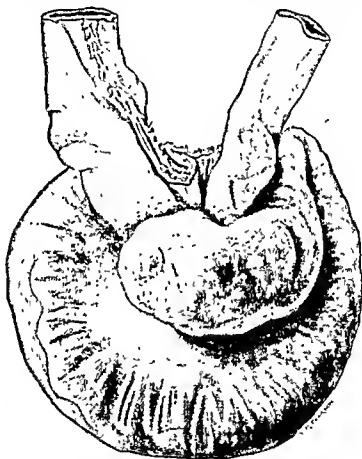


Fig. 551.—VOLVULUS OF SMALL INTESTINE.  
(From St. Mary's Hospital Museum.)

when this cannot be done, the distended loop must be brought out and methodically untwisted. In a difficult case the intestine should be deflated by puncture, which is closed with a purse-string suture, and then it will nearly always untwist. The loop is now washed with hot saline and watched; usually it recovers completely and can be returned safely, taking the precaution of passing a long rectal tube into the gut proximal to the volvulus. If the colon is gangrenous it should be resected, both ends being brought out and drained with Paul's tubes;



the anastomosis can be performed when the patient has recovered from the effects of the obstruction.

Recurrence of the volvulus is not uncommon, and should it occur the correct procedure is to excise the pelvic colon.

#### VOLVULUS OF THE SMALL INTESTINE

This is mostly seen in young and middle-aged people, and nearly always follows a previous pathological condition (e.g. tuberculous mesenteric glands, localised peritonitis, or abdominal operation). The usual cause is a local deformity or fixation of the mesentery by fibrous shrinkage and adhesions, the corresponding loop or loops rotating on their mesenteric axis. The lower portion of the ileum forms the site of election, but at times a large part of the small intestine and its mesentery may undergo rotation. The direction of the twist is generally clockwise.

The *morbid changes* resemble those of volvulus of the sigmoid, but since the obstruction is higher the clinical picture differs. *Diagnosis* is rarely made, unless the patient has had previous attacks of the same trouble (I have known one case on which four operations had to be performed for recurrent volvulus of the same loop). The clinical picture is one of acute low small-gut obstruction with strangulation. The œdematous and distended loop may be palpable through the abdominal wall, and there may be signs of free fluid. The condition is sometimes mistaken for an acute appendicitis.

The *treatment* does not differ from that of sigmoid volvulus, except that when gangrene is present or the mesentery cannot be untwisted, resection and immediate anastomosis should be performed in preference to exteriorisation and drainage of the volvulus.

The following case, recently under my care, provides a fairly typical picture of the lesion :

A young woman, aged 20, was admitted as an acute appendicitis of 12 hours duration. There was a history of several similar attacks in the past four years. The pain was umbilical and "gripping," vomiting was slight, but there was no passage of flatus. The patient seemed fairly well, there was tenderness and some rigidity over the right iliac fossa, while near the umbilicus the abdomen was distended and tympanic. On auscultation, there was marked increase of peristaltic sounds in the umbilical area. A diagnosis of low small-gut obstruction was made, and the abdomen opened by a right paramedian incision (under spinal anaesthesia). Two pints of blood-stained fluid escaped, and some three feet of low ileum were seen to be extremely œdematous, congested, and moderately distended. No cause for this could be found until the mesentery was examined, when one part of it was seen to be twisted through

almost a complete turn. It was readily untwisted, and proved to be shortened by fibrosis round a mass of tuberculous glands. The intestine rapidly lost its congestion and the contents passed on into the collapsed loops beyond. The abdomen was closed and the patient made an uninterrupted recovery.

Four weeks later she returned with a recurrence of the volvulus. This was reduced, but owing to the poor condition of the patient nothing further was done. It was decided to resect the affected segment a fortnight later, but two or three days before the proposed date she again became obstructed. At operation the affected segment was found to be twisted again and numerous recent adhesions were binding it to the scar. Four feet of intestine were resected and a primary anastomosis performed. The operation was followed by paralytic ileus, but this yielded to treatment and she ultimately made a complete recovery.

Some years ago Dott (26) drew attention to a *volvulus neonatorum*, in which the whole mesentery and small gut are involved; shortly after birth vomiting sets in and marked distension of the upper abdomen soon follows. Possibly, minor degrees of this condition exist in which years may pass before the obstruction becomes complete.

*Volvulus of the Cæcum.* This occurs in young and middle-aged people, and is usually attributable to congenital anomalies, particularly to the presence of a mobile cæcum and ascending colon (with a free mesocolon). Rotation again usually takes place round the mesocolic axis, but occasionally the cæcum bends back on itself, in front or at the side of the colon. The cæcum may distend to an enormous size, but otherwise the picture is one of low small-gut obstruction. Treatment is as for sigmoid volvulus, but reduction of the twist tends to be more difficult.

### MESENTERIC EMBOLISM AND THROMBOSIS

The frequency of this most fatal variety of acute obstruction is almost certainly under-estimated. Some years ago I wrote a thesis on the subject (27), and was able to collect 92 cases from two London hospitals.

*Pathology.* Mesenteric occlusion is either arterial or venous.

*Arterial occlusion* is in most cases by *embolism*, and only rarely by *primary thrombosis*. The source of the emboli is the heart in endocarditis, the lungs in pyæmia, or the arteries in atheroma and aneurysm. The effects of embolism vary with its level; with blockage of the main superior mesenteric trunk, the mesenteric circulation fails at once and

gangrene is inevitable in the small gut and right colon ; but when the embolus lodges in the lower part of the artery, or in one of the intestinal rami, the rich collateral circulation in the mesentery may suffice to keep the gut alive. Unfortunately, the vitality of the gut is ultimately endangered by a *secondary peripheral thrombosis*, starting from the embolus and spreading into the terminal mesenteric arcades, beyond which no anastomosis exists. Only a timely operation can put a stop to this secondary thrombosis, which is actually the essential morbid factor in most cases, and which, by a lateral spread, devitalises far more intestine than that supplied by the vessel originally blocked (see fig. 558).

*Venous occlusion* is always a *thrombosis*, and is secondary either to *portal obstruction* (fibrosis, obliteration, or external compression of the

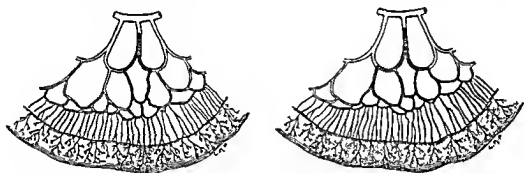


Fig. 558.—EXTENSION OF NECROSIS IN MESENTERIC ARTERIAL OCCLUSION OWING TO PERIPHERAL SPREAD OF SECONDARY THROMBOSIS.

portal vein), or to *peripheral sepsis* (septic thrombosis originating in appendicitis, infected piles, or pelvic inflammation). The effects of venous thrombosis are less severe than those of arterial occlusion, as the process is gradual and there is time for the abundant collateral circulation to open out.

The *morbid changes* in the intestine are classified into *anæmic gangrene* and *hæmorrhagic infarction*. The former is excessively rare, and is caused by occlusion of arteries previously narrowed by disease. Hæmorrhagic infarction is the usual result of mesenteric occlusion, whether arterial or venous in origin. The gut is first congested, then the distended capillaries burst and extravasation of blood occurs into the wall, the lumen, and the peritoneum ; finally, the gut becomes gangrenous. The occurrence of *infarction* is *not inevitable*, and its spread can be arrested by timely removal of the infarcted gut and thrombosed mesentery. Whether infarction occurs or not, paralytic ileus of the devascularised segment is unavoidable.

*Diagnosis.* Typically, the clinical picture is one of *sudden very acute obstruction with internal hæmorrhage*. There may or may not be evidence of the source of embolism or thrombosis (e.g. endocarditis, appendicitis, cirrhosis of the liver, pyæmia, etc.). The obstructive symptoms are sudden and severe, and there is *marked shock*. A falling temperature and rising pulse, pallor, early and rapid distension, the presence of *hamatemesis* or *melana* (in about 50 per cent of cases), and the discovery of free fluid in the peritoneum, combine with absolute obstruction to form a symptom-complex which should at least suggest mesenteric occlusion. The value of the "diagnostic enema," in cases without visible hæmorrhage, was pointed out by the writer (27); usually some of the blood in the infarcted segment is propelled into the large intestine before paralytic ileus sets in, and this can be washed out by a high enema.

*Treatment.* The only hope of saving life lies in immediate laparotomy; when possible, resection of the infarcted segment and thrombosed mesentery is performed, with a foot of gut proximally and distally (to get beyond the limits of the spreading thrombosis). Anything up to 15 feet can be resected successfully. The writer's series showed a total operation-mortality of 83 per cent, but of the cases in which resection was performed 50 per cent recovered.

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## CHAPTER II

### STRANGULATED HERNIA

#### INTRODUCTION

STRANGULATED external hernia is the commonest cause of intestinal obstruction and accounts for about half the hospital admissions under this heading (3267 of Vick's 6892 cases of acute obstruction (1)). Its frequency is explained by the high incidence of the lesion of which it is a complication.

A hernia may strangulate at any stage of its existence. It may do so on its first descent in a child or a young adult; more often many years elapse between its first appearance and strangulation, the bulk of the patients being middle-aged or elderly.

Its diagnosis is easier than that of any other abdominal emergency. A tense, tender, and irreducible swelling at a hernial orifice, associated with the general symptoms of obstruction, presents a clinical picture capable of only one interpretation. The treatment of an early case is equally simple, consisting in release of the constricting agent, return of the contents, and removal of the sac.

It follows that the mortality is lower than that of other obstructive lesions. Vick's 3267 cases show a total death-rate of 17·8 per cent, Frankau's (2) 1487 cases of 15·7 per cent, and the Friedrichshain Hospital (Berlin) (3) 1795 cases of 15·6 per cent. These figures compare favourably with the average mortality of internal obstructions (40 per cent), but what appears more remarkable is that a lesion so easy to diagnose and treat in its early stages should have a death-rate as high as 15 to 18 per cent.

An enquiry into the *causes* of this *mortality* at once shows that the principal factor is *delayed operation*, which usually also means late diagnosis. The part played by this delay is demonstrated very strikingly in the following table, compiled from Frankau's statistics.

TABLE I. Comparison of Duration and Mortality of Strangulated Hernia (based on 1487 cases collected by Frankau for the Association of Surgeons of Great Britain and Ireland).

Variety.	Duration of Strangulation.	Number of Cases.	Deaths.	Mortality.
Inguinal	0-24 hours	413	25	6 per cent.
	over 24 hours	161	48	30 " "
Femoral	0-24 hours	269	10	3.7 " "
	over 24 hours	320	66	20.6 " "
Umbilical	0-24 hours	59	14	23.7 " "
	over 24 hours	66	36	54.4 " "
Total	0-24 hours	741	49	6.6 " "
	over 24 hours	547	150	27.4 " "

A study of these figures shows that the mortality of inguinal and femoral strangulations is multiplied more than *five times* by delaying operation beyond the first twenty-four hours. One reason for this enormous increase is that in late cases we are confronted with the local problem of devitalised or actually gangrenous intestine, resection of which very greatly increases the operation death-rate. Thus, in Frankau's collection resection was done 105 times with 45 deaths (42.8 per cent), while in the Friedrichshain cases 286 resections were followed by 144 deaths (50.4 per cent). But even when resection is unnecessary the mortality of late cases is much higher than that of early ones. It must be remembered that strangulated hernia is an intestinal obstruction, and that anatomical and physiological changes of a cumulative nature occur, which increase the morbidity of the lesion almost hourly (see article on intestinal obstruction, page 979). By applying ourselves assiduously to the prevention and treatment of these death-causing processes (dehydration, plasma-depletion, renal failure, intestinal distension and paralysis, shock, loss of blood, toxic absorption, etc.) we can do a great deal towards lowering the mortality.

The overwhelming importance of the *time-factor* should not blind us to the significance of other factors bearing on the *prognosis* of strangulated hernia. Among these are: (i) the age and constitution of the patient; (ii) the type, size, contents, and location of the hernia; (iii) the length of intestine involved; (iv) the intensity of the constriction; (v) the preparation and post-operative treatment; (vi) the anaesthetic; (vii) the actual operative procedure; (viii) the incidence of post-operative complications.

## PATHOLOGY

*Causation.* Occasionally strangulation occurs at the *first descent* of a hernia into a pre-formed congenital sac, the immediate cause being a sudden severe strain. Much more frequently, strangulation is a complication of a *previously existing* and reducible hernia, and is produced by one of the following causes :

(a) *Descent of additional contents.* This may be sudden and result from excessive exertion (e.g. lifting or pushing heavy objects), or more gradual and occasioned by repeated minor strains (coughing, dysuria, or constipation). In either case a point is reached at which the sac and hernial canal can no longer accommodate the contents without pressure. Rarely, a sudden strain causes strangulation by producing a volvulus of the loop in the sac, or of a coil proximal to it (McIver, 4).

(b) *Rapid increase of contents.* The intestine may become distended from a temporary obstruction of its lumen (e.g. incarcerated hernia), or the omentum may swell from inflammation.

(c) *Gradual increase of contents.* This includes hypertrophy of the intestine or omentum, fecal impaction in large-gut hernia, and the growth of tumours in the sac.

(d) *External violence.* Clumsy attempts at taxis may cause a "*reduction en masse*" (see page 1047), or produce an inflammatory œdema of the sac or contents.

*Incidence.* The incidence of strangulation among the various types of hernia is difficult to determine, for whilst all strangulated cases come to us for treatment, an unknown proportion of simple herniæ never seek medical aid. According to McIver (4) strangulation occurs in 2-4 per cent of inguinal, 25-30 per cent of femoral, 15-20 per cent of umbilical, and 3-5 per cent of incisional herniæ.

The frequency of strangulation in *femoral hernia* is, of course, explained by the small size and unyielding nature of the crural canal, and by the rigid edge of Gimbernat's ligament. Similarly, *umbilical herniæ* are susceptible because of the dense fibrous tissue round the neck of the sac, and of the enormous hypertrophy of the contained omentum. The relative freedom from strangulation of *inguinal* and *incisional* herniæ is due to the larger size of the neck, and the more yielding character of its surroundings.



Nevertheless, owing to the enormous preponderance of inguinal hernia over the other types, the chances are that of any 100 cases of external strangulation, about 50 will be inguinal, 40 femoral, and 10 umbilical, incisional, and other rare varieties.

The *age-incidence* varies with the type of hernia. Inguinal strangulation is common in infancy, in middle life, and in old age. Strangulated femoral hernia is rare in young people, becomes more common with increasing age, and reaches its highest incidence after 60. Umbilical strangulation is almost limited to people between 40 and 70.

The *sex-incidence* also varies with the type. Frankau's statistics show that strangulated inguinal hernia is eight times more common in men than in women, but that femoral and umbilical strangulations affect women more often than men, in the ratios of four to one, and six to one, respectively.

#### MORBID ANATOMY

(1) *Definition.* A hernia becomes strangulated when the blood supply of its contents is arrested by constriction of the supplying vessels. Intestinal obstruction is not an essential feature, and indeed is occasionally absent, e.g. in epiplocele, Richter's hernia, or the strangulation of a diverticulum. Nevertheless, complete obstruction accompanies a large majority of strangulated herniæ. The obstruction is mostly mechanical, the strangulated coil being occluded by the constricting agent, but sometimes the ileus is paralytic, and due to reflex stimulation of the sympathetic (e.g. strangulation of an epiplocele or a diverticulum).

(2) *Contents.* The *small intestine* is present, alone or with omentum, in 85 per cent of inguinal, 80 per cent of femoral, and 60 per cent of umbilical strangulations. The *omentum* often accompanies small intestine, but occurs alone (epiplocele) in 17 per cent of femoral, and about 8 per cent of inguinal and umbilical strangulations. The *large gut* is a rare content of inguinal (6 per cent) or femoral (2 per cent) cases, but it is often present in umbilical herniæ (30 per cent). Still rarer contents are the bladder, appendix, Meckel's diverticulum (Littre's hernia), appendix epiploica, and pelvic organs.

(3) *Constricting agent.* The site of strangulation and the constricting mechanism vary with the type of hernia. The worst constriction and the most rapid strangulation are seen in *femoral hernia*, where the narrow crural canal, with the unyielding "deep crural arch"

anteriorly, and Gimbernat's ligament medially, constitute an ideal strangulating mechanism (fig. 559). Another contributory factor is the sharp upper edge of the saphenous opening (Hey's ligament), which fixes the hernia as this turns up over it, and so allows the knife-like edge of Gimbernat's ligament to press against the herniated gut.

Strangulation of *inguinal hernia* is much less acute, the agents of constriction being more yielding in character. In small boys and elderly men the constricting mechanism is sometimes provided by the pillars of the external abdominal ring; but a more usual agent is the

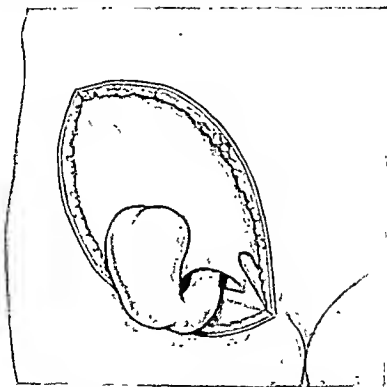


Fig. 559.—ANATOMY OF FEMORAL HERNIA.

condensed fibrous tissue round the neck of the sac and near the internal ring (fig. 560). Rarely the cause of strangulation is a band within the sac.

The constricting agent in *umbilical hernia* is the extremely tough ring of fibrous tissue which forms round the actual neck of the sac. The almost cartilaginous consistency of this ring is responsible for the high incidence and severity of strangulation.

(4) *Morbid Changes.* The process of strangulation of a coil of intestine comprises three morbid stages. Although the transition between them is gradual, these stages are discernible from one another

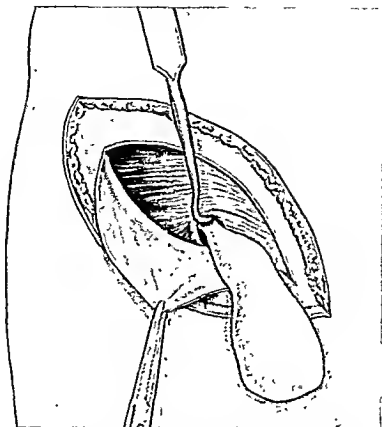


Fig. 560.—ANATOMY OF INGUINAL HERNIA SHOWING CONSTRICTION AT INTERNAL RING.

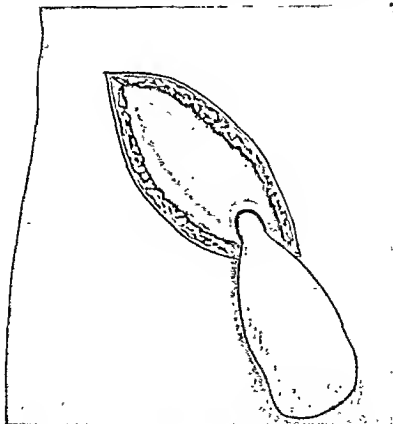


Fig. 561.—ANATOMY OF INGUINAL HERNIA SHOWING CONSTRICTION AT EXTERNAL RING.

at operation, and afford essential guidance in the treatment and prognosis of the lesion.

*Stage 1.* The intestine becomes intensely congested owing to obliteration of its veins. It is swollen and œdematous, of a dusky red, purple or claret colour, but it retains its smooth and shiny appearance, is still elastic in consistency, and is capable of peristalsis. *The vessels in its mesentery still pulsate.* Progressive distension of the coil occurs

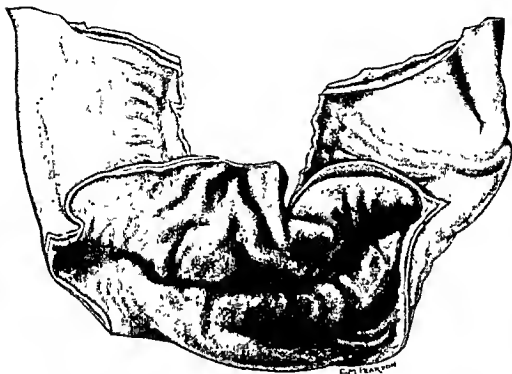
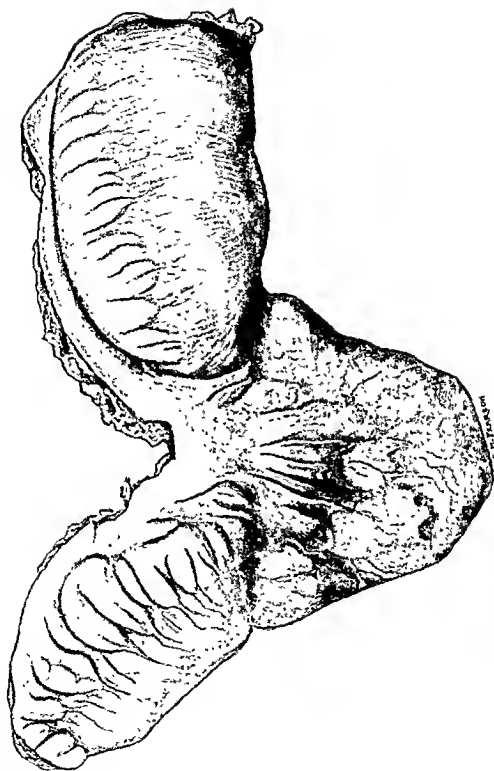


Fig. 562.—INTESTINE FROM FATAL CASE OF STRANGULATED HERNIA, SHOWING HÆMORRHAGIC INFARCTION.  
(S. Mary's Hospital Museum.)

because the gases formed in it cannot be absorbed. The sac fills with fluid exuding from the engorged contents; at first serous, the fluid soon becomes blood stained.

This is the stage of *viable gut*.

*Stage 2.* The essential change of the second stage is *hæmorrhagic infarction* of the strangulated coil (fig. 562). The distended venules and capillaries of the engorged intestine rupture and the extravasated blood infiltrates the gut-wall, at the same time escaping into the lumen and sac. The gut is now of a black, dark grey or chocolate colour, loses its shine and elasticity, and is incapable of peristalsis. The fluid in the sac becomes definitely hæmorrhagic and may be faecal in odour.



F. 503. — RESECTED INTESTINE FROM CASE OF STRAGULATED HERNIA SHOWING ACTUAL CANCER.  
(St. Mary's Hospital Museum.)

By this time the arteries are obliterated as well as the veins, and so the vessels in the mesentery *no longer pulsate*.

This is the stage of *non-viable gut*.

*Stage 3.* The devitalised gut now becomes *gangrenous*. Necrosis tends to start at the sites of constriction, or at the anti-mesenteric

border of the apex of the loop (fig. 564), at which points perforation is likely to occur. Later, the necrotic process may spread to the whole loop (fig 563).

Perforation of the gangrenous coil is followed by the formation of an abscess in the hernial sac. Sooner or later this bursts, and the resulting faecal fistula occasionally leads to a natural cure of the disease.

Not infrequently the loop as a whole is viable, but linear patches of pressure necrosis (næmic ulceration) occur at the points of constriction. Unless the loop is pulled down sufficiently, these localised lesions will be overlooked, with the

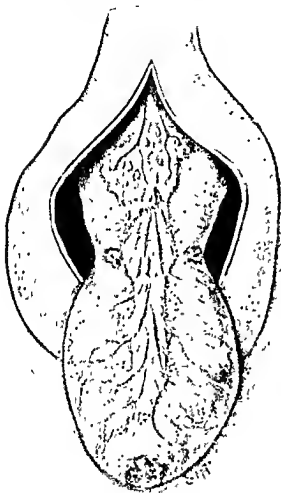


Fig. 564.—STRANGULATED FEMORAL HERNIA, SHOWING SITES OF COMMENCING NECROSIS.

result that perforation (with peritonitis), or fibrous stricture, may develop as early or remote sequelæ.

The intestine proximal and distal to the strangulated segment shows the changes of any acute obstruction. These have been discussed in sufficient detail already.

(5) *Unusual Types of Strangulated Hernia.* To this group belong : (i) epiplocele, (ii) Richter's and similar herniæ, (iii) Maydl's hernia.

Strangulated omentum (*epiplocele*) rarely becomes gangrenous, as it receives an adequate collateral blood supply through adhesions which connect it to the sac. On the other hand, diagnosis may be made difficult by the absence of obstructive symptoms, and this may occasion dangerous delay.

*Richter's hernia* (partial enterocoele) is a particularly serious type of strangulation (fig. 565), and occurs in more than 10 per cent of strangulated femoral herniæ (Frankau). The hernial protrusion is so small that it is likely to be missed, especially in obese women; moreover, the obstruction is partial and the symptoms are often inconclusive. On the other hand, the herniated portion of the gut-wall is subjected to very severe constriction by a tight crural ring, with the result that gangrene and perforation occur early. It is, therefore, not surprising that two-thirds of the cases remain undiagnosed until after the second day, and that the mortality is very high. Indeed, many of them are treated by exploratory laparotomy for an undiagnosed acute abdomen.



Fig 565.—RICHTER'S HERNIA WITH PERFORATION.  
(St. Mary's Hospital Museum.)

*Maydl's hernia* (retrograde strangulation) is a hernia of W formation, with two loops in the sac, and an intervening loop in the abdomen; the latter becomes strangulated owing to kinking or compression of its mesentery (fig. 566). The diagnosis is fraught with obvious difficulties, but the condition is fortunately very rare.

#### MORBID PHYSIOLOGY OF STRANGULATED HERNIA

The physiological disturbances in strangulated hernia are in the main similar to those of internal obstruction, but they are not

subject to quite the same range of variation. Dehydration, plasma depletion, and renal failure, play a part in many fatal cases, but since the segment to be involved is the lower ileum, these disturbances cannot be accepted as the chief cause of death. Distension of the intestine proximal to the strangulation is another contributory morbid factor, as it may lead to the absorption of toxic proteoses and amino-acids at the "patches" of mucosa damaged by *distension-necrosis* (Holt) (5), or to perforation of these patches and *peritonitis*.

It must be noted that the bulk of the patients are *elderly*, and that many of them are suffering from *chronic disease*, e.g. bronchitis, which may be aggravated by the abdominal emergency, or by the operation which it calls for. Furthermore, *broncho-pneumonia*, *acid digestion*, and other pulmonary lesions may develop as complications, either following the inhalation of vomit, or from the employment of chloroform and ether as anæsthetics.

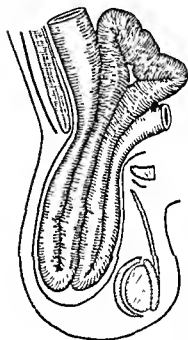


Fig 266. - MAYDL'S HERNIA.

Although any of the above factors may play a part in the causation of death, the most important lethal disturbances are undoubtedly *local*. When the onset of strangulation is sudden, as is usually the case, the first disturbance is *shock*, produced by trauma to the mesentery and intestine. In large herniæ, with two feet or more of intestine strangulated, sufficient blood may be lost by engorgement and extravasation to cause a rapid and fatal

exacerbation of the shock. When smaller loops are involved the fluid which exudes from the engorged intestine becomes intensely *toxic* (Foster and Hausler, 6), and enough of it may be absorbed by the sac (which receives its blood supply from the coverings) to cause a fatal toxæmia. These toxins are probably proteoses or amino-acids produced by the action of bacteria on the devitalised or gangrenous gut. The toxic contents of the loop itself cannot be absorbed while it remains intact, because the veins draining it are obliterated by the constriction; but after gangrene or perforation of the loop has occurred, there is nothing to prevent the absorption of toxins by the sac.

Finally, perforation of a necrotic patch at the actual site of constriction



may lead to escape of highly septic contents into the peritoneal cavity, a rapidly fatal peritonitis resulting. This is particularly likely to occur in a Richter's hernia.

### DIAGNOSIS

Diagnosis is made easy by the presence of a swelling with characteristic features, and over a hernial orifice, in a patient who has symptoms of acute obstruction.

The *obstructive symptoms* are generally those of a low small-gut blockage, e.g. intermittent colicky pains, persistent vomiting, and absolute constipation. Initial *shock* is common and the onset usually *sudden*. Abdominal distension is a late sign and must not be waited for; this also applies to collapse, low blood-pressure, dehydration, and toxæmia. In neglected cases all these symptoms will be present, and also associated complications, of which oliguria and a high blood urea are perhaps the most striking.

The diagnostic abdominal sign is *increased peristalsis*, which can be easily discovered on auscultation. In late cases the absence of this sign is of serious significance, since it means that intestinal paralysis *has set in*.

The *local swelling* is directly over a hernial orifice, and there is usually a history of previous reducibility. With the development of strangulation, the hernia becomes *irreducible, tense, and tender*; above all, it *loses its impulse* on coughing. When gangrene has ensued, the swelling becomes less tense and less tender, but it does not regain its impulse. Provided the patient lives long enough, the skin over the hernia takes on a red and oedematous appearance, and ultimately sloughs, giving rise to a fæcal fistula.

The occurrence of strangulation is sometimes the culmination of previous "threatening attacks" from which the patient has recovered spontaneously. These previous occurrences are, of course, not true strangulations, but represent attacks of irreducibility, inflammation, or incarceration of the hernia, and as such are of importance in the differential diagnosis.

While the diagnosis of a typical strangulated hernia is usually a very simple matter, atypical varieties, particularly *epiplocele* and *Richter's hernia*, are often extremely difficult to diagnose. The local signs may be absent or negligible, and the symptoms of obstruction may be inconclusive. It is only by being mindful of the frequency of these conditions, and by early operation in doubtful cases, that we can protect the patient against the grave dangers of delay. There are two

signs which I have found useful in such cases: one is *increased peristalsis* on abdominal auscultation, and the other is *tenderness* of a hernial aperture (usually the femoral canal). The second sign is particularly valuable, since it may be the only local evidence obtainable of a strangulated femoral hernia in an *obese* woman.

*Differential Diagnosis.* A *strangulated femoral hernia* is usually quite small, and in stout people it may be discovered only after careful palpation. Its position to the outer side of the pubic spine differentiates it from the inguinal variety. The only condition likely to be mistaken for it is an *adenitis* of the femoral glands; apart from the absence of obstructive symptoms, the lobulated and more diffuse character of the glandular swelling, with peri-glandular thickening and redness and œdema of the overlying skin, should, however, prevent errors in diagnosis. A strangulated hernia is usually much more tense and globular.

*Strangulated inguinal hernia* is generally large, occupying and distending the scrotum. The swelling is painful and any attempt at reduction is resisted. It must be differentiated from irreducible hernia, inflamed hernia, and incarcerated hernia. A simple *irreducible hernia* is painless and non-tender, and can be easily distinguished from a strangulation. An *inflamed hernia* presents greater difficulty in diagnosis, since it is both tender and irreducible; but it feels hot to the touch, the skin over it may be red and œdematous, and there are no symptoms of obstruction. An *incarcerated hernia* produces symptoms of chronic rather than acute obstruction, while the swelling is neither tense nor particularly tender; instead, it is heavy and doughy to the feel, as it consists of large gut obstructed with feces. It occurs in elderly men who suffer from constipation.

It should be remembered that irreducibility, inflammation, or incarceration of a hernia are predisposing causes of strangulation. Such cases should, therefore, be carefully watched, operation being advisable at the first reasonable opportunity.

A *strangulated umbilical hernia* is usually very large, and occurs in obese and "chesty" women between 40 and 70. The diagnosis is simple in the extreme, but the treatment is fraught with difficulties. Among these are the huge size and massive contents of the hernia, the smallness of the aperture, the existence of extensive adhesions between the contents and the sac, and the frequent presence of sacculi. The special dangers attending operation are shock, paralytic ileus, and chest complications.

TREATMENT

Every case of strangulated hernia demands *operation* without delay. The chance of a natural cure by the formation of a fæcal fistula is too slender to be taken into account, and the only alternative is the extremely dangerous one of reduction by taxis. In these days the operation of *herniotomy*, performed by a skilled surgeon under local or spinal anæsthesia, can be undertaken safely even in the presence of advanced constitutional disease, and in very aged patients, provided the intestine is still healthy.

The *prognosis* depends mainly on the duration of the strangulation before operation. Thus, Frankau's cases show the following mortalities :

	1st day.	2nd day.	3rd day.	4th day.	5th day.
Inguinal strangulations . .	6%	15%	28%	40%	36%
Femoral strangulations . .	3.7%	18%	23%	16%	27%

*Taxis*. This is justified only when the patient obstinately refuses operation, and on the rare occasions when surgical help is not available. Even in these circumstances, certain *conditions* must be observed if taxis is to have the remotest chance of success. They are : (i) the strangulation must be not more than six hours old ; (ii) the hernia must have been previously reducible ; (iii) the hernia should be an inguinal one ; (iv) the manipulations must be carried out gently and on a relaxed patient.

The *dangers of forcible taxis* are so well known that it is not necessary to do more than indicate them in the form of a list (*a*, *b*, and *c* are types of "*reduction en masse*" ) :

(*a*) The entire sac and contents may be displaced into a position between the layers of the abdominal wall (fig. 567).

(*b*) The sac may be ruptured, and the contents only forced into the scrotum or abdominal wall.

(*c*) The sac may remain *in situ*, but the contents are forced into a diverticulum (fig. 568), through a hole in the omentum (fig. 569), or across a "bridle" adhesion at the neck (fig. 570).

(*d*) The gut may be ruptured or seriously bruised (fig. 571), the reduction being followed by peritonitis.

(e) The gut may be reduced, but on its return into the abdomen it may rotate on its mesenteric axis, and so produce a volvulus (rare).

(f) The gut may be non-viable, and its return to the abdomen will be followed by perforation and peritonitis (fig. 572).



Fig. 567.—REDUCTION EN MASSE, TYPE I.



Fig. 568.—REDUCTION EN MASSE, TYPE II.



Fig. 569.—REDUCTION EN MASSE, TYPE III.



Fig. 570.—REDUCTION EN MASSE, TYPE IV.



Fig. 571.—GUT RUPTURED BY FORCIBLE TAXIS.



Fig. 572.—REDUCTION OF NON-VIABLE INTESTINE.

"*Reduction en masse*" is obviously an extremely dangerous complication, as it tends to suggest a cure when it really is an aggravation of the lesion. It should be suspected if an apparent reduction is followed by pain, shock, and persistence of the obstructive symptoms. The only chance left to the patient lies in immediate laparotomy, but the mortality is bound to be very high.

In view of these dangers, and of the slender prospect of success, the

procedure of taxis should be completely abandoned, and our energies devoted to the far more promising outlook of *early operation*, coupled with measures directed to the associated obstruction.

#### OPERATIVE TREATMENT

This comprises (i) preparation, (ii) choice of anæsthetic, (iii) the operation itself, (iv) post-operative treatment.

*Preparation.* Although the need for operation is urgent, there is always time for pre-operative measures which will improve the chances of recovery. For at least an hour after admission to hospital or nursing home, the patient should rest in a warm bed, under the quietening influence of a small dose of morphia. In a really *early* case (within 12 hours of strangulation) nothing further need be done, apart, of course, from the skin preparation.

In cases seen after the first 12 hours, certain pre-operative measures are indicated, which are almost as important as the operation itself. They are :

(i) *Gastric or duodenal drainage.* A duodenal catheter is passed by the oral or nasal route, and employed for siphoning off or aspirating the contents of the stomach and upper intestine (see page 998). This is of the utmost value, since it prevents air-swallowing and inhalation of vomit, and at the same time decompresses the distended intestine above the strangulation, thus maintaining its tone and blood supply, and avoiding paralytic ileus and distension-necrosis.

(ii) *Anti-shock measures.* Heat and morphia come under this heading, but the most useful remedy we possess is *intravenous glucose-saline*. About a litre of this should be run in slowly, but in a dehydrated or very toxic patient as much as two litres may be given before operation.

(iii) *Blood-transfusion.* With *large herniæ*, in which loss of blood is an important morbid factor (see page 1044), a *blood-transfusion* is likely to make all the difference between recovery and death. It not only replaces the blood lost, but also effectively combats shock, and protects the patient against the dangers of an operation which may prove long and precarious.

Although *anti-Welchii serum* has been used for some years, the claims that it lowers mortality have not been confirmed. Indeed,

recent work, both experimental and clinical, suggests that it is practically valueless.

Under no circumstances should an *aperient* be given before operation, and we do not favour pre-operative *enemata*.

*The Anæsthetic.* The results of *local anæsthesia* are so satisfactory, and the method so safe, that we consider it should be used in every operation for strangulated hernia. In the few cases in which local infiltration proves inadequate (e.g. large scrotal or umbilical herniæ), and in nervous patients, *gas and oxygen* may be administered for the more difficult part of the operation. *Chloroform* and *ether* are dangerous anæsthetics for the type of patients who mostly suffer from strangulated hernia, and moreover exert an inhibitory effect on peristalsis which may have serious consequences. *Spinal anæsthesia* is usually very effective, and is free from most of the disadvantages of chloroform and ether, but it should not be employed in the presence of severe shock or a low blood-pressure.

*Technique of local anæsthesia for herniotomy.* One per cent novocaine is the standard solution, but  $\frac{1}{2}$  per cent should be used in stout patients, as a larger quantity will be needed. The limit of safety in an adult is 100 cc. of 1 per cent, and 250 cc. of  $\frac{1}{2}$  per cent, but the full amount is rarely required. Care must be taken not to inject the solution into a large vessel.

In *inguinal* and *femoral* strangulations it is first necessary to block the inguinal and iliohypogastric nerves. This is done by injecting 10 cc. of 1 per cent novocaine through a wheal a little medially to the anterior superior spine (fig. 573). The injection is started when the needle is felt to pierce the external oblique aponeurosis, and continued for  $\frac{1}{2}$  to  $\frac{3}{4}$  inch more deeply.

For *inguinal hernia* (fig. 573) two wheals are then made at the borders of the external ring, and from them the subcutaneous tissues and cord are infiltrated in lines running down into the scrotum and up to the first wheal. The coverings and sac are infiltrated in layers after making the skin incision, while the intestine is anaesthetised by injecting the mesentery both before and after pulling the loop down. The omentum can be infiltrated by injecting it as high as possible.

For *femoral hernia* (fig. 574), wheals are raised over the neck and lower part of the swelling, and connected by subcutaneous infiltration to each other, and to the wheal for the inguinal and iliohypogastric nerve-block (see above). The sac and coverings are infiltrated after

making the skin incision, while the neck of the sac and other constricting agents are injected from above, after opening the inguinal canal (it is assumed that Lotheissen's inguinal route is used).

For *umbilical strangulation*, weak solutions should be employed ( $\frac{1}{4}$  to  $\frac{1}{2}$  per cent), a large quantity of fluid being required. Four wheals are made round the hernia and connected by intradermic lines of infiltration. From these lines, deep injections are made into the abdominal wall, with the object of obtaining a "pyramidal" block of

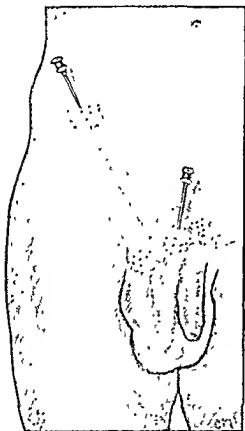


Fig. 573.—SHOWING TECHNIQUE OF LOCAL ANÆSTHESIA FOR INGUINAL HERNIA.

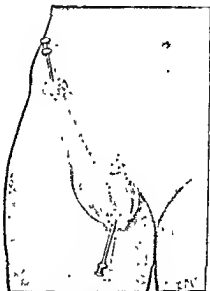


Fig. 574.—TECHNIQUE OF LOCAL ANÆSTHESIA FOR FEMORAL HERNIA.

anæsthesia. After making the incision, novocaine is injected into the rectus sheath, and through this into the extra-peritoneal fat, all round the neck of the sac; this step is very important and should be carried out with great thoroughness. Further infiltration of the peritoneum may be necessary after the neck of the sac has been opened. The contents are anæsthetised by injecting the mesentery and omentum, as they emerge at the neck.

*The Operation.* Details vary with the type of hernia, hut the general principles are the same in all types. They are best dealt with as steps of the actual operation.

(i) The incision is made over the hernial swelling, and should be large enough to provide easy access.

(ii) Everything is divided down to the actual sac, which is separated from its coverings if time permits.

(iii) The sac is opened at the fundus with the flat of the blade, to avoid injury of the presenting distended intestine. That the sac has been opened is shown by the free escape of fluid and by the glistening appearance of its endothelial lining.

(iv) The escaping fluid is examined. When this is hæmorrhagic or foul-smelling, great care must be exercised in handling the intestine, which is likely to be very friable.

(v) The sac is then incised up to the neck and the contents inspected. No decision is arrived at until after the constriction has been released.

(vi) The constriction is now released by division or stretching of the constricting agents. This release must be adequate enough to permit the free withdrawal of the strangulated coil.

(vii) The strangulated loop and the intestine above it are then pulled down very gently, and examined very carefully, special attention being given to the site of constriction. If any doubt exists, the loop is covered with a hot saline cloth, and examined again after a few minutes.

(viii) Now comes the crucial part of the operation—we have to decide whether the intestine is *viable*, *doubtful*, or *non-viable*.

*Viable* intestine regains its colour evenly throughout its entire length, the vessels in its mesentery pulsate, its surface is smooth and glossy, and there are no patches of anæmia or necrosis at the sites of constriction. The intestine is *non-viable* when the abnormal colour (black, grey, or chocolate) persists after releasing the constriction, when definite areas of necrosis are observed anywhere in the loop, when the vessels no longer pulsate, and the intestine is dull, œdematous, and inelastic (like “wet blotting-paper”). The intestine is *doubtful* when the colour returns slowly and unevenly, when it has lost its normal shine, and when the constriction-rings remain outlined on its surface, or show small linear patches of anæmic ulceration.

(ix) If the intestine is definitely *viable*, the loop is returned into the abdomen, any omentum is transfixed, ligatured, and removed, and the sac is excised. When the patient's condition permits, the operation is completed by a radical cure of the hernia.



The treatment of *non-viable* and *doubtful* intestine is dealt with at the end of this article. In late cases with a viable loop, but with gross distension of the intestine above it, drainage of the latter by *enterostomy* (through a separate incision) may prove to be a life-saving measure.

*After-treatment.* The complications to be feared most are pulmonary collapse, pneumonia, paralytic ileus, and peritonitis.

*Pulmonary collapse* and *pneumonia* may be avoided by nursing the patient in a sitting position, and by encouraging deep breathing and coughing. Expectorants are of some service for this purpose, but much better results are obtained by the inhalation of a 5 or 10 per cent mixture of carbon dioxide with oxygen or air, for ten minutes every hour or two.

For the prevention and treatment of *paralytic ileus* the reader is referred to the article on this subject. *Peritonitis* is mostly caused by the return of doubtful gut or by perforation of the distended intestine proximal to the strangulated loop; the only hope of saving life when peritonitis has developed lies in immediate laparotomy, dealing with the cause, and instituting drainage.

In the *ordinary post-operative case* the duodenal catheter should be left in for the first day or two, and oral feeding should be started only when nothing further can be aspirated through it. In the meantime *subcutaneous* and *intravenous fluids* are continued. Except when a resection and anastomosis has been performed, peristalsis may be encouraged after the first 24 hours with small doses of morphine (see page 1087), or with ox-bile and other enemata. If the return of peristalsis is sluggish, more powerful intestinal stimulants should be given, e.g. 500 cc. of 10 per cent *hypertonic saline* administered slowly by the intravenous route, an enema being given after an hour's interval. *Acetyl-choline* (see page 1088) may be employed as an alternative. A *rectal tube* should be inserted to avoid meteorism and gas pains, while a heated pad to the abdomen is useful for the same purpose.

When a *primary resection and anastomosis* has been performed peristaltic stimulants should be avoided for the first 48 hours, reliance being placed on duodenal drainage, intravenous and subcutaneous fluids, and a flatus tube.

If an *enterostomy* has been established, great care is needed to avoid blocking of the catheter. When drainage is sluggish, it may be stimulated by injecting hypertonic saline intravenously or locally into the intestine.

*Retention of urine* is a complication which not infrequently follows herniotomy; while it lasts the bladder should be emptied by catheter, at least six-hourly. A remote sequela which should be borne in mind in the after-course of a strangulated hernia is *fibrous stricture* of the intestine; this may occur either at the site of constriction or at the anastomosis (when a resection has been necessary).

#### OPERATIVE TECHNIQUE IN THE VARIOUS TYPES OF HERNIA

(1) *Strangulated Inguinal Hernia.* A free incision is made in the long axis of the tumour, starting a little above the internal ring (fig. 575). The inguinal canal is opened widely and the coverings of the sac are incised. The sac is identified and opened. The finger is inserted up the canal, either outside or inside the sac, the site of constriction discovered, and an attempt made to stretch the constricting agent. A nick with a scalpel (guided along the finger) in a direction upwards and slightly inwards (to avoid the deep epigastric vessels) may be required to start with, after which the finger will do all that is necessary. The return of a viable loop may be made difficult by distension, or because a second constriction is present higher up. Therefore, before returning the intestine, make certain that there is no obstacle to reduction, relax the canal by flexing the thigh, and deflate the intestine by squeezing it gently with a saline swab. Other obstacles to reduction which may have to be dealt with are hour-glass contraction of the sac, an interstitial sacculus, or an undescended testis.

(2) *Strangulated Femoral Hernia.* The inguinal (Lotheissen's) route should be chosen, for four reasons: (a) it facilitates division or rupture of the constricting structures; (b) it avoids injury to the accessory obturator artery, which is easily seen; (c) when resection is required, it can be performed with greater ease and safety; (d) a radical cure can be done with greater certainty of success.

The incision is oblique, starting  $1\frac{1}{2}$  inches above Poupart's ligament, and running down and in over the swelling (fig. 576). The coverings are incised, and any constricting fibres at the saphenous opening (e.g. Hey's ligament) stretched or divided. The sac is opened near the fundus and the contents are inspected. The external oblique aponeurosis is then slit up for a short distance from the external ring, and the arching fibres of the internal oblique and transversalis are identified. The crural ring is now exposed from above, and enlarged by stretching with the finger, assisted by division of Gimbernat's ligament and of the

fibrous band in front of the ring. The neck of the sac is then opened above Poupart's ligament, and an attempt made to push the contents up into the inguinal part of the wound. Great care must be exercised at this stage to avoid injury to the distended and friable intestine, and if the ring is not roomy enough to ensure its safe transmission, Poupart's ligament should be divided. After reduction of the intestine,

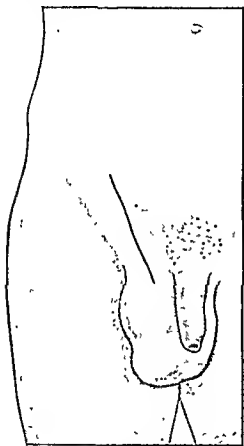


Fig. 575.—INCISION FOR STRANGULATED INGUINAL HERNIA.

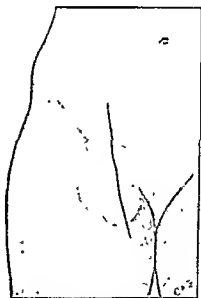


Fig. 576.—INCISION FOR STRANGULATED FEMORAL HERNIA.

the cut ends of the ligament are sutured, the sac ligated and excised, and a radical cure effected by fixing the conjoint tendon to Cooper's ligament, and by overlapping the external oblique aponeurosis.

(3) *Strangulated Umbilical Hernia.* It is best to commence the incision round the lower part of the neck, where the coverings are thicker and the contents least likely to be adherent to the sac. The ring, the neck of the sac, and the adjacent aponeurosis must be exposed thoroughly. The neck is opened and the sac slit up towards the fundus. The constriction is nicked laterally, and the ring well stretched with the fingers. The contents are now examined, and adherent intestine is

separated from the sac and omentum, and reduced. The omentum is ligatured off at the neck and divided there, its adhesions to the sac being left undisturbed. The sac (with adhering omentum) and the redundant skin are now removed together, by a curved incision passing over the upper aspect of the neck and joining the previous incision round its lower aspect. The operation is completed by performing one of the well-known methods of radical cure.

#### THE TREATMENT OF NON-VIABLE AND DOUBTFUL INTESTINE

(1) *Doubtful Intestine.* No hard and fast rules govern the treatment of *doubtful intestine*, and a great deal will depend on the condition of the patient, the experience of the surgeon, and the actual degree of damage to the gut. A small patch of necrosis at the site of constriction can be safely segregated (invaginated with Lembert sutures), provided the lumen is not narrowed by this procedure and the rest of the loop is quite healthy. But when the constricted portion is severely damaged, or the recovery of the strangulated loop is in question, undoubtedly the safest course is to deal with it as if it were actually non-viable, i.e. by resection. However, with an old and feeble patient, and with the chances in favour of recovery of the loop, the surgeon may return the loop into the abdomen, just within the hernial orifice, and insert a drain to it, as a safeguard against subsequent perforation. This is certainly a sensible move for the inexperienced or occasional operator. As an alternative, he may leave the doubtful loop in the wound, and await subsequent developments. In either case, an *enterostomy* should be performed through a separate incision, if the degree of obstruction is such as to indicate immediate decompression.

(2) *Non-viable Intestine.* Strangulated *large intestine* must not be treated by primary resection and anastomosis. The loop should be either exteriorised and drained, or resected and both ends drained (see below). The faecal fistula may close spontaneously, or a second operation may have to be undertaken for its closure, after the complete recovery of the patient.

The treatment of gangrenous or *non-viable small-gut* cannot be dismissed so easily. From the commencement of this century surgeons have preached the doctrine of *immediate resection and anastomosis*, and, in this country at least, this has come to be regarded as the treatment of election. However, the mortality has proved so high, that a

reaction in favour of more *conservative* methods is setting in, particularly in America (Elman (7), Wangenstein, and others). One feels bound to agree that a procedure with the mortality of immediate resection and anastomosis (42.8 per cent in Frankau's series and 50.4 per cent in the Friedrichshain series) should not be advocated and followed blindly unless it is certain that other methods will not give a lower mortality.

What the ultimate position will be we cannot forecast, but it is our view that both *immediate resection* and the more conservative methods of *exteriorisation* and *fistula formation* have their indications and usefulness, and that neither should be advocated to the exclusion of the other.

A. *Immediate Resection and Anastomosis.* This procedure has three advantages over exteriorisation: (i) the patient does not lose ground through non-absorption of nourishment; (ii) it is free from the danger of sloughing and cellulitis of the abdominal wall, owing to auto-digestion by the escaping digestive juices; (iii) it avoids the subsequent problem of closing an intestinal fistula.

For these reasons, immediate resection should be chosen in all cases in which there is a good chance of survival: i.e. an *early strangulation* of a small loop in a patient in good or moderately good condition. It is impossible to fix a time limit, and there are bound to be borderline cases, but the procedure is particularly desirable in *young children*, who rarely tolerate an intestinal fistula, and in *jejunal strangulation*, where a high fistula is very apt to lead to severe auto-digestion and rapid starvation. The condition of the intestine *above* the strangulation may also influence our choice: the healthier it is, the better are the prospects of immediate resection. The *length* of the segment to be resected may be an important factor in large inguinal or umbilical herniæ; enormous lengths of intestine have been removed with little or no harmful consequences, but as a rule the shorter the necrosed segment the better is the chance of survival. Not infrequently the *experience* of the operator should be the deciding factor; whilst a skilled surgeon may be justified in taking the immediate risk of a primary resection, the occasional operator will be well advised to choose the simpler and, in his hands, less dangerous procedure of exteriorisation, unless the circumstances are exceptionally favourable for the more radical measure.

The *technique* of intestinal resection and anastomosis is more

properly dealt with in text-books on operative surgery. Four points, however, require consideration here.

(a) *It is never enough to remove only the non-viable segment.* Most fatal cases can be attributed to *peritonitis*, from leakage of the anastomosis, or from perforation or paralysis of the proximal intestine. The latter is always distended, and its blood supply is interfered with, particularly in the last one or two feet; this portion, moreover, may show patches of "distension necrosis" along its anti-mesenteric border, which are a grave source of danger. For these reasons it is *essential* to the success of the operation to *resect the more grossly damaged proximal intestine* (usually one or two feet), as well as the necrosed loop itself.

(b) The second point worthy of consideration is the *anastomosis*. Ideally, the end-to-end method should be employed, but this may prove impracticable on account of the disproportion between the dilated proximal end and the very narrow and contracted distal end. In such cases a *lateral anastomosis* should be preferred; owing to the close proximity of the invaginated stumps the anastomotic opening must be *not less than two inches* in length. On several occasions I have performed an *end-to-side anastomosis* after a small-gut resection, implanting the distal end into the anti-mesenteric border of the proximal piece: the results were most satisfactory.

A number of technical points require mention at this juncture: (i) Whichever method of anastomosis be adopted, but particularly in the end-to-end method, separate lengths of catgut should be employed for each of the four layers of suture, i.e. one for the posterior sero-muscular, one for the posterior through-and-through, one for the anterior through-and-through, and one for the anterior sero-muscular (fig. 577). Only in this manner can we make certain of avoiding a narrowing of the lumen by the purse-string action of the continuous suture. (ii) The intestine should be divided or incised with the canter, as this arrests bleeding and ensures an aseptic edge for the anastomosis. (iii) Non-crushing clamps should be used, unless the clamped portion is to be invaginated (e.g. in lateral anastomosis). (iv) When lateral (side-to-side) anastomosis is employed, the stumps must be closed and invaginated with the utinost enre, and then fixed with several sutures to the adjacent limbs of the anastomosis, the actual ends of the stumps being turned in against the intestine (fig. 578). By this procedure we can absolutely safeguard the patient against the very real danger of "bursting" or leakage of the stumps, especially the proximal one.

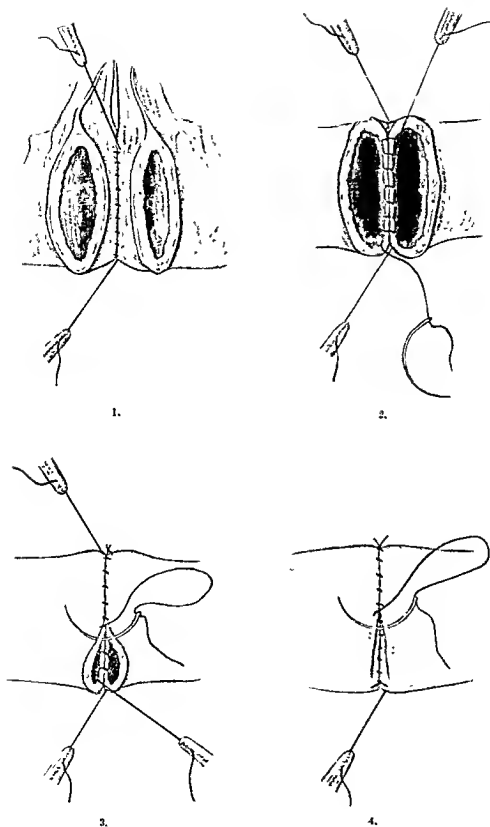


Fig. 577.—STAGES OF END-TO-END ANASTOMOSIS AFTER RESECTION OF SMALL INTESTINE.

(c) The third point refers to the much-debated question—should the resection and anastomosis be performed *in situ* (i.e. outside the hernial orifice) and the intestine then pushed back into the abdomen, or should the abdomen be opened by a *separate incision*, and the damaged bowel withdrawn through this wound for the resection and anastomosis? The first course is undoubtedly the safer of the two, as it avoids the danger of soiling the peritoneum. By enlarging the hernial orifice sufficiently, and by employing the inguinal route (Lotbeissen's operation) in femoral strangulations, the bowel can be both pulled down for the resection, and returned after the anastomosis, without any danger of trauma.

(d) The last point deals with the question of *enterostomy*: should one trust the anastomosis to relieve the proximal distension, or should one safeguard it, and ensure the decompression of the distended

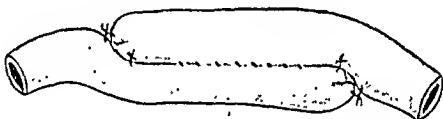


Fig. 574.—DIAGRAM OF COMPLETED LATERAL ANASTOMOSIS WITH TURNING-IN OF STUMPS.

intestine, by draining it above the anastomosis? Our decision will be influenced mainly by the condition of the proximal intestine. Some of its contents should be allowed to escape during the resection, and if we are then satisfied that it is not unduly distended, and that it is *undergoing peristalsis*, enterostomy is unnecessary. On the other hand, if it remains grossly distended, and particularly if *peristalsis is sluggish or absent*, an enterostomy becomes essential. The intestine should be drained a foot or so above the anastomosis, the catheter being brought out through a stab wound clear of the main incision.

B. *Exteriorisation* (Resection and Anastomosis by Stages). In the presence of non-viable gut, the only alternative to immediate resection and anastomosis is to bring the loop out and leave it in the wound (exteriorisation). The necrosed segment, with a margin on the proximal side, may be excised at once, and a Paul's tube fixed into each open end; but it is usually wiser to postpone the resection for 24 to 48 hours, contenting oneself at the first operation with an enterostomy of the



estine a little proximal to the exteriorised segment. After an interval of four to ten days, varying with the condition of the patient and the level of strangulation, the operation is completed by closing the faecal fistula and re-establishing the continuity of the intestine.

Although this procedure is simple in theory, and has a much lower primary mortality than the more radical method of immediate resection and anastomosis, in practice it is associated with the disadvantages separable from an intestinal fistula. It should be observed that the version of the intestinal contents is *complete*, and that the fistula is uncontrollable. While the Paul's tubes remain *in situ*, this complete version is not of serious consequence, but after a few days the tubes loosen, and the highly proteolytic contents of the intestine escape into and on to the abdominal wall. The resulting auto-digestion is complicated by sepsis, and large parts of the abdominal wall become necrotic and may slough, or even a fatal cellulitis may supervene. The opening of the peritoneal cavity for the completion of the anastomosis now becomes an extremely formidable procedure, owing to the danger of peritonitis. Furthermore, especially with a high or moderately high fistula, the patient progressively deteriorates through malnutrition.

The existence of these well-known dangers is responsible for the preference which many surgeons show for primary resection and anastomosis. But with care and ingenuity most of the unpleasant consequences of exteriorisation can be avoided, and we believe that it could be chosen as the lesser of two evils, unless the circumstances are unfavourable for primary resection (see page 1057).

The following is recommended as the best *exteriorisation procedure* (g. 579): The strangulated coil, with 18 inches of proximal and 18 inches of distal intestine, is brought out of the wound. A *lateral anastomosis* is established as far as possible from the necrosed segment, and the anastomosed portion is returned into the abdomen. A *Witzel enterostomy* is then made a little proximally to the anastomosis, the catheter being brought out through a separate stab wound. The strangulated coil, with a margin of viable gut at each end, is fixed outside the wound by a rod passed through its mesentery, and the wound closed snugly round its base; if it is very distended a self-retaining catheter may be fixed into it for drainage. The entire operation is carried out under *local anaesthesia* and should cause practically no shock.

After 24 to 48 hours the exteriorised loop is *resected* with the gutters, and a Paul's tube is tied into each open end (fig. 579). In

jejunal cases the contents escaping from the proximal end can be injected into the distal tube, thus maintaining the nutrition of the patient, but in the common ileal cases this is unnecessary. After the Paul's tubes come out, the fistulæ should be closed without delay, e.g. about the fifth day; by this time the patient and the intestine should have recovered sufficiently to make the closure safe, particularly if the proximal enterostomy is still functioning.

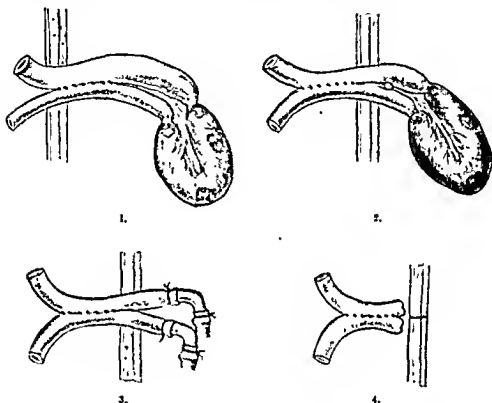


Fig 579.—ILLUSTRATING EXTIRPATION WITH LATERAL ANASTOMOSIS AND DELAYED RESECTION (IN STAGES).

When a lateral anastomosis has been established at the first operation, all that now remains is to free, close, and invaginate both open ends, and, after thorough cleansing of the skin, deliberately to reopen the peritoneum and return them into the abdomen. But if the lateral anastomosis has for some reason (e.g. desperate condition of the patient) been omitted, an anastomosis has to be performed now. This is likely to prove both difficult and dangerous. Any attempt to effect an extra-peritoneal anastomosis is practically doomed to failure, and the opening of the abdomen, in the presence of a complete fistula and infected parietes, is fraught with danger.

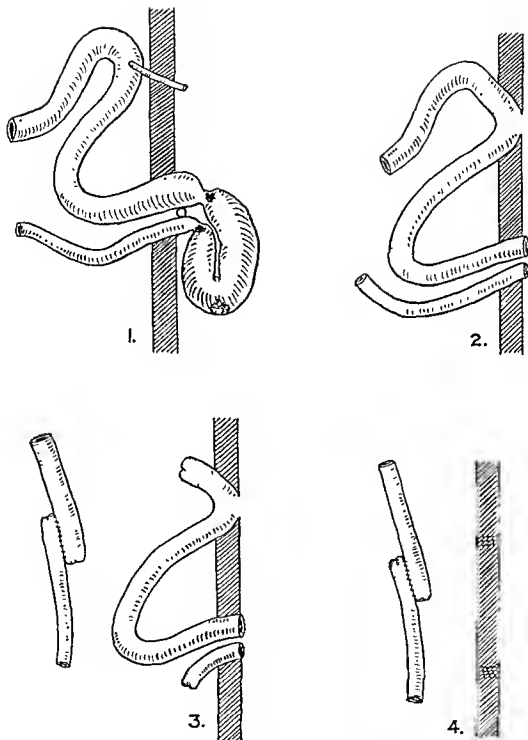


Fig. 589.—BILATERAL EXCLUSION AND SUBSEQUENT RESECTION OF FISTULOUS INTESTINE AFTER EXTERIORISATION FOR STRANGULATED HERNIA.

The following case, recently under my care, illustrates a procedure by which this difficult problem can sometimes be solved :

A man of 72 was admitted with a *left femoral hernia* which had been *strangulated* for eight days, with absolute constipation from the first day, and *faecal vomiting* for four days. He was almost moribund. Extreme dehydration was shown by deeply sunken eyes, a cracked brown tongue, marked shrinking of the skin and subcutaneous tissue, almost complete anuria, and a *blood urea* of 240. The systolic blood-pressure was under 90, the temperature very low, and he was in a state of extreme drowsiness, bordering on uræmic coma. The *hernia* was tense, tender, and about the size of an orange.

Six hours were spent in attempts to improve his condition sufficiently to make operative intervention possible. A *duodenal catheter* was passed by the oral route, and the contents of the stomach and upper intestine were aspirated. A *radiant heat* cradle was placed over him and  $\frac{1}{2}$  gr. of *morphine* was administered. Two litres of *glucose-saline* were then slowly run into the median basilic vein. At the end of the six hours there was a very definite improvement in his general condition, and operation was begun.

*Local anaesthesia* was induced with 1 per cent novocaine. The sac was exposed by an oblique incision, and found to contain faecal fluid and pus, a six-inch coil of gangrenous ileum, with a large perforation at the deeply constricted neck, and some gangrenous omentum. It was not considered feasible to do either a primary resection or a proximal lateral anastomosis. The gangrenous coil was therefore *exteriorised*, and an *enterostomy* was performed by the Witzel method some distance proximally to the strangulation.

Next day he was much better. The blood urea dropped to 150 and the systolic pressure rose to 120. A further litre of glucose-saline was given intravenously, with continuous subcutaneous saline. On the second day the coil was *resected* and a Paul's tube was tied into each open end. After this stage, duodenal drainage was discontinued and oral feeding commenced.

His general condition continued to improve steadily, but five days later the Paul's tubes came out, and unfortunately the enterostomy wound also started to leak. I attempted to perform an end-to-end anastomosis *in situ*, but this, as we expected, broke down very soon. Marked auto-digestion and sepsis of the abdominal wall set in, with extensive sloughing, to an extent which forbade any attempt at a local intra-peritoneal anastomosis.

On the advice of Prof. C. A. Pannett, I then decided to do a *bilateral exclusion* of the fistulous segment of intestine, between the enterostomy and the resection (see fig. 580). The left half of the abdomen was shut off with a barrier of gauze and mastisol, the right half thoroughly cleaned, and a right para-rectal incision made, under spinal anaesthesia. The loop between the two fistulous openings was identified and excluded, the four ends were invaginated, and a *lateral anastomosis* was performed between the intestine above and below the excluded segment (see fig. 580).

He made an excellent recovery, the abdominal wall healed well, and the excluded loop (measuring 51 inches) was *resected*, under spinal anaesthesia, some ten weeks later, the fistulous openings in the abdominal wall being repaired by a plastic operation. During the interval, the isolated loop, opening to the surface at both ends, was utilised for physiological and pharmacological experiments.

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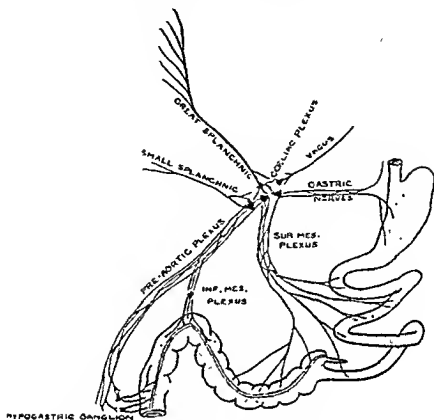


Fig. 541.—EXTRINSIC INNERVATION OF ALIMENTARY TRACT.

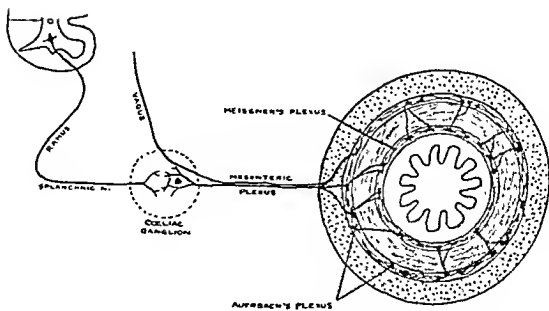


Fig. 542.—INTRINSIC INNERVATION OF INTESTINE.

The *intrinsic mechanism* is provided by *Auerbach's plexus*, situated between the circular and longitudinal coats of the intestine. From its cells short post-ganglionic fibres proceed to the plain muscle of the circular coat. There is a second and less conspicuous plexus in the submucous layer (Meissner's), but this is not concerned with intestinal movements.

*Physiology of Peristalsis.* Four more or less distinct movements occur in the small intestine: *rhythmic contractions* (causing segmentation of the contents), *pendulum movements*, and two kinds of *peristalsis*. Rhythmic contractions and pendulum movements are myogenic in origin, since they are not abolished by cocaine or nicotine.

Peristalsis is of two varieties: (i) a short slow *peristaltic wave*, and (ii) a long swift *peristaltic rush*. Both are undoubtedly neurogenic in origin as they are abolished by cocaine and nicotine. The movement of peristalsis is a wave of contraction of the circular coat, travelling distally, and immediately preceded by a wave of relaxation. The control of the movement is primarily vested in *Auerbach's plexus*, which originates, conducts, and co-ordinates the motor stimuli. Although connected with the central nervous system by sympathetic and parasympathetic nerves, the plexus appears able to function independently, since peristalsis continues after section or excision of both sets of extrinsic nerve fibres. We thus conclude that Auerbach's plexus forms a complete reflex centre, receiving stimuli from the mucosa and muscularis, originating both the contraction and relaxation waves of the circular muscle, and co-ordinating them so that the latter just precedes the former to constitute an effective forward peristaltic movement.

The plexus, in its turn, is subject to the control of antagonistic sympathetic and parasympathetic influences. Stimulation of the *vagus* and *sacral autonomic* augments peristalsis, while *sympathetic* stimuli inhibit peristalsis and produce contraction of the sphincters. The parasympathetic control is direct, since the *vagus* and *sacral autonomic* reach the plexus as pre-ganglionic fibres; but the sympathetic control is indirect, the fibres reaching the plexus being post-ganglionic and interrupted at the cell-stations of the *celiac ganglia*.

Normally, a balance is struck between the sympathetic and parasympathetic influences, any predominance of one over the other being slight and short-lived, and determined by the physiological requirements of the moment. Under certain circumstances, however, one or other of the extrinsic controls gains the upper hand and completely

For the reader's convenience the above causes are summarised in the form of a table :

Paralytic Ileus	Lesions of intestinal muscle (ischæmia or fatigue)	<ul style="list-style-type: none"> <li>Strangulation</li> <li>Mechanical obstruction (late)</li> <li>Over-purgation (?)</li> </ul>
	Myo-neural junction (? loss of acetyl-choline)	<ul style="list-style-type: none"> <li>Surgical shock</li> <li>Chloroform or ether</li> </ul>
	Lesions of Auerbach's plexus (inflammatory, ischæmic, or toxic)	<ul style="list-style-type: none"> <li>Peritonitis</li> <li>Strangulation</li> <li>Embolism or thrombosis</li> <li>Pneumonia</li> <li>Uremia</li> <li>Toxæmia</li> </ul>
	Sympathetic irritation	<ul style="list-style-type: none"> <li>Operative trauma</li> <li>Peritonitis</li> <li>Shock (severe injuries)</li> <li>Internal torsions</li> <li>Renal calculus</li> <li>Chemical peritonitis (bile, blood, etc.)</li> </ul>

Causes of  
"reflex"  
ileus

**Morbid Anatomy of Paralytic Ileus.** The morbid changes vary with the cause of paralysis. In *peritonitis*, local or diffuse, the ileus is often mechanical as well as functional; coils of intestine may be glued together with the formation of kinks and spurs, or actual filmy adhesions are present. It may be difficult in such a case to decide whether the mechanical or paralytic factor is predominant.

The paralysis which supervenes on an ordinary *mechanical obstruction* (e.g. carcinoma, fibrous bands, gall-stones, etc.) is always a late result, and is thus associated with advanced morbid changes in the intestine (see page 979).

A more typical picture of paralytic ileus is provided by *non-peritonitic post-operative obstruction*, for which irritation of the sympathetic is responsible. The outstanding changes in such a case are :

(i) *Cessation of peristalsis.* In the early stages this may be limited to a segment of the intestine, but later it spreads to the greater part or the whole of it.

(ii) *Extreme distension of the paralysed intestine.* Typically the distension is regional, being interrupted by areas of sphincteric spasm. An advanced case may show four regions of distension, namely, the



stomach, the duodenum, the small gut, and lastly the colon. When only part of the intestine is involved the transition from distended to normal gut is always *gradual*.

(iii) *Spasm of the sphincters.* The pylorus, duodeno-jejunal flexure, ileo-cæcal junction, recto-sigmoidal junction, and the anus are the sphincteric regions of the intestinal tract. Any of these may be the seats of spasm in paralytic ileus, but the ileo-cæcal sphincter is the one most constantly involved. According to Rohb (2) the lower end of the ileum may share in the ileo-cæcal spasm, being usually empty and sometimes contracted.

We have no means of knowing whether the actual obstruction in paralytic ileus is mainly caused by sphincteric spasm or by paralysis of inter-sphincteric segments of intestine. Certainly in milder cases spasm of the sphincters appears to play an important part, and it probably always precedes total loss of peristalsis. For the common post-operative meteorism without a true ileus, spasm of the anal sphincters often seems to be mainly responsible.

In the *last stage* of paralytic ileus distension becomes general and extreme. The wall of the intestine is congested and shows areas of oedema, ulceration, or necrosis (distension-necrosis); ultimately peritonitis supervenes, whether perforation occurs or not. Dehydration and depletion of the blood-plasma result, as in mechanical obstruction. *Renal failure*, with anuria, is a common sequela of intestinal paralysis; in part this association is explained by dehydration, but another likely cause is reflex spasm of the renal arteries, produced by the sympathetic irritation.

## (2) PATHOLOGY OF RECENT POST-OPERATIVE ADHESIVE OBSTRUCTION

*Causation.* A large majority of recent adhesive obstructions follow operations for *acute appendicitis*. According to Wilkie (3), over 2000 deaths occur in this country from "post-appendix" obstructions every year. This complication rarely follows operations on the cold appendix or extra-peritoneal drainage of an abscess, but it is a common result of removals of difficult third or fourth day appendices, particularly in the hands of inexperienced surgeons (Newland, 4). Rough handling, tearing of protective adhesions, indiscriminate use of gauze swabs, prolonged drainage, and unsuitable incisions all contribute to the development of adhesive and paralytic ileus; but dissemination of sepsis, with a consequent local or general *peritonitis*, is the usual cause

of the more serious cases. The peritoneum is mostly infected by rupture of a pus-filled appendix during its removal, or by allowing the contents of an abscess to escape into the general peritoneal cavity.

*Pelvic operations*, and operations on the *gall-bladder, colon, rectum, and stomach* are less common causes of early adhesive obstruction. Again, operative trauma and infection are the factors usually responsible, but adhesions may also be occasioned by denuding viscera, the abdominal wall, or the pelvic floor of their peritoneal covering.

### *Mechanisms and Morbid Anatomy.*

(a) *Simple Obstruction.* The great bulk of post-operative adhesive obstructions are *simple* and involve the *lower part of the ileum*. The fundamental cause of obstruction is the *fibrinous exudate* produced in response to injury or infection of the peritoneum.

Sometimes actual *adhesions* are formed by the rapid organisation of this fibrinous exudate into soft filmy sheets or strands. These adhesions loosely bind the intestine to the abdominal incision, the posterior parietes, or to an inflamed or denuded viscus. The fixation of the intestine, although slight, may interfere with the straightening out of the bends of the affected coil which is necessary to the forward movement of peristalsis. A state of partial obstruction is thus produced, and this leads to *distension* of the intestine proximal to the "fixed" point; the distension, in turn, drags on the fixed point and so causes a *kink*, which may convert the partial obstruction into a complete one (see fig. 545).

After a few days the fibrinous adhesions are usually absorbed and the "fixed" intestine once again becomes free. During the period of fixation, however, a definite obstacle is offered to the forward passage of contents. Strong peristaltic efforts may clear the obstacle, and thus, by keeping down distension, avoid a complete obstruction and so tide the patient over the danger period. Not infrequently, however, some degree of paralytic ileus is also present: the powerful peristalsis which is called for cannot be produced, and so the obstruction becomes complete. This combination of partial mechanical blockage with paralytic ileus is common and is doubtless responsible for many of the fatal results.

In some obviously mechanical post-operative obstructions *no actual adhesions* can be seen when the abdomen is opened. This is a common experience in appendicular ileus, where the obstruction is caused by *acute angulation* of the terminal coil of ileum. The two limbs of the coil are *glued together* with fibrin; a severe *kink* is thus produced, opposite

which a *valve-like spur* is formed by the lightly adherent apposed walls. Peristaltic waves reach the glued segment but cannot straighten out the kink; instead, they push the valvular spur before them and so obliterate the lumen beyond (see fig. 583). It was pointed out by Pannett (5) that the same thing may occur if neighbouring coils are thrust against the loop in question owing to a rise of the intra-abdominal pressure, e.g. from distension. When the abdomen is opened, the release of pressure may suffice to straighten out the kink, and so the cause of obstruction may no longer be obvious.

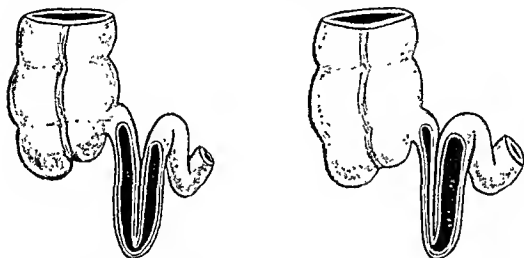


Fig. 583.—ANGULATION AND SPUR-FORMATION OF TERMINAL ILEUM IN APPENDICULAR OBSTRUCTION.

We must repeat that this state of things is very temporary, for in a few days the fibrin is absorbed and the angulated ileum straightens out. Unfortunately, however, the obstruction may last long enough to kill the patient.

The *morbid changes* in the distended proximal intestine and the *physiological disturbances* are those of any simple low small-gut obstruction. Special dangers are associated with the presence of the glued "closed" loop of appendicular obstruction, depending mainly on its contents at the time of operation. If the contents are abundant, extreme gaseous distension occurs, and rapid tension-necrosis and peritonitis follow almost inevitably. More often the contents are scanty and the dangers of necrosis slight.

(b) *Strangulation*. Early post-operative strangulation of the intestine is rare. *Volvulus* has been known to occur, the fibrinous adhesions forming a fixed point round which the gut rotates. Very rarely, *internal strangulation* may follow operations, such as

gastro-enterostomy or colostomy, in which a hole or arch is left; through or under this, a coil of intestine herniates and becomes strangulated.

### (3) CLINICAL TYPES, DIAGNOSIS, AND COURSE OF POST-OPERATIVE OBSTRUCTION

Three clinical grades of post-operative obstruction may be distinguished, although very often they follow one another or occur together: (i) mild inhibition of peristalsis, (ii) paralytic ileus, (iii) adhesive ileus.

(i) *Mild Inhibition of Peristalsis.* Weakening of peristalsis, with mild spasm of the sphincters, is a very common sequel of any major abdominal operation, and is no doubt dependent on slight stimulation of the sympathetic. Clinically, this grade manifests itself by *constipation, gas-pains, and meteorism*. Although these symptoms may be distressing, there is no actual obstruction. Commencing towards the end of the first twenty-four hours, the worst of the discomfort is over before the third day, the symptoms often disappearing with the administration of the first enema. At most only moderate distension of the intestine occurs, perhaps with temporary kinking. Spasm of the anal and other sphincters seems to be an important factor; indeed, much of the discomfort of gas-pains can be abolished by routine stretching of the anus in abdominal operations, or by the use of a flatus-tube.

Although this grade of post-operative stasis is in itself quite harmless, it should never be regarded as unimportant, since it might well prove the forerunner of a serious paralytic or adhesive ileus. Fortunately it is responsive to treatment of the right kind (see page 1084).

(ii) *Paralytic Ileus.* Post-operative paralytic ileus is of gradual onset, the symptoms of complete obstruction supervening almost imperceptibly on the gas-pains and meteorism of first-grade stasis. Sometimes the development of intestinal paralysis can be anticipated (e.g. gross peritoneal contamination or trauma at operation); at other times it catches us unawares. Most cases start on the *second or third day* following operation, not infrequently a few hours after the first aperient (Wheeler, 6).

The *symptoms* are abdominal pain, vomiting, distension, and absolute constipation. The *pain* is continuous, dull, wearing, and situated in the umbilical and hypogastric regions; it is caused by

tension on the muscle-fibres of the dilated intestine. *There is no colic. Vomiting is persistent and copious. Tympanitic distension of the abdomen appears early and increases rapidly; at first it is usually central, but soon the abdomen becomes barrel-shaped. Absolute constipation is present from the start, inability to pass flatus being a conspicuous feature. The lower colon may be emptied with an enema, but no flatus is passed. Further enemata produce no response, while aperients only increase the distension and vomiting.*

The diagnostic sign is *absence of peristalsis*. It cannot be seen, felt or heard. *Dead silence on auscultation of the abdomen can only mean paralytic ileus.* At an early stage this silence may be limited to one region, but before long, particularly in peritonitic ileus, peristaltic sounds cease everywhere. X-rays reveal gaseous distension of the small intestine with fluid levels, but we do not favour this method of investigation.

The *duration and course* of paralytic ileus is variable. It may be overcome in a few hours, it may continue for two or three days and then clear up, or it may progress to gross distension, dehydration, peritonitis, and fatal collapse. An unrelieved case usually dies on the third, fourth, or fifth day, but occasionally life is prolonged for a week. *Recovery* is always heralded by the *return of peristalsis*, which should be listened for frequently. Soon after peristaltic sounds become audible again, flatus is passed, vomiting ceases, and the distension subsides; complete recovery rapidly follows, unless mechanical obstruction is present as well.

(iii) *Adhesive Obstruction.* The symptoms of early mechanical obstruction make their appearance usually between the *third and seventh day* after operation, the highest incidence falling on the fourth and fifth days; this is two or three days later than the usual commencement of paralytic ileus. The *onset* is almost always gradual, the picture changing insidiously from post-operative constipation and gas-pains to a true mechanical obstruction. This stealthy beginning may occasion dangerous delay in diagnosis and treatment, but much of this danger can be avoided if we regard with suspicion all cases in which post-operative pain, vomiting, and constipation persist beyond the second or third day.

An established mechanical obstruction produces three outstanding symptoms: (i) *intermittent colicky pains* (very different from the continuous dull ache of paralytic ileus), (ii) *persistent vomiting*, and (iii) *cessation of flatus*. Abdominal distension is a relatively late symptom and must not be waited for. The diagnostic sign is *increased peristalsis*,

particularly obvious on *auscultation*, and presenting a striking contrast to the absolute silence of paralytic ileus.

Although colicky pains and increased peristaltic sounds in a post-operative patient usually mean a mechanical obstruction, the picture may be altered by the presence or subsequent development of peritonitis. Another change is produced by the supervention of paralytic ileus, a mild degree of which may, incidentally, have been present from the start. It is thus evident that the sooner we recognise the presence of a post-operative obstruction, the more likely we are to diagnose its type.

A rather different variety of early adhesive obstruction is sometimes seen, in which there is a clear interval of a *week* or *ten days* between the operation and the obstructive symptoms. The onset here is usually much more definite, peristalsis tends to be very vigorous (owing to absence of paralytic ileus), and there is no difficulty in making an early diagnosis. The outlook in this type is correspondingly more favourable.

#### (4) DIFFERENTIAL DIAGNOSIS BETWEEN ADHESIVE AND PARALYTIC ILEUS

In uncomplicated cases it is usually possible to differentiate a mechanical obstruction from paralytic ileus, and occasionally a good deal may depend on this differentiation. More often the distinction is not so important as might at first sight appear. The two conditions frequently co-exist, and the treatment of both is in the first place conservative, and in many respects similar. Nevertheless, an accurate diagnosis should always be attempted.

The main differences have already been indicated, but they may be conveniently summarised as follows:

	<i>Paralytic Ileus.</i>	<i>Adhesive Obstruction.</i>
Onset . . . .	2nd or 3rd day.	3rd to 7th day.
Pain . . . .	Continuous dull ache.	Intermittent colicky pains.
Vomiting . . .	Persistent from start.	Becoming persistent.
Visible peristalsis . .	Absent.	Sometimes present.
Audible peristalsis . .	Absent.	Always present and increased.
Abdominal distension .	Early; rapidly progressive.	Later; progresses more slowly.
Course . . . .	Death in 3 or 4 days.	Usually longer.

In doubtful cases three procedures are available, which serve to distinguish paralytic from mechanical obstruction. They are:

(i) Certain *drugs* which stimulate peristalsis, e.g. morphine, *eserin*, and acetyl-choline. They may overcome intestinal paralysis but can

have no beneficial effect on mechanical obstruction. Bile enemata come under the same heading.

(ii) Intravenous *hypertonic saline* produces a more definite action in the same direction. Given at the right time it is a powerful remedy against paralytic ileus, but in mechanical obstruction its only effect is to increase the colicky pains.

(iii) *Spinal* (or splanchnic) *anaesthesia* is of the greatest possible value since it absolutely differentiates paralytic ileus of sympathetic origin from other types of post-operative obstruction. By abolishing the inhibitory action of the sympathetic, it overcomes intestinal paralysis from this cause in a few minutes, while it can have no effect on a pure mechanical obstruction.

An important observation must be made in connection with these three procedures, i.e. they may produce some improvement in mechanical obstructions complicated by paralytic ileus. Here the mechanical block is usually partial, the obstruction being made absolute by paralysis of the intestine. Abolition of the latter may result in some return of intestinal function, but this return is never complete and rarely more than *temporary*.

### PREVENTION OF POST-OPERATIVE OBSTRUCTION

We have seen that the chief causes of post-operative obstruction are: (i) infection (local or general peritonitis) and (ii) trauma at operation. To these we must add: (iii) shock or anxiety (especially in nervous people); (iv) prolonged chloroform or ether anaesthesia; (v) drastic pre-operative catharsis; (vi) a full intestine before operation, or too early and generous fluids after it; (vii) anal spasm; (viii) denudation of peritoneum; (ix) prolonged drainage.

Most of these causes are connected with the operation itself, but some are antecedent to it, while others follow it. It is thus convenient to consider prophylactic measures under the headings of pre-operative, operative, and post-operative.

*A. Pre-operative Prophylaxis.* Essential measures before operation are: (i) to reassure the patient and prevent anxiety or fear; (ii) to have a reasonably empty stomach and intestine; (iii) to avoid drastic pre-operative purging.

Patients of "nervous" temperament are more susceptible to paralytic ileus after abdominal operations than are those of the placid type.

This may be explained by sympathetic activity from cerebral stimuli (Stout, 7), or perhaps from increased suprarenal secretion. It follows that every effort must be made to allay the natural fears and anxiety of the pre-operative patient. A cheerful and confident manner on the part of the surgeon and nurses will do a good deal, but for bad cases of "operation nerves" nothing acts so well as a small dose of morphia ( $\frac{1}{2}$  to  $\frac{1}{4}$  gr.).

The importance of a reasonably *empty alimentary canal* is obvious. The cleaner the intestine the less the matter available for bacterial or enzyme decomposition; should paralytic or mechanical obstruction follow, distension of the intestine will be minimal, and the prospect of recovery materially improved. Therefore, in all "set" operations entailing a risk of subsequent ileus (e.g. intestinal resections, difficult pelvic operations, etc.), the patient should be kept on a low residue diet, mainly clear fluids, preferably for two days prior to operation; the lower bowel should be emptied by a wash-out a few hours before it. With urgent operations (e.g. acute appendicitis) there is no time to do more than empty the lower colon, but fortunately the patient has often been starved for the preceding day or two.

Drastic pre-operative *catharsis* undoubtedly predisposes to paralytic ileus. Whether it acts by exhausting the intestines or by making it more sensitive to sympathetic stimuli we do not know. Indeed, it may be that the excessive peristalsis uses up the available supply of acetylcholine in the gut-wall, while a contributory factor may be loss of bile, which is hurried out of the body instead of being re-absorbed. Whatever the mechanism responsible, there can be no question of the harmful consequences of pre-operative purging. If catharsis is essential (e.g. in intestinal resections) it should be done several days before operation, and the patient kept on clear fluids and non-residue food in the interval.

**B. Prophylaxis at Operation.** The main prophylactic measures may be summarised very briefly: (i) prevent and combat infection; (ii) avoid every form of unnecessary trauma; (iii) abstain from chloroform or ether anaesthesia; (iv) cover all raw areas and carefully suture all peritoneal wounds; (v) avoid prolonged or incorrect drainage.

The most important cause of post-operative obstruction is *peritoneal infection*. Peritonitis causes paralytic ileus by stimulating the sympathetic or by implicating Auerbach's plexus in the inflammatory process (see page 1071); it produces adhesive ileus by glueing adjacent coils together or by the formation of fibrinous adhesions.

Sometimes the peritoneum becomes infected through no fault of the



surgeon, but more often such *infection can be prevented*. *Spillage* of intestinal contents must be most scrupulously avoided, and the strictest *aseptic routine* maintained from the first to the last step of the operation. Above all, the greatest care should be taken to prevent the escape of the contents of a localised abscess or of a pus-containing viscus (e.g. a purulent appendix). Adhesions limiting a suppurative process must be treated with the greatest respect, and localised abscesses should, whenever possible, be reached and drained without traversing the uninfected peritoneal cavity.

Should peritonitis be present already, the infecting focus must be removed and adequate drainage provided. The toxæmia must be fought energetically. In America much emphasis is laid on the value of *bacteriophage*. Smith (8) advises its administration at operation by a catheter inserted into the upper abdomen through the wound or drainage-tube; 30 to 60 cc. of autogenous or mixed anti-coli and anti-staphylococcus and streptococcus bacteriophage is given and repeated daily for the first few days. *Anti-Welchii serum* has been strongly advocated for peritonic ileus, but the results are on the whole rather disappointing.

*Operative trauma* is the second great cause of post-operative obstruction, and is the more deplorable inasmuch as it can often be attributed to bad surgery. The *types* of trauma responsible include: (i) unnecessary "explorations"; (ii) attempts to reach and remove an organ through an unsuitable incision (e.g. removing an adherent appendix through a mid-line or epigastric incision); (iii) forcible and prolonged retraction of the abdominal wall; (iv) rough handling, pushing and pulling of the peritoneum or viscera; (v) evisceration and exposure of the intestine, which soon dries and gets chilled; (vi) tearing of adhesions, omentum, mesentery, and peritoneal coverings; (vii) forcing packs and masses of gauze-roll against the intestine and highly sensitive peritoneum (e.g. in difficult cholecystectomies and pelvic operations); (viii) "flushing" the peritoneum with irritating or cold lotions and constant "mopping" with swabs; (ix) unnecessary denudation of viscera or parietes of their peritoneal covering, and failure to recover the bare areas.

Naturally, such operative faults are more likely to be suffered at the hands of the unskilled and "occasional" surgeon, than at those of the experienced and skilled operator. But errors of judgment, rashness, lack of patience, and the indifference bred of familiarity may lead even the skilled surgeon to commit one or other of these inexcusable errors. The only way to protect the patient against post-operative obstruction

is to regard it as a likely complication of every abdominal operation, and to take all possible precautions against it.

In this connection, *spinal anaesthesia* has the great advantage of providing full muscular relaxation, thus avoiding forcible retraction of the abdominal wound, and reducing visceral and peritoneal manipulation and packing to a minimum. It is, moreover, completely free from the great drawback of chloroform and ether, which is a definite tendency to initiate or aggravate a paralytic ileus; on the contrary, spinal anaesthesia has a well-marked stimulating effect on peristalsis. When the spinal route is inadvisable we should employ local and regional infiltration, with gas and oxygen or one of the safer forms of pre-anaesthetic narcosis.

*Incisions* must be planned to provide the easiest approach to the lesion, again with the object of minimising operative trauma. The attempt to remove a difficult appendix through a mid-line or epigastric incision is extremely bad surgery. If such an incision has already been made, and the caecum cannot be brought to it with ease, it should be closed and a second incision made over the appendix. The use of *packs* should be reduced to a minimum, and they must be inserted gently and loosely. For difficult pelvic operations Guthrie (9) advises an extreme *Trendelenburg position* at the commencement of anaesthesia, which ensures that by the time the abdomen is opened the intestine is already out of the way.

Intestine, other viscera, omenta, and mesenteries must be handled with the *utmost gentleness*. *Evisceration* of more than one or two coils at one time is rarely necessary, and all exposed intestine should be covered with hot packs. *Denuded areas* on viscera or parietes must be carefully covered with peritoneum, or failing this with omentum. When the closure of the abdomen is commenced it is essential to *oppose and suture accurately the edges of peritoneum*, otherwise one or more coils of intestine are extremely likely to become adherent to the scar. Any *openings* made in the course of anastomotic or other operations (e.g. posterior gastro-jejunostomy, entero-anastomosis, etc.) must be closed, and *kinking or narrowing of the intestine* guarded against. When *drainage* is necessary, the tube should be inserted into the most dependent part of the region to be drained, and in such manner that it takes the shortest possible route through the peritoneal cavity. Drains should be removed early as they encourage the formation of adhesions.

In short, the surgeon should avoid meddling surgery, limit his intervention to what is strictly essential for the welfare of the patient, and carry out all intra-peritoneal procedures speedily and gently.

*Prophylaxis in Appendix Operations.* Most cases of recent post-operative obstruction occur after operations for appendicitis. I have never seen it follow retroperitoneal or rectal drainage of appendix abscesses, and only twice have I known it to occur after a "cold" appendicectomy. But it is a common complication of acute appendicectomies and also of transperitoneal drainage of abscesses.

The incidence of post-operative ileus is highest in the difficult third or fourth day appendices, where it affords the only real argument in favour of the so-called Ochsner-Sherren or conservative treatment for such cases. I cannot help thinking that the incisions in common use are partly responsible for its occurrence. The attempt to free and

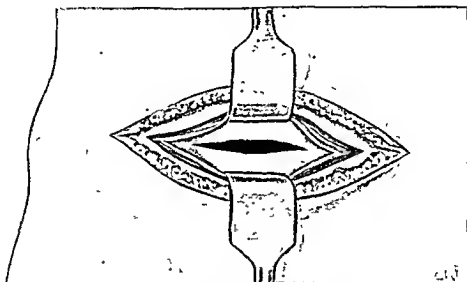


Fig. 584.—AUTHOR'S INCISION FOR APPENDICECTOMY.

remove a fixed and friable appendix through a median or paramedian incision is almost bound to prove difficult and to cause trauma; indeed a not uncommon result is rupture of the purulent appendix and infection of the peritoneum.

For these and other reasons I have for several years employed in all undoubted appendix cases a *transverse incision* (fig. 584) immediately medial to the anterior superior spine (10). This provides much the easiest approach to the cæcum and appendix, and permits the removal of difficult appendices with a minimum of disturbance or trauma. Further, it is particularly suitable for the retroperitoneal drainage of an *iliac appendix abscess* (see fig. 585), which by this method can be reached without opening the peritoneal cavity.

The drainage of a *pelvic appendix abscess* should again not be attempted through the peritoneal cavity unless circumstances force

one to do so. Operation should, whenever possible, be postponed until the abscess can be opened into the *rectum*. Finally, no attempt is to be made to remove the appendix in the presence of an abscess, unless it is lying free in the abscess-cavity.

C. *Post-operative Prophylaxis.* A mild inhibition of peristalsis should be regarded as a natural result of every abdominal operation, and premature attempts to force the intestine into activity are likely to do more harm than good. The first essential is *rest*, which is best secured with small doses of morphine. Nothing should be allowed by mouth, not even water, until the patient is free from nausea. Meteorism and gas-pains caused by *anal spasm* will be relieved greatly

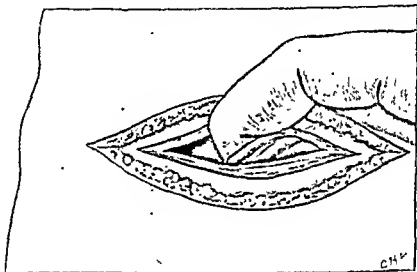


Fig. 553.—RETROPERITONEAL APPROACH FOR ILEO-CECAL ABSCESS.

by the insertion of a *flatus-tube*; to prevent anal spasm and facilitate the expulsion of flatus, Brown (11) recommends routine *stretching of the anal sphincters* after every abdominal operation. A *heated pad* to the abdomen, or a radiant heat cradle, is also of great value in diminishing post-operative distension and gas-pains. If vomiting continues after the first six hours a *stomach tube* should be passed without delay; this avoids distension and *prevents air-swallowing* (an important consideration). *Rectal saline* should be given slowly and continuously, and only when it is retained easily and absorbed at a fair rate. If post-operative ileus is likely it is better to avoid the rectal route and to give the fluid *subcutaneously* or *intravenously*. Oral feeding is started when the patient is free from nausea, preference being given to substances (hot water, fat-free broth, etc.) which stimulate peristalsis (Ochsner and Gage, 12).

## MANAGEMENT OF POST-OPERATIVE OBSTRUCTION

The treatment of post-operative ileus is governed by the *temporary* nature of the obstructing agent, whether this be paralytic or adhesive. In the former, recovery depends on the return of intestinal peristalsis, while in the latter the obstruction clears up with the absorption of fibrinous exudate and adhesions, and the consequent freeing of glued coils and straightening-out of kinks. Therefore, the primary objects of treatment should be: (i) to *keep the patient alive*, and (ii) to *prevent irrecoverable damage of the intestine*, whilst awaiting the re-establishment of the functional or anatomical continuity of the alimentary canal. Operative intervention is justified only when these objects cannot be attained without it.

At the first appearance of obstructive symptoms, it may not be easy to decide whether the ileus is mechanical or paralytic, and valuable time will be lost if we wait until a definite diagnosis can be made. Moreover, in many cases, particularly of peritonitic ileus, adhesions and paralysis are both present, and the proportion of the total obstruction for which each is responsible may vary from day to day. It is, therefore, unwise to draw too sharp a line between the two types of post-operative ileus, in the actual management of cases.

*Management of Early Cases.* The onset of post-operative obstruction is gradual. When it appears in the first three days it is almost always paralytic, while after the fourth day it is probably mechanical. In either case nothing can be worse for the patient than a rushed laparotomy. For the *first twenty-four hours* the obstruction tends to be incomplete, and more often than not can be overcome by rational conservative treatment.

The restlessness and anxiety of the patient is countered by keeping him quiet and by giving small doses of *morphine* or *heroin*. Nothing whatever is allowed by mouth, not even sips of water. A *duodenal catheter* should be inserted without delay, and the distended stomach and upper intestine decompressed by suction-siphonage (see page 998), or by repeated aspiration with a strong syringe. These measures prevent air-swallowing and relieve distension, thus favouring the straightening-out of kinks and preserving the tone and blood supply of the intestine.

Dehydration is avoided and strength maintained, either by continuous *intravenous* glucose-saline, or by generous subcutaneous saline,

punctuated with four-hourly feeds of glucose-saline intravenously. Glucose should not be given alone, or if it is, insulin should be administered with it; without saline or insulin glucose has a depressing effect on peristalsis. Rectal saline is inadvisable in the presence of obstruction. One of the most valuable remedies I know of for early paralytic ileus is *continuous heat* to the abdomen; this may be supplied by an electrically heated pad or by a radiant heat cradle, the former being much the more comfortable.

In paralytic cases the *return of peristalsis* is shown by the passage of flatus, but may be discovered before this on auscultation of the abdomen. Whether peristalsis returns or not, an *enema* should be given at the end of the first twenty-four hours. My own preference is for an *ox-bile* enema, six ounces of bile being run in slowly through a high catheter, and followed by a simple or turpentine enema half an hour later.

In most cases these measures prove successful and nothing further need be done. Above all, no attempt should be made to force the intestine into activity during the first twenty-four hours. Purges, pituitrin, and eserine should be absolutely forbidden.

*Management of Established Obstruction.* If after twenty-four hours the above measures fail to give relief, the case must be regarded as an *established obstruction* and more energetic treatment adopted. Duodenal drainage, heat, and intravenous or subcutaneous saline are continued, but care must be taken not to overload the body with fluid, by checking the intake with the output.

Unless it is obvious that the obstruction is wholly mechanical, the case should be treated in the first place as an established *paralytic ileus*; our object being to encourage the return of peristalsis, either by direct action on the intrinsic neuro-muscular mechanism or by abolishing the inhibiting control of the sympathetic. For these purposes three measures of proved efficiency are available: (i) drugs, (ii) hypertonic saline, (iii) spinal anaesthesia.

*Drugs.* Apart from the ordinary cathartics, four drugs exert a stimulating action on intestinal movements, namely, pituitrin, eserine, morphine, and acetyl-choline.

Of these, *pituitrin* and *eserine* (physostigmine) are the least dependable, and prove of little real value in the treatment of paralytic ileus. In experiments on the *human ileum*, I have shown that they both cause areas of sustained local spasm, without any appreciable increase of

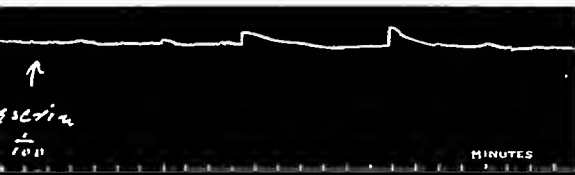


Fig. 586.—TRACING SHOWING EFFECT OF HUMAN ILEUM ON INTRAMUSCULAR INJECTION OF ESERIN.  
(AUTHOR'S EXPERIMENT.)

peristalsis (fig. 586). Moreover, according to Ochsner and Gage (12) they finally produce an after-relaxation of the intestine which more than neutralises any stimulating property they may possess. On the other hand, especially in the case of pituitrin, a very definite increase of peristalsis is produced in the *large gut*, and the drug may be of real service in the treatment of colonic stasis.

**Morphine.** Contrary to what might be expected, and is generally taught, *morphine* acts as a definite stimulus to peristalsis (see figs. 587 and 588). This action is shown particularly on paralysed or denervated intestine, and there is abundant clinical evidence of its beneficial effect on paralytic ileus. At first it was thought that this effect could be attributed to the mental and physical rest induced by the drug, but we now have ample experimental proof that morphine, in small doses, actually increases the tone of, and initiates peristalsis in, paralysed intestine (McIver, 13). I have been able to show that this stimulating property is also exerted on the healthy human ileum (figs. 588 and 589).

It is difficult to speak too highly of the value of this drug in the treatment of paralytic ileus. Its administration should always be well

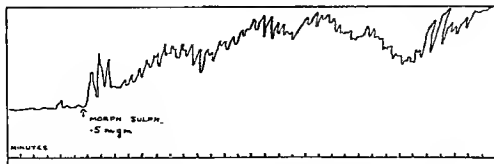


Fig. 587.—EFFECT OF MORPHINE (HYPODERMICALLY) ON INTESTINE OF DOG, SHOWING INCREASE IN MUSCULAR TONE AND PERISTALSIS. (After Plant and Miller.)

controlled, and only small doses, e.g.  $\frac{1}{4}$  gr. six-hourly, should be given, since they are less likely to mask the symptoms of a mechanical obstruction. Morphine is invaluable in *peritonitic ileus*, where,

in addition to its stimulating action on peristalsis, it supplies much-needed rest and sleep, and allays mental and physical distress.

*Acetyl-choline.* In an article published a few months ago, Lawrence Abel (1) brings forward convincing evidence that acetyl-choline is a powerful peristaltic stimulant, and claims that it is practically a specific in its curative action on paralytic ileus. That acetyl-choline does increase peristalsis I have proved conclusively by experiments on a coil of human ileum, which was isolated (bilaterally excluded) in the course of treatment

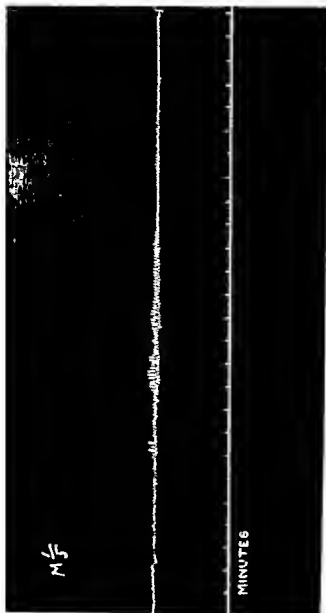


Fig. 588.—EFFECT OF HYPODERMIC INJECTION OF MORPHINE ( $\frac{1}{4}$  GR.) ON HUMAN ILEUM.  
TRACING OF SLOW DATA. AUTHOR'S EXPERIMENT.

of a case of strangulated hernia, which is recorded on page 1064. A small balloon was inserted into the coil and connected to a tambour, the movements of the intestine being recorded on a clock-work drum. Figure 590 shows the result of one of these experiments, 0.1 gm. of freshly dissolved acetyl-choline being injected intramuscularly at the point marked with an arrow; the tracing demonstrates a



well-marked increase of peristaltic movements, starting about 10 minutes after the injection, reaching its maximum after 20 minutes, and then subsiding rather rapidly to normal. Figure 591 is a record of a similar experiment, in which a more rapid drum was used.

In actual cases of paralytic ileus, however, my experience with acetyl-choline has been less satisfactory than Ahel's. It is given in 0.1 gm. doses by intramuscular injection, hourly or two-hourly, until flatus or fæces are passed, or up to six doses. In desperate cases larger amounts can be injected. We must admit that this substance *does* evoke peristalsis, often after only one or two injections; but, as might be expected from the results of the above experiments, this effect is very *temporary*, and the intestine relapses into inactivity after it has worn off.

More experience is undoubtedly needed before the true value of this drug can be properly assessed, but it is certainly worthy of trial when other measures fail and before operation is resorted to.



Fig. 580.—EFFECT OF MORPHINE ON HUMAN ILEUM. TRACING ON RAPID DRUM. (AUTHOR'S EXPERIMENT.)



Fig. 50A.—Effect of ACETYLCHOLINE (0.1 gm.) on HUMAN ILEUM. (ACTION'S KINETIC.)



Fig. 50B.—Effect of ACETYLCHOLINE (0.2 gm.) on HUMAN ILEUM. (ACTION'S KINETIC.)

*Hypertonic Saline.* Hypertonic saline increases intestinal movements when directly applied to the muscular wall (fig. 592), and also when given *intravenously*.

Figure 593 shows a tracing obtained after an intravenous injection of 15 cc. of 25 per cent saline, during the experiments on the human ileum already referred to; it will be seen that the stimulating effect on peristalsis is more powerful than that obtained with acetylcholine (fig. 590), also that its action is much more rapid.

The *clinical results* of hypertonic saline are undoubtedly superior to those of drugs, and it should be administered in all cases of paralytic ileus which persist after the first twenty-four hours. Not only does this stimulate peristalsis, but it also replaces the chlorides of the depleted plasma, and combats the alkalosis which is often present (see page 983). An effective dosage is 500 cc. of 10 per cent solution, which should be run in very slowly. If during the injection colicky pains occur, and no faeces or flatus pass, a mechanical obstruction should be diagnosed.

Recently, Ochsner and Gage (12) have shown that hypertonic Ringer's solution (2.5 per cent of 20 N Ringer per kilo

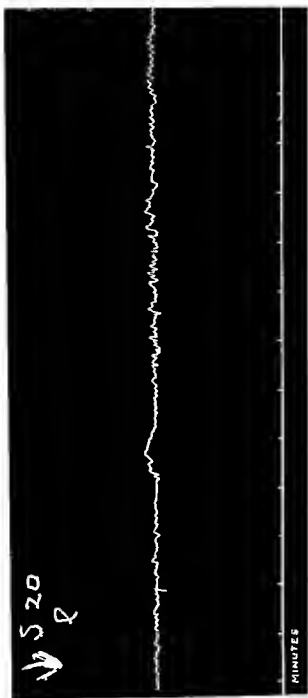


FIG. 593.—EFFECT OF HYPERTONIC SALINE APPLIED LOCALLY TO HUMAN ILEUM. (AUTHOR'S EXPERIMENT.)

of weight) stimulates peristalsis more than does hypertonic saline, and that hypertonic Hartmann's solution gives the best results of all (fig. 594).

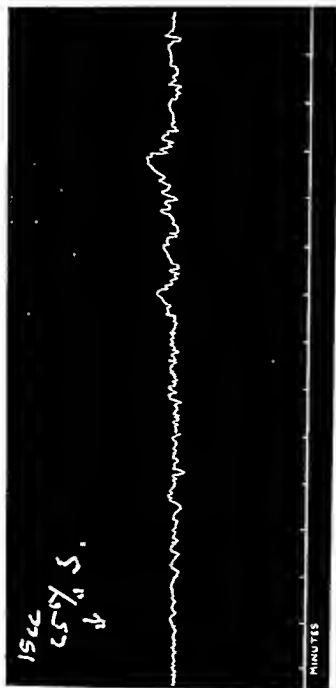


FIG. 594.—TRACING SHOWING EFFECT OF INTRAVENOUS HYPERTONIC SALINE IN ILEUM. (AUTHOR'S EXPERIMENT.)

*Enemata.* The first enema (simple or turpentine), preceded by an injection of ox-bile, is given at the end of the first twenty-four hours (see above). This should be repeated at intervals of twelve hours, each enema being timed to follow an injection of acetylcholine or intravenous hypertonic saline. If the obstruction persists at the end of the second twenty-four hours, the outlook becomes very serious, and we have to resort to our last line of defence, namely, spinal anaesthesia and operation.

*Spinal Anaesthesia.* In paralytic ileus caused

by sympathetic irritation, the effect of *spinal anaesthesia* eclipses that of every other remedy. Unless gross and prolonged distension has already damaged the intrinsic neuro-muscular mechanism, obstruction from this cause is almost bound to yield when a spinal anaesthetic is

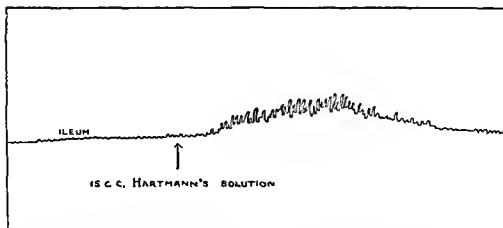


Fig. 594.—TRACING SHOWING EFFECT OF INTRAVENOUS ADMINISTRATION OF HYPERTONIC HARTMANN'S SOLUTION ON ADYNAMIC INTESTINE (After Ochsner and Gage.)

administered, since this completely frees Auerbach's plexus from the inhibitory influence of the stimulated sympathetic. The effect is often dramatic, a copious motion and much flatus being passed within a few minutes.

Actually, *splanchnic anaesthesia* (fig. 595) produces a more direct and powerful action on the sympathetic, and has the added advantage of causing a lesser fall of blood-pressure. The technical difficulty of obtaining a successful splanchnic infiltration, without opening the abdomen, however, renders this procedure frequently impracticable, while the results of the simpler spinal route are satisfactory enough to make it the method of choice. Ephedrine must never be given to counter the fall of blood-pressure, since this drug nullifies the effect of spinal anaesthesia by stimulating the sympathetic nerve-endings; instead, an intravenous saline may be administered with the spinal, if the blood-pressure shows signs of falling to a dangerous level.

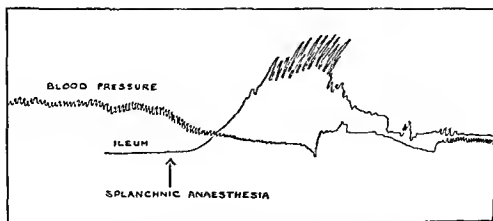


Fig. 595.—TRACING SHOWING STIMULATING EFFECT OF SPANCHNIC ANALGESIA ON PARALYTIC GUT OF DOG, WITH SIMULTANEOUS FALL OF BLOOD-PRESSURE. (After Ochsner and Gage.)

If no action occurs in ten or fifteen minutes, either the obstruction is mainly mechanical, or the intestine is so damaged by "intramural strangulation" that it is past spontaneous recovery. In both events the patient's only hope lies in operation.

There is an unfortunate tendency to regard spinal anaesthesia as "a last resource," and to use it only after everything else has been tried. Indeed, one rarely hears of its employment before the fourth day of a post-operative ileus, by which time irrevocable damage may have been suffered by the intestine, or the blood-pressure may have fallen so much that the procedure is likely to prove fatal. There is no reason why the spinal injection should not be given as soon as it becomes obvious that the ileus will not yield to hypertonic saline and enemata; it should certainly never be postponed beyond the end of the second day. Some people recommend a fractional dose, e.g. three-fifths of a full anaesthetic dose, but I prefer the full dose, as it may be necessary to proceed to operation at once.

#### OPERATIVE TREATMENT

Operation for post-operative obstruction should not be undertaken until a reasonable attempt has been made to overcome it by other means. On the other hand, there is no justification for delay once it becomes clear that the ileus will not yield to conservative treatment. The rational thing to do is to give a spinal anaesthetic and, if this fails to produce an action in ten or fifteen minutes, to proceed at once to operation.

In *early cases* (i.e. second or third day of obstructive symptoms) I feel certain that it is proper to *re-open the original wound*, whether the ileus is paralytic or obstructive. The operation-site is inspected and obvious adhesions are carefully freed; too much time should not be expended over this procedure, and any denuded areas must be covered with peritoneum. Post-operative peritonitic effusion is sucked out and drained.

It now remains to decide whether or not to drain the distended gut by *enterostomy*. The modern tendency is to do an enterostomy whenever the abdomen is opened for post-operative obstruction, and whether the ileus is paralytic or mechanical. This certainly seems rational. In paralytic cases it relieves the gross distension and intramural strangulation of the intestine, and thus restores its blood supply and motility; while in mechanical obstructions the decompression of the distended gut should help in straightening out kinks and releasing

valvular spurs. It is probably wise to perform the enterostomy through a separate stab wound near the main incision. This usually means a low ileostomy, since most operative obstructions involve the lower ileum. The upper jejunum is already drained by the *duodenal catheter*, which should always be left in. When extreme distension is present, it may be wise to drain the intestine in two or more places.

In late desperately-ill cases re-opening of the operation wound, and all procedures of the radical type, are practically certain to end fatally. A "blind" enterostomy, through a small left para-umbilical incision, bringing out and draining the first distended loop which presents, is the only measure which gives a chance of survival. Unless a spinal anaesthetic has been given, this operation should always be performed under local anaesthesia only.

The great drawback in connection with enterostomy is that it does not always succeed in its object, which is to drain the obstructed intestine. Every surgeon of experience knows that after the first gush of faecal contents, the enterostomy often becomes quite "idle." Sometimes this is due to blockage of the catheter, and constant attention will be needed to prevent this, by syringing it through with hot saline at frequent intervals. But more often the explanation is that, although the outlet may be patent, the intestine had not enough tone and motility to take advantage of it. Therefore, every effort must be made to stimulate peristalsis after enterostomy, by local heat, acetylcholine, or, best of all, hypertonic saline. Furthermore, duodenal drainage must be continued, and dehydration prevented by adequate subcutaneous or intravenous fluid, until peristalsis becomes established and oral feeding can be commenced.

*Summary of Treatment.* The above measures are summarised chronologically for the reader's convenience, without insisting on a rigid observance of the order or details of the list. In the case of an obvious mechanical obstruction a similar routine may be followed, except, of course, that peristaltic stimulants must be omitted.

*First 24 hours :*

- (1) Nothing by mouth.
- (2) Intravenous glucose-saline or Hartmann's solution; subcutaneous saline.
- (3) Duodenal or gastric drainage by nasal or oral catheter.

- (4) Continuous heat to abdomen.
- (5) Flatus tube or stretching of anal sphincter.
- (6) Small infrequent doses of morphine or heroin.

*End of first 24 hours :*

Ox-bile enema (6 ounces) followed by simple or turpentine enema.

*Second 24 hours (if obstruction persists) :*

- (1) Continue intravenous or subcutaneous fluid.
- (2) Continue duodenal or gastric drainage.
- (3) Continue heat to abdomen.
- (4) Morphine,  $\frac{1}{2}$  gr. six-hourly.  
Try these measures for 12 hours ; if no result :
- (5) Give slow intravenous hypertonic saline (500 cc. of 10 per cent), and follow with ox-bile and then turpentine enema.
- (6) If this fails, try acetyl-choline, 0.1 gm. hourly, up to six doses.  
(5 and 6 may be reversed, at the surgeon's choice).

*End of second 24 hours (if obstruction still persists) :*

Give spinal anæsthetic ; if no action in 15 minutes, operate.

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## CHAPTER IV

### PERITONITIS

#### GENERAL DISCUSSION

*In the vast majority of cases peritonitis is a complication of a pre-existing abdominal disease, which is usually an infective or ulcerative lesion of one of the hollow viscera. Bacteria may also invade the peritoneum through a penetrating injury, or after traumatic rupture of an abdominal or pelvic organ. Very rarely, and then mainly in children, peritonitis occurs for no apparent reason, and we call it primary; but even here the infecting organisms are brought to the peritoneum from another focus, either by the blood or lymph stream, or via the genital tract.*

Peritonitis may be *diffuse* or *localised*, and both types can occur in *acute* and *chronic* forms.

*Acute diffuse peritonitis* is one of the most dreaded and fatal of human diseases, being the terminal event in most acute abdominal emergencies, and also in many of the more chronic lesions. Timely surgical intervention, aimed primarily at the source of infection, saves a large proportion of cases, but the mortality of cases treated less promptly is extremely high. Sometimes death occurs rapidly, i.e. within 24 or 48 hours, the lethal factor being an overwhelming *bacterial toxæmia* or *septicæmia*. Other cases survive the initial toxæmia, only to succumb some days later to the effects of a widespread *paralytic ileus*.

The vital necessity for early surgical attack on the causal lesion has been recognised for many years, with the result that the incidence and mortality of diffuse peritonitis are appreciably lower than they were. But two equally important therapeutic principles are of more recent development, namely, an energetic combat against the peritonitic toxæmia, and intelligent treatment of the paralytic or adhesive ileus which tends to complicate all but the most fulminating cases.

*Acute localised peritonitis* is mostly a protective process, which tends to limit an infective abdominal lesion to its site of origin, and then to

resolve with the subsidence of the visceral infection. At times, however, this resolution is incomplete and terminates in suppuration, with the production of an *intra-peritoneal abscess*. Although far less fatal than diffuse peritonitis, the localised variety is not without danger, particularly if it implicates one or more of the compartments of the sub-diaphragmatic space. Local suppurative peritonitis may also be encountered as a *residual abscess*, which has been left behind after the subsidence of a diffuse peritoneal infection.

While the essential step in the treatment of diffuse peritonitis is to remove the cause of infection, the chief part of the treatment of a local intra-abdominal abscess is to *let out the pus* and provide *drainage*. Latterly, there has been a tendency to delay the opening of appendix and other abdominal abscesses, in the hope of their becoming absorbed, or of their localising still further. In our view this is a dangerous doctrine; apart from certain very definite exceptions, we believe that a peritoneal abscess should be opened and drained as soon as it is diagnosed.

*Chronic diffuse peritonitis* is most commonly tuberculous, but occasionally it is caused by pneumococcal, gonococcal, and ordinary septic infection. The condition is not immediately fatal, but death may be the ultimate result of general systemic infection, whilst some cases terminate with intestinal obstruction by adhesions, or from exhaustion following a prolonged illness.

*Chronic local peritonitis* is rarely a suppurative process, but it frequently leads to the development of fibrous adhesions or bands, which may eventually cause a fatal intestinal obstruction or strangulation.

This article will be devoted to a consideration of the various kinds of diffuse or generalised peritonitis. Local abdominal abscesses are dealt with on page 1142.

#### THE PERITONEUM: ITS ANATOMY AND FUNCTIONS

The peritoneum is a thin serous membrane which lines the abdominal and pelvic walls (*parietal layer*), and is reflected from them to provide coverings for the viscera (*visceral layer*). The double-layered reflections attaching the viscera to the parietes, or to one another, are known as mesenteries, omenta, or ligaments, the terms varying with the viscus.

The peritoneal membrane itself is perfectly smooth, and consists of a layer of mesoblastic endothelium, resting on a thin layer of connective tissue. Immediately outside the peritoneum is the extra-

peritoneal areolar connective tissue, which in several places is loaded with fat.

Between the parietal and visceral layers is a *potential space*, occupied by a small amount of lubricating serous fluid. Originally a part of the very simple embryonic coelom, this peritoneal "cavity" becomes, in the process of development, extremely complicated, by the invagination into it of the numerous derivatives of the primitive gut, and of the

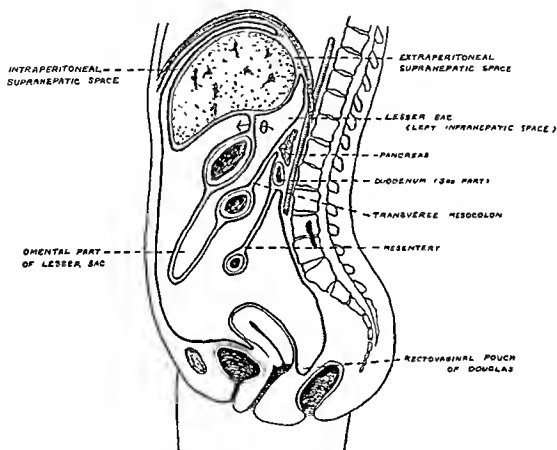


Fig. 596.—DIAGRAM SHOWING MAIN PERITONEAL SPACES.

organs which grow from the "anlage" of the genito-urinary system. In the male the peritoneal cavity is a closed sac, but in the female it communicates via the ostium of the Fallopian tube with the uterus and vagina, and so with the exterior.

Anatomically, the peritoneum forms two main compartments, the *greater* and *lesser sacs* (fig. 596), communicating through the foramen of Winslow, which lies in front of the inferior vena cava, behind the portal vein, and below the neck of the gall-bladder. The *lesser sac* is situated behind the stomach and lesser omentum, and extends upwards beneath the liver, and downwards into the great omentum (see fig. 596);

the omental portion is often obliterated by adhesions. The foramen of Winslow rapidly closes when an inflammatory lesion is present in its vicinity, with the result that the lesser sac becomes completely shut off from the greater.

For the purposes of clinical surgery, the *greater sac* may be divided into regions or compartments, which are normally in free communication, but which under pathological conditions become isolated by the adhesion of neighbouring peritoneal surfaces. Peritonitic effusions tend to gravitate or accumulate in these compartments, with the production of localised abdominal abscesses.

The main compartments are five in number, namely, the sub-phrenic, the right lumbar, the left lumbar, the central, and the pelvic.

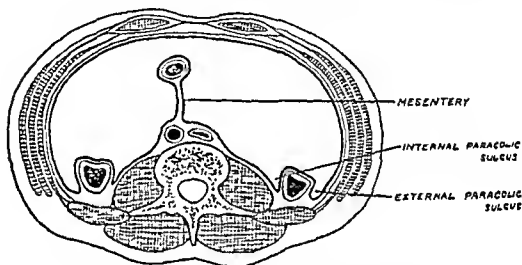


Fig. 597.—HORIZONTAL SECTION THROUGH ABDOMEN, SHOWING PARACOLIC SULCI.

In the supine position, the *lumbar* (paravertebral) *fossæ* and the *pelvis* form three deep cavities, which are accentuated by the forward projection of the lumbar vertebræ, and by the projecting pelvic brim (the abdominal watersheds of Box), and into which free effusions tend to gravitate. The two *lumbar fossæ* are subdivided by the ascending and descending colon into internal and external *paracolic sulci* (see figs. 597 and 598). The *pelvic cavity* contains several peritoneal pouches, differing somewhat with the sex, and produced by the projection into the cavity of the pelvic viscera. The most dependent of these pouches (therefore, the one into which pelvic effusions tend to accumulate) is known as the pouch of Douglas (recto-vesical in the male, and recto-vaginal in the female).

The central compartment lies in front of the lumbar vertebræ and

contains most of the small intestine. It is shut off from the subphrenic region by the shelf-like transverse colon and mesocolon (fig. 598), and

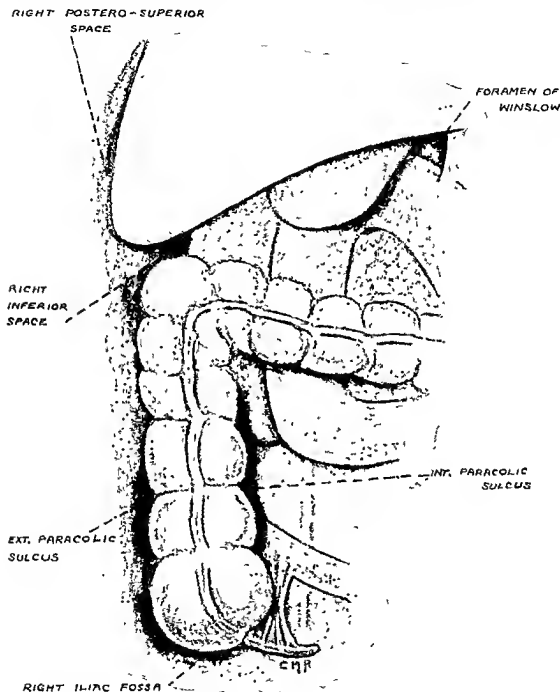


Fig. 538.—PERITONEAL SPACES IN RIGHT HALF OF ABDOMEN.

by the apron-like great omentum which comes down from the former. Laterally, it is in wide communication with the internal paracolic sulci of the lumbar fossæ, whilst below it opens into the pelvis. Obliquely bisecting the central compartment is the mesentery, which is attached

to the posterior abdominal wall along an oblique line, starting to the left of the second lumbar vertebrae, and ending in front of the right sacro-iliac synchondrosis.

The *subphrenic compartment* is a many-chambered potential space, above the transverse colon and mesocolon, and below the diaphragm. It opens into the right and left lumbar fossae at the cephalic ends of the corresponding external paracolic sulci (fig. 598); the communication is somewhat restricted, especially on the left side, by the phrenico-colic ligaments, and may be completely closed by inflammatory adhesions.

The somewhat complicated anatomy of the subphrenic region will be considered more fully in the article on subphrenic abscess (see page 1154). It may be pointed out briefly, however, that the main space is divided into supra- and infra-hepatic compartments, and that each of these is subdivided into right and left. The *right infra-hepatic compartment*, also known as the "*right renal pouch*," is a region of the greatest possible surgical importance (see fig. 598). It communicates with the right iliac fossa via the right external paracolic sulcus, with the right supra-hepatic space round the free border of the liver, with the lesser sac through the foramen of Winslow, and includes among its boundaries the pylorus, duodenum, gall-bladder, liver, and hepatic flexure of the colon.

**Functions of Peritoneum.** The peritoneum ensheathes and separates the viscera, suspends them to the abdominal parietes, carries to them their blood-vessels, lymphatics and nerves, and provides a smooth and lubricated medium, which permits intestinal peristalsis and other visceral movements to occur without interference or friction.

Moreover, it provides a *defence-mechanism* of astonishing efficiency against the results of injury or infection. Its thinness and its huge serous surface (said to equal the total area of the surface of the body) ensure a rapid protective reaction, both local and general, whatever the site of infection or injury, while the endothelial cells, which line it without interruption, are valuable allies to the extravasated leucocytes in the cellular part of the inflammatory process.

Unfortunately, the enormous serous surface is *intensely absorptive*. Fluids and minute solid particles (e.g. bacteria) are absorbed mainly by the peritoneal blood-vessels (Bolton), while larger particles are removed mostly by the lymphatics; both absorptive processes are increased by respiratory or intestinal movements, and also by the raising of the intra-abdominal pressure.

Free absorption of bacteria and their toxins is largely responsible

for the fatal results of peritonitis, especially when the infection is extended rapidly to involve the whole or most of the peritoneal cavity. More often, however, the peritoneum resists the spread of infection, by localising the mischief to a limited area ; this function of *localisation* depends on the production by the inflamed peritoneum of a coagulable *fibrinous exudate*, which glues together adjacent peritoneal surfaces, and so produces a barrier to the further spread of infection.

The *omentum* plays an important part in the defence against peritonitis. It is a protective curtain, richly vascular, with numerous mobile fringes along its free edge. Covering, or coming into contact with the bulk of the viscera in the central and lumbar compartments, it is always ready to participate in the reaction against trauma or infection, and, by adhering to a perforation or an inflamed viscus, to limit the diffusion of septic material, and to localise suppurative processes to their site of origin. Furthermore, by becoming adherent to the abdominal wall, it may completely isolate the upper half of the abdomen from the lower, thus limiting a gross infection to one or other region. A relatively unimportant function of the omentum is to act as a storehouse for fat.

*Nerve supply.* The peritoneum is supplied by the nerves which innervate the muscular wall of the abdomen, i.e. the lower six thoracic nerves and, to a small extent, the fifth thoracic. Whilst the visceral layer is probably insensitive to any form of stimulation (Cushing), the parietal layer is sensitive to pressure, traction, and other forms of gross *trauma*. Such trauma to the parietal peritoneum, and particularly to the mesentery, is productive of severe *shock*.

The segmental similarity of the nerve supply of the peritoneum and abdominal wall explains the muscular rigidity and superficial tenderness of peritonitis ; both phenomena come under the heading of segmental *reflexes* (see page 990).

*Lymph drainage.* For many years it was thought that absorption by the peritoneum occurred more readily and more rapidly in the upper regions of the abdomen than in the lower ; in fact, this was put forward as an explanation of the well-known fact that infections of the sub-diaphragmatic area have a higher mortality than those of the lower abdomen and pelvis. It has even been reported that actual spaces, or "stomata," exist between the endothelial cells of the peritoneum covering the diaphragm, through which a direct communication occurs between the peritoneal cavity and the diaphragmatic lymphatics.

Actually, no such stomata exist, neither is there any reliable evidence to suggest that the main path of toxic absorption from the peritoneum is by the lymphatics. It has, in fact, been shown that the lymphatics become rapidly occluded by a participation in the inflammatory process, and thus they very soon cease to play any part in the process of toxic absorption.

Anatomically, however, there is a close connection between the diaphragmatic peritoneum and the extra-pleural lymph plexus above the diaphragm. Pouch-like lymphatic vessels pass down from the latter plexus, between the muscular and tendinous fibres of the diaphragm, and come into direct relation with minute evaginations of the peritoneum, which also penetrate into the diaphragm. This close contact of the peritoneum and pleura account for the frequency with which infections of the latter complicate suppurative lesions of the upper abdomen.

### ACUTE DIFFUSE PERITONITIS

*Causation.* Acute peritonitis, from the ætiological point of view, may be divided into *primary*, *secondary*, and *post-operative*.

*Primary acute peritonitis* is rare and almost limited to children; there is no discoverable cause in the abdomen, and the infecting organisms are brought to the peritoneum by the blood stream, or possibly via the genital tract or lymphatics. This is an extremely fatal condition, in which a great deal of interest has been shown recently, and to which a separate section is devoted on page 1135.

*Post-operative peritonitis* is unfortunately a common complication of abdominal operations, and is of obvious importance. It is also dealt with in a separate section (see page 1132).

*Secondary peritonitis* is much the most common variety, and will receive consideration here. It may complicate almost any abdominal lesion, whether traumatic, infective, obstructive, ulcerative, or neoplastic.

*Ætiology of Secondary Peritonitis.* Causative lesions may be grouped under the following headings:

(a) *Infective lesions*—particularly appendicitis, cholecystitis, salpingitis, uterine and pelvic sepsis, localised abscess, diverticulitis, and pancreatitis. *Appendicular peritonitis* is by far the most common variety of acute peritoneal infection, and is of sufficient importance to merit a section to itself (see page 1131).



(b) *Acute perforations*—of stomach, duodenum, appendix, gall-bladder, and intestine (e.g. typhoid).

(c) *Ulcerative lesions* (chronic perforations)—e.g. carcinoma of stomach or colon, ulcerative colitis, tuberculous enteritis, etc.

(d) *Obstructive lesions*—late simple obstruction, paralytic ileus, internal strangulation, intussusception, Richter's hernia, etc.

(e) *Traumatic rupture of hollow viscera*—particularly the intestine and urinary bladder. Rupture of *solid organs* (liver, spleen, etc.) may also terminate in peritonitis, owing to infection of the resulting hæmatoma.

(f) *Penetrating wounds of the abdomen*—especially when the alimentary tract is involved.

With the object of discovering the *relative incidence* of the various causes of secondary peritonitis, I examined the records of fatal cases at St. Mary's Hospital, between the years 1920 and 1931, inclusive. The total number of deaths from acute diffuse peritonitis in this period was 94, and the causative lesions are tabulated in Table I.

TABLE I. INCIDENCE OF CAUSES OF FATAL PERITONITIS

(From St. Mary's Hospital records, 1920-1931)

Total number of cases—94.

Appendicitis . . . . .	40 cases (42.6 per cent)
Acute Perforations . . . . .	20 cases (21.3 per cent)
(a) Gastric . . . . .	11
(b) Duodenal . . . . .	8
(c) Gall-bladder . . . . .	1
Carcinoma . . . . .	8 cases (8.5 per cent)
(a) Colon . . . . .	5
(b) Rectum . . . . .	2
(c) Stomach . . . . .	1
Intestinal Obstruction . . . . .	6 cases (6.4 per cent)
Pelvic Lesions . . . . .	6 cases (6.4 per cent)
(a) Salpingitis . . . . .	3
(b) Septic abortion . . . . .	1
(c) Ruptured ectopic . . . . .	1
(d) Suppurating ovarian cyst . . . . .	1
Subphrenic Abscess . . . . .	4 cases (4.2 per cent)
Rupture of Intestine (traumatic) . . . . .	2 cases (2.1 per cent)
Primary Peritonitis . . . . .	2 cases (2.1 per cent)
Other Causes . . . . .	6 cases (6.4 per cent)
(a) Penetrating wound . . . . .	1
(b) Ulcerative colitis . . . . .	1
(c) Empyema . . . . .	1
(d) Chronic nephritis . . . . .	1
(e) Cause unstated . . . . .	2

**Bacteriology of Peritonitis.** A variety of organisms may be found in the exudate of acute peritonitis, including *B. coli*, *streptococcus*, *staphylococcus*, *pneumococcus*, *gonococcus*, *B. pyocyaneus*, *B. Welchii*, and other anaërobes. More often than not the infection is mixed.

The predominance of *B. coli* in most purulent or sero-purulent effusions is evidence, not only of its frequency as a causal factor of peritonitis, but also of its ability to outgrow other organisms in the peritoneal exudate. It is certainly the most common causal organism in peritonitis of *appendicular* or *intestinal* origin, and it also appears in the later stages of peritonitis from other causes.

The *streptococcus* is responsible for the more fulminating varieties of peritoneal infection, and may occur in pure culture. It is found in about half the cases of *primary peritonitis*, and also in *post-operative* and *puerperal* infections of the peritoneum. Finally, it is the causative organism of the worst types of peritonitis complicating *appendicitis*, *cholecystitis*, and other abdominal infections, and also of the more serious infections which follow *penetrating wounds* of the abdomen.

The *pneumococcus* is found in the remaining half of *primary* cases, and is occasionally seen in peritonitis of *appendicular*, *cholecystitic*, *puerperal*, or *pelvic* origin, and also in the rare cases of peritonitis which complicate *pneumonia* and *empyema*.

The *gonococcus* is mostly encountered in *pelvic peritonitis* complicating *gonorrhœa* in women, but occasionally it is responsible for diffuse infection of the peritoneum.

The *B. Welchii* and allied anaërobes frequently find their way into the peritoneum (e.g. in ruptured or perforated intestine, internal strangulations, late simple obstructions, etc.), but they are soon overshadowed by pyogenic organisms, and especially by the *B. coli*. In occasional cases, however, they seem to play the principal part in the morbid changes, and to be responsible for most of the peritonitic toxæmia.

#### PATHOLOGY

Acute diffuse peritonitis is usually infective from the beginning, but it may start as a reaction to chemical irritation or trauma.

A primarily *irritative* peritonitis is nearly always *sudden* in origin, and may be caused by the escape into the peritoneal cavity of *blood*, *urine*, *bile*, *gastric juice*, or *pancreatic fluid*. The peritoneal inflammation is in the first place non-bacterial, and considerable exudation of serous or sero-fibrinous fluid may occur without demonstrable evidence of infection with pathogenic organisms. Indeed, the reaction

may pass on to the deposition of flakes or masses of *fibrin* ("plastic lymph"), which by its adhesive properties glues adjacent peritoneal surfaces together, and may actually seal perforations, thus tending to limit the extent of the peritonitis before pathogenic bacteria even make an appearance. Sooner or later, however, bacterial invasion occurs, and the peritonitis becomes infective. The results depend partly on the virulence of the infection, but also on the degree of localisation effected (and therefore, on the coagulability or fibrin-content of the exudate) in the stage of aseptic inflammation.

Perhaps the best example of a primarily irritative peritonitis is that caused by perforated gastro-duodenal ulcers, in which the exudate may remain free from pathogenic organisms for several hours. Other causes are intra-peritoneal rupture of the urinary bladder, gall-bladder, bile-ducts, pancreas and gravid tube, and traumatic hæmorrhage from the liver and spleen.

Primarily *infective* peritonitis may be sudden or gradual in onset.

The *sudden type* mostly follows acute *perforation* or *rupture* of the *intestine* or *appendix*, and intra-peritoneal *rupture* of localised *abscesses*. In these conditions septic material escapes and spreads so rapidly that most of the peritoneal cavity becomes soiled in the space of a few hours. Peritonitis from these causes, therefore, tends to be diffuse from the start, since there can be little if any attempt at early localisation. Given an infection of low virulence, however, or a patient with high resistance, some localisation may occur later, the infection being successfully dealt with in the more remote parts of the peritoneal cavity, and the worst mischief confined to the region of greatest soiling.

The *gradual type* of peritonitis mostly results from extension of a local infective process, such as *appendicitis*, *cholecystitis*, or *salpingitis*, without perforation. Here, bacterial invasion is preceded by a protective inflammation of the serous membrane in the vicinity of the lesion, with the production of fibrinous adhesions which localise the mischief from the start. With a virulent infection, or a non-resistant patient, however, the localisation may be so imperfect that it fails to arrest the development of a diffuse peritonitis.

*Morbid Changes.* The morbid changes in the peritoneum are too well known to need more than passing mention. The membrane becomes hyperæmic and swollen, and an *exudation* of fluid occurs into the cavity. At first serous and clear, this fluid contains a variable amount of *fibrin*, on which depends its coagulable and adhesive properties. Later the exudate becomes increasingly turbid (sero-purulent

and finally purulent), owing to the accumulation of phagocytic cells; these comprise polymorphonuclear and eosinophil leucocytes, and endothelial cells. The *phagocytes* collect in particularly large numbers on the surface of the great omentum. In cases which recover, the peritoneal exudate becomes rich in anti-bodies.

*Free gas* in large quantities is often encountered in perforations of the stomach or intestine. In small amounts it is sometimes found in localised abscesses, where it is manufactured by gas-forming organisms.

The exudate of peritonitis, although it helps by attacking bacteria and their toxins, unfortunately becomes distributed over the peritoneal cavity, and so tends to disseminate the infection, unless localisation has already occurred. Infection can also be spread directly along the serous membrane, or by the peritoneal and extra-peritoneal lymphatics.

In the event of survival, *resolution* of the infection occurs by absorption of the exudate. Fibrin may also be absorbed, after phagocytic digestion, or it may be organised into *fibrous adhesions*. The worst type of adhesions are formed when parts of the peritoneal membrane have been completely destroyed by the inflammatory process, the granulation tissue which develops in the stage of repair giving rise to dense fibrous tissue. Intestinal obstruction may be produced by both fibrinous and fibrous adhesions, but while the first variety causes obstruction in a few days, the second may do so after an interval of years.

Not infrequently resolution is imperfect, and a localised *inflammatory mass* or tumour results. This consists of the inflamed viscus, perhaps with one or more coils of intestine, and portions of swollen omentum and mesentery, matted together by adhesions, which more or less entirely isolate the mass from the general peritoneal cavity. Such a mass may resolve completely, but it may also pass on to the formation of a *localised abscess*.

Occasionally, this failure of resolution in diffuse peritonitis is a widespread phenomenon. Numerous pockets of pus are left, partly shut off by glued coils of intestine or by adhesions, while the peritoneum itself and parts of the intestine remain œdematous, congested, and friable. This condition may continue for weeks, and constitutes what some writers describe as "*chronic septic peritonitis*" (see page 1139). In these cases, a fatal result from chronic toxæmia, or from paralytic or adhesive ileus, is almost inevitable.

In cases of diffuse peritonitis important changes also occur in the *extra-peritoneal tissues*. The vessels become engorged, the lymphatics

are inflamed, and œdema of the areolar tissue is produced by an extravasation of serous fluid into it. The most fatal cases of peritonitis (usually streptococcal) may show little evidence of peritoneal reaction or effusion; instead, the outstanding morbid change appears to be an intense and rapidly spreading *lymphangitis* of the extra-peritoneal lymphatics, not unlike erysipelas of the skin (*fulminating peritonitis*). In such cases the naked-eye findings at operation or post-mortem may be negligible.

*Paralytic Ileus.* Except in these fulminating cases, the most common complication of diffuse peritonitis is paralytic ileus. The great length and complete peritoneal covering of the small intestine exposes it in a marked degree to the inflammatory changes which affect all extra-peritoneal tissues. The intestinal wall becomes congested and œdematous, and the neuro-muscular mechanism responsible for peristalsis may, in consequence, be thrown out of action. Moreover, particularly in the early stages of peritonitis, the extrinsic sympathetic nerves of the intestine are likely to be irritated by congestion or œdema of the mesentery in which they run, and intestinal inhibition may also result from this cause.

"Peritonitic" ileus is thus produced by a combination of local action on the intestine and irritation of the inhibitory sympathetic nerves; there can be no doubt that it plays the major part in the morbidity of many cases of diffuse peritonitis, and is a contributory cause of death in all except the most fulminating ones.

*The Toxæmia of Peritonitis.* According to Maybury and Williams (1) the lethal results of peritonitic ileus depend on the absorption of *B. Welchii* toxin from the stagnant contents of the distended intestine. There can be no question that there is a great increase of *B. Welchii*, and other anaërobæ, in obstructed intestine; and, at first sight, there is a certain resemblance between the clinical picture of the last stage of peritonitis (rapid weak pulse, subnormal temperature, grey pallor, and acute consciousness) and that of severe anaërobic infections. But the claims made by Williams (2) in 1926 with regard to the importance of *B. Welchii* toxæmia in intestinal obstruction, and the curative value of *anti-Welchii serum*, have not been confirmed by other observers. Indeed, very recently Holt (3) brought forward convincing evidence that the toxic symptoms of peritonitis and obstruction are not caused by a *B. Welchii* toxæmia, but by the absorption of *toxic proteoses* and *amino-acids* from the obstructed and devitalised intestine and

furthermore that *anti-Welchii serum* is valueless in the treatment of these conditions.

This and allied questions are discussed more fully in the articles on intestinal obstruction and paralytic ileus, to which the reader is referred for further details.

Although the source of the intestinal toxæmia is probably non-bacterial, it is quite certain that the immediate cause of death in many cases of diffuse peritonitis is a *bacterial toxæmia*, the toxins being absorbed directly from the peritoneal cavity. In some cases (e.g. perforative appendicitis, or rupture of obstructed intestine) it seems probable that the toxæmia is in part anaërobic, owing to the escape of large numbers of *B. Welchii* and other anaërobics into the peritoneal cavity; but far more often the organisms responsible for the toxæmia are the *pyogenic cocci* and *bacilli* commonly found in peritonic exudate. The intensity of the toxæmia varies with the virulence of the organisms, the resistance of the patient, and the success or failure of attempts at localisation.

The *streptococcus* is the most virulent of the organisms which attack the peritoneum, and has been the cause of nearly all the cases of fulminating peritonitis seen by the writer. Probably more often than is realised, the actual cause of death is a *septicæmia* rather than a pyogenic toxæmia.

#### DIAGNOSIS

A typical case of diffuse peritonitis presents an unmistakable clinical picture, but the onset and course are both variable.

The *onset is sudden* when the cause is a *perforation* or *rupture* of a viscus, such as the stomach, intestine, or bladder; the initial symptoms come on abruptly and constitute the well-known triad of extreme peritoneal irritation or "peritonism" (agonising pain, severe shock, and initial vomiting). After a brief interval of partial recovery (I have known a patient drive a car a hundred miles during such an "interval"), the characteristic symptoms and signs of diffuse peritonitis set in.

The *onset is more gradual* in peritonitis caused by *non-perforative* infective lesions, such as appendicitis or cholecystitis: the symptoms and signs of the causative lesion may merge imperceptibly into those of the first stage of peritonitis.

In most cases an attack of peritonitis is clearly *secondary*, the condition responsible for it being either quite obvious (e.g. penetrating wound, puerperal infection, recent operation), or easily discoverable on examination (e.g. appendicitis, salpingitis, gastro-duodenal ulceration)

In others there may be no clinical evidence of a causative lesion, but this is found at operation or post-mortem. Finally, a few cases remain in which no obvious local cause can be found: these are the cases of *primary peritonitis* (see page 1135).

The course of peritonitis is very variable. A minority of cases succumb within 24 or 48 hours to an intense pyogenic toxæmia or septicæmia: these are examples of *fulminating peritonitis* and are usually streptococcal in origin. At operation or post-mortem, little may be found beyond œdema and lymphangitis of the peritoneum and extra-peritoneal tissues. A larger number of cases run a less rapid course, but the infection spreads to the greater part of the peritoneal cavity; a definite reaction occurs, with profuse effusion, but death is likely to result from toxæmia and paralytic ileus, between the third and seventh day. These are examples of what we might term *acute spreading peritonitis*.

Perhaps the largest group of cases are those in which, after a short stage of diffuse peritoneal inflammation, the mischief tends to become *localised*, either to the vicinity of the causative lesion, or to one of the dependent or "blind" compartments of the peritoneal cavity. This is obviously the most favourable variety, to which the term *acute localising peritonitis* may well be given. The localised infection may either resolve completely, or it may terminate in a "residual" abscess.

#### TYPICAL SYMPTOMS AND SIGNS

(1) *Early Stage.* The *earliest* symptoms are the most important, since the patient's recovery depends on timely diagnosis and operation.

Whether the onset is sudden or gradual, the first symptom is *abdominal pain*. The pain varies in intensity, but is generally severe and constant, with temporary exacerbations produced by coughing, micturition, tension and pressure on the abdominal wall, or any action which disturbs the inflamed peritoneum. Initially the pain may be referred to the navel, or to the region of an inflamed organ, but it soon becomes more generalised; it is usually most intense in that part of the abdominal wall which lies over the spreading edge of the peritoneal inflammation. With the subsidence or localisation of the infection, the pain diminishes, and once again becomes localised.

*Vomiting* occurs at the onset of most cases of peritonitis, and then recurs at intervals. In the early stage only gastric contents are brought up, and if fluids by mouth are discontinued the vomiting may cease. The initial vomiting is reflex in origin; later it is probably mainly toxic, but in the final stages it is attributable to paralytic ileus.

The *pulse* may show little or no change for some hours, although in cases with a sudden onset (e.g. perforated ulcer) it may be somewhat weak from initial shock. After two to four hours, the pulse-rate begins to rise, and the increase progresses steadily to 120-140, or more, but the volume becomes fuller with recovery from the initial shock. This *progressive* increase of the pulse-rate is one of the most valuable symptoms of peritonitis, and a *half-hourly chart* should be used for all suspected cases.

The *temperature* may already be elevated by the causative lesion, but in cases with sudden onset it is often sub-normal at first. A moderate and steady rise is usual after a few hours, but it should be observed that the increase of temperature occurs *after* the rise in the pulse-rate. Diffusion of peritoneal infection usually causes a fall of temperature, particularly in fulminating cases; a rising pulse-rate with a dropping temperature constitutes one of the gravest prognostic features. On the other hand, a rising temperature with a falling pulse strongly suggests localisation of the infection.

*Breathing* soon becomes shallow and thoracic in type. The *tongue* is furred but moist, and the *breath* is heavy. The *bowels* are usually constipated, but in some cases (e.g. pelvic peritonitis or peritonitis in children) there may be diarrhoea.

Although the early symptoms are suggestive, the actual diagnosis of early peritonitis is made on the *physical signs*.

*Inspection* of the hard abdomen shows complete or almost complete *absence of abdominal respiratory movements*. The patient lies mostly on his back, with the knees flexed to relax the abdominal muscles and to keep off the bed-clothes. An important early sign is *retraction* of the abdomen, which in thin patients assumes a characteristic *scaphoid* appearance.

*Palpation* reveals two signs of the utmost value, i.e. tenderness and rigidity. *Tenderness* is the more constant but less reliable of the two signs; at first often localised to the causative focus, it spreads with diffusion of the inflammation, and soon becomes extreme. Two allied signs of great importance are *rebound-tenderness* (pain caused by sudden release of pressure), and pain produced over the affected region by pressure on an uninvolved part of the peritoneum.

*Abdominal rigidity* is the most reliable sign of peritonitis. In sudden cases (e.g. perforated ulcer) the rigidity is *board-like* from the first, and soon becomes generalised. But in more gradual cases (e.g. non-perforative appendicular peritonitis) the rigidity may remain localised for several hours, and only becomes severe and more general when a large area of parietal peritoneum is involved.



*Auscultation* of the abdomen with a stethoscope is a most valuable step in the examination. To the experienced ear, intestinal sounds are diminished almost from the start, and over the area of greatest mischief they may be completely absent. This method of examination is of the greatest possible assistance in distinguishing early peritonitis from mechanical obstruction (see page 993).

*Percussion* of the abdomen gives an increased tympanitic note, owing to early gaseous distension of the intestine. In perforations, sufficient gas may accumulate in the upper part of the peritoneal cavity to obliterate the liver dullness, but this is by no means a constant sign. Later, one may be able to make out shifting dullness in the flanks, owing to collections of copious exudate in the paravertebral hollows, but this again is an inconstant sign.

(2) *Late Stage.* After the first 12-24 hours the picture changes and becomes more "classical," the patient beginning to show signs of *toxæmia* and *paralytic ileus*. Pain continues and is aggravated by the wearing ache of intestinal distension. Vomiting becomes more profuse and regurgitant, and finally assumes the "fecal type." The pulse is rapid and full, but soon begins to weaken. The temperature may be high or sub-normal. The tongue becomes brown and dry, the breath very unpleasant, and the face grey, pinched, and anxious (Hippocratic facies). Constipation is obstinate and absolute, no flatus being passed. The abdomen is distended and tympanitic, and the tenderness and rigidity are generalised; on auscultation, it is usually completely silent. Failure of the circulation (falling blood-pressure, coldness and slight cyanosis of the face and extremities, very rapid and thready pulse), restlessness, and renal failure presage the coming of death, but the mind remains remarkably clear, often to the very end.

*Atypical Cases.* The above picture is so extremely typical that a late case of diffuse peritonitis might almost be said to diagnose itself. In two groups of cases, however, this "typical" stage is never reached: one is the *fulminating type*, in which death occurs from an overwhelming toxæmia or septicæmia before the "classical" symptoms have had time to become established; the other is the *localising type*, in which, after a short stage of early generalised symptoms and signs, most of the abdomen clears up, and an indefinite localised mass begins to be palpable.

In a third group the picture is atypical, not because the peritonitis

is fulminating or localising in character, but because the clinical features are *modified* by pre-existing disease, extremes of age, or by the action of drugs. For instance, a perforation of the intestine in the third week of typhoid, or in a late stage of a mechanical obstruction, may be missed for several days, because the patient is too ill to complain of, or even to appreciate his symptoms, and because the abdominal rigidity and other signs are rendered indefinite by the exhausted condition of the patient. Indeed, the only signs of peritonitis in such cases may be persistent vomiting, rapid pulse, cessation of flatus, and distension of the abdomen. To a lesser extent, the symptoms and signs are modified in extreme infancy and old age, and in puerperal cases. The "masking" effects of *morphine* are too well known for it to be necessary to enlarge upon them; suffice it to say that this drug is a deadly danger in undiagnosed cases.

#### DIFFERENTIAL DIAGNOSIS

This is only difficult in the early, and therefore important stage. In an advanced case the diagnosis presents no difficulties, but the favourable time for surgical intervention has gone.

The "sudden" cases of peritonitis, particularly those caused by gastro-duodenal perforations, must be distinguished from the colics (biliary, renal, or intestinal), and from the abdominal crises of tabes. In most cases the differentiation is easy, the points in favour of peritonitis being a history of an antecedent lesion likely to cause it, a rising pulse-rate, retraction and unyielding rigidity of the abdomen, and tenderness at some distance from the suspected region. Occasionally it may be difficult to reach a decision, but delay is never justified in these "sudden" cases, and any doubt must be settled by immediate laparotomy.

It should be remembered that the discovery of one likely cause of an abdominal crisis does not necessarily exclude another. Thus, I have seen a perforated duodenal ulcer missed in an in-patient, because he happened to be in hospital for tabes; the autopsy provided a complete surprise.

The "gradual" cases of peritonitis are more difficult to diagnose at a really early stage. The first symptoms may be obscured by the clinical features of the causative lesion, or by the weak state of the patient, and the peritoneal mischief may have spread extensively before we become aware of its existence. This applies with particular force to cases of *post-operative peritonitis*, where the gas-pains and meteorism

of post-operative stasis, and the vomiting which follows general anaesthesia, may completely mask a spreading peritoneal infection. Only by being constantly "aware" of the likelihood of peritonitis when a likely cause exists, and by a most careful watch on the case, can we protect the patient against the terribly serious consequences of delayed diagnosis.

The signs are there if we search for them. Continuous abdominal pain, vomiting, and a *rising pulse-rate*, should always suggest peritonitis; while the physical signs to look for are rigidity, spreading tenderness, "rebound" tenderness, pain over the seat of trouble by pressure on another part of the abdomen, and diminution or loss of intestinal sounds on auscultation.

No diagnosis of an acute abdomen is complete without an examination of the *chest*, and of the *urine*. I have on two occasions (both children) avoided at the last moment what could only have proved a harmful laparotomy, by discovering signs of commencing pneumonia, after this had been missed by others. Many cases of pleurisy have an onset similar to that of an acute abdomen. Finally, it would be difficult to estimate how many cases of pyelitis and pyelonephritis have had their abdomen opened, only because an examination of the urine has been omitted; the number must certainly be a very large one.

#### TREATMENT

The *prevention* of peritonitis requires separate and careful consideration. It is hardly necessary to point out that if every acutely inflamed appendix or gall-bladder could be removed at the start of the attack, every gastric and duodenal ulcer healed before perforation, every intestinal operation performed without spillage or leakage, and every obstruction relieved at once, diffuse peritonitis would become a rare disease. Although this ideal is unattainable with any degree of completeness, we can go a long way towards it by advocating and practising the principle of immediate operation in all acute abdominal emergencies, and by carrying out all abdominal operations with the most scrupulous care and asepsis. Prophylaxis is of particular importance in appendicular and post-operative peritonitis, and will receive further consideration under those headings.

The *treatment of diffuse peritonitis* comprises five distinct steps: (1) removing or shutting off the source of infection; (2) cleansing the peritoneum (if necessary); (3) drainage of the infecting focus, or of the peritoneum (if necessary); (4) dealing with the paralytic ileus;

and (5) attacking the peritonitic toxæmia. The first three steps come under the heading of operative treatment, while the last two cover the main part of the after-treatment.

### (1) REMOVAL OR CLOSURE OF SOURCE OF INFECTION

This is the first and most important step in the treatment, and the survival of the patient depends far more on *how soon it is done* than on anything else. In Kirschner's words (4): "The indication is not merely the closure of the source of infection; it should be stated as the *immediate closure of the source of infection.*"

Therefore, all cases in which diffuse peritonitis has been diagnosed, and in which a local cause is known to be present, or there is the least chance of a local cause being present, should be operated upon immediately. The only *exceptions* are cases of gonococcal peritonitis, some cases of post-operative peritonitis (see page 1132), and primary peritonitis in children when a local cause can be excluded (see page 1135); moribund patients must come under the same heading, although even in these it may occasionally be worth while to make a small incision under local anaesthesia and drain away some of the exudate, and perhaps to fix a catheter into a distended loop of intestine.

*Preparation.* With early cases (within 12 hours of onset) special preparation is unnecessary, except where recovery from initial shock is partial; here, one to two pints of intravenous glucose-saline should be administered whilst arrangements are made for the operation. A small dose of morphine ( $\frac{1}{6}$ – $\frac{1}{4}$  gr.) is of great value, as it allays anxiety, secures rest, and prepares the patient for anaesthesia.

In later cases, particularly when paralytic ileus is already present, or vomiting is persistent, a *stomach tube* should always be passed, either by the nasal or oral route, and the contents of the stomach and upper intestine removed by siphonage or aspiration. The tube should be left *in situ*, and will prove of the greatest possible assistance in preventing air-swallowing, relieving intestinal distension, and hastening the recovery of paralysed intestine. All cases operated on more than twelve hours after the onset of peritonitis should be given intravenous fluid; usually normal glucose-saline is sufficient, but desperately ill patients may be given a *blood-transfusion* instead.

*Anæsthetic.* Spinal anaesthesia should be chosen deliberately in all operations for peritonitis, except when the circulation is failing. It

has overwhelming advantages over general anæsthesia, in that it abolishes the inhibitory action of the irritated sympathetic on the intestine (thus tending to diminish paralytic ileus), and also secures excellent relaxation of the abdominal wall (thus facilitating the search for and removal of the source of infection). Chloroform and ether we condemn, because they increase the likelihood of paralytic ileus, and because they are dangerous anæsthetics in the presence of a severe toxæmia. A serious fall of blood-pressure during spinal anæsthesia should be countered by intravenous fluid, or by vasomotor stimulants.

When spinal anæsthesia is unsuitable, e.g. in small children, or in badly shocked patients, we must rely on *local anæsthesia with gas and oxygen*. A preliminary injection of morphine, omnopon and scopolamine, or one of the pre-anæsthetic narcotics in present-day use, usually ensures sufficiently deep anæsthesia to enable all necessary abdominal manipulations to be carried out.

*The Operation.* The *incision* should be one which gives easy access to the cause, whether this is diagnosed with certainty or only suspected. Small incisions are permissible only when we know what to look for. In doubtful cases the best incision is a right paramedian, one-third above and two-thirds below the umbilicus. In the first place it should be about three inches long, but it may be extended upwards or downwards, as the circumstances indicate.

Evisceration must be avoided at all costs, and the greatest gentleness observed in both the search for and treatment of the causative lesion. Nothing should be attempted except what is strictly essential to the patient's immediate recovery, and non-urgent procedures should be left over to a later date. The golden rules in operating for diffuse peritonitis are: (1) do no more than is absolutely necessary; (2) do it gently; (3) do it speedily.

The actual *procedure*, of course, depends on the cause. An inflamed appendix must always be removed when diffuse peritonitis is present. A perforation of the stomach, duodenum, or the rest of the intestine, must be closed, however difficult such closure may prove. Gangrenous intestine should be resected or exteriorised, according to the condition of the patient and the level of the affected segment (see page 1056).

An acutely inflamed or gangrenous gall-bladder is best resected. But if the patient is too ill to stand this, or the gall-bladder is so adherent that its removal entails unjustifiable risks, it may be brought to the surface and drained, at the same time draining the peritoneum in its immediate vicinity.

An abscess opening into the peritoneal cavity must be emptied and drained by a separate incision, which should if possible be extra-peritoneal. Infective foci which cannot be removed or closed (e.g. acute pancreatitis) should also be connected to the exterior by drainage.

It is hardly necessary to warn the surgeon against the danger of pricking his own or his assistant's fingers during operations for peritonitis.

## (2) CLEANSING THE PERITONEUM

It is probably always correct to remove the peritonitic exudate, for although this may be rich in anti-bodies, it is also toxic, and it may act as a culture-medium for bacteria and thus favour re-infection of the peritoneum. The more gently this removal is effected, however, the better it is for the patient.

Of the various cleansing procedures, *irrigation* is the most dangerous, since it may spread the infection to hitherto uninvolved parts of the peritoneum, besides damaging the serous endothelium, and washing off protective deposits of fibrin. Its use is justified only where there is gross contamination of the peritoneal cavity with food or faecal material, in which case other methods of cleansing cannot possibly prove effective. When irrigation is employed, it should be done very gently, normal saline being the only fluid permitted.

Ordinarily, *aspiration* with a sucker, and cautious *sponging* with soft gauze packs, will remove the bulk of the exudate. Collections of pus near the infecting focus or in dependent cavities, such as the pelvis, should be mopped up very completely, and all foreign bodies (e.g. gall-stones, appendicular concretions, fragments of food) must be carefully picked out. On no account may peritoneal surfaces be rubbed or swabbed roughly; this is bound to injure the delicate endothelium, and to peel off protective flakes of lymph.

In general, it can be said that if the infecting focus has been removed, the peritoneum may be trusted to look after itself.

## (3) DRAINAGE

The question of drainage in peritonitis constitutes a much debated problem, and many things have been said and practised which have little foundation in fact. It was customary for many years to insert drains into the pouch of Douglas and other dependent parts of the peritoneal cavity, with the idea that the purulent exudate from the

whole cavity could be drained off in this way ; indeed, some surgeons still adhere to the suprapubic drain in Douglas's pouch, and even leave such drains *in situ* for as long as four or five days, trusting in the adoption of the Fowler position to bring the exudate to the drainage-tube.

We now know that this procedure is of very temporary service, if any, and that it is *impossible to drain the general abdominal cavity*. The peritoneum seals off any opening into it within a few hours, and all that the tube does after this is to drain its own track. Moreover, the general peritoneal cavity is very soon broken up into separate spaces, by the agglutination of neighbouring peritoneal surfaces through the action of the fibrinous exudate.

With regard to drainage of an *infected focus*, or of a region of *localised suppuration*, the position is very different. Drainage here is vitally necessary, since it tends to isolate the dangerous area from the general cavity of the peritoneum, and so to avoid infection or re-infection of the latter. The shape and material of the drains matter little (a simple rubber tube is the best), but our aim should be to drain the dangerous area, as far as possible without trespassing on the general peritoneal cavity.

The present position of enlightened surgical opinion may be summarised as follows :

(a) Drainage of the "peritoneal cavity" is impossible, and therefore should never be attempted. After effective treatment of the cause of the infection, and removal of the peritonitic exudate, the peritoneum can deal with any further exudation without external aid.

(b) Drainage is unnecessary when the infecting focus has been excluded with certainty, and there is no likelihood of the development of other infective foci.

(c) All areas of *local infection*, and all areas of *potential infection*, must be drained. We include under this heading all local abscesses, necrotic processes (e.g. gangrenous gall-bladder, acute pancreatitis, etc.), unsafe closures of perforations, and insecure or doubtful suturing of intestine and other hollow viscera. A cavity with infected walls, e.g. the inflamed, friable, and semi-necrotic bed left behind after removing a purulent second or third day appendix, should always be drained ; if this is not done the case might easily pass on to a localised abscess, or even to a diffuse post-operative peritonitis. Furthermore, an area with persistent oozing of blood (e.g. the cavity left after separating an acutely inflamed gall-bladder) should also be drained, as a local accumulation of blood under such circumstances is likely to become infected.

The object of drainage in all these cases is to place the focus of infection, or of possible infection, in communication with the exterior, and thus to seal off the general peritoneal cavity from it. In some cases it may be wise to assist this process of "sealing off" the peritoneum from an infected area by packing gauze loosely round the drainage-tube.

Another debated question is—when should a drain be removed? If for any reason a tube is inserted with the object of draining the general peritoneal cavity, it should not be left in for more than 24 hours. But there is no object in hurrying the removal of a tube which is draining an actual or potential focus of infection. In fact, the early manipulation or removal of such a drain is definitely dangerous. We should wait until reliable adhesions have walled off the focus and drainage-track (usually a matter of three to five days), and then start to shorten the tube gradually. The practice of removing the tube for cleaning purposes, and then replacing it, is not recommended, but there can be no objection to the replacement of a large drain by a smaller one, once the discharge begins to diminish.

#### (4) PREVENTION AND TREATMENT OF PARALYTIC ILEUS

It must not be imagined that intestinal paralysis is of invariable occurrence in diffuse peritonitis. Cases of the fulminating type, and also the primary peritonitis of children, may go through their entire course without any evidence of obstruction. Furthermore, prompt removal of the source of infection in the more common "secondary" peritonitis may be expected to *prevent* the development of paralytic ileus, certainly in a majority of cases. Much may also be done, in the way of prevention, by insisting upon strict asepsis and the utmost gentleness in all abdominal operations, particularly those performed for peritonitic lesions, and by the avoidance of chloroform and ether anaesthesia.

The fact, however, remains that many cases of diffuse peritonitis develop paralytic ileus, and that this plays an important part in the causation of death. As a rule, we can detect some evidence of intestinal inhibition at, or even before, the operation performed for the peritonitis, but the symptoms and signs of *complete* paralytic ileus usually appear 24 to 48 hours *after* the operation.

For a full discussion of paralytic ileus and other forms of post-operative obstruction the reader is referred to the special article on this subject; in this place we can only consider the pathology and management of the paralytic obstruction of peritonitis.



Peritonitis produces intestinal paralysis, partly by irritating the inhibitory sympathetic nerves, but also by direct interference with the intrinsic neuro-muscular mechanism of peristalsis, owing to inflammatory changes in the gut-wall. We must agree with Rohh (5) that *sympathetic irritation* is the more important of these two morbid processes, and that in the early stage of peritonitic ileus the obstructive symptoms are caused by spasm of the several alimentary sphincters, as well as by paralysis and distension of the intersphincteric segments of the intestine. Paralysis from sympathetic irritation can be overcome, but once the intrinsic mechanism is deranged (as is the case in the late stage of peritonitis) recovery becomes extremely unlikely.

We have already seen that *acetyl-choline* appears to play an essential part in peristalsis (Abel, 6, and others), and it is at least possible that peritonitic ileus may in part be caused by destruction of this substance.

One thing appears fairly certain, and this is that the inhibition of the intestine in the *early stage* of peritonitis is *physiological* and *protective*. The abolition of intestinal movements secures rest to the inflamed peritoneum, and tends to minimise the spread of infection. Therefore, the early stage of sympathetic over-activity should not be disturbed. After the first 24 hours, however, unless the attack is of the fulminating type, some immunity should be acquired by the peritoneum, the exudate becoming purulent, and the worst of the mischief showing some signs of localisation. At this stage intestinal immobilisation no longer serves any beneficial purpose; it is, in fact, definitely injurious, since the increasing paralytic distension of the intestine is bound to cause damage, which is progressive in character, and which may prove irrecoverable. We conclude that in this "*second stage*" of peritonitic ileus our main object should be *to secure the return of peristalsis*.

These pathological considerations form the basis of the modern *treatment* of peritonitic ileus, which may be briefly outlined as follows (for further details see page 1085):

(i) In the *first twenty-four hours* of obstructive symptoms we must abstain from vigorous attempts to stimulate peristalsis, but we can do a good deal towards assisting the ultimate recovery of the intestine and patient. The most helpful measures in this stage are: (a) *gastric* or, preferably, *duodenal drainage*, by the nasal or oral route; (b) *mental and physical rest*, best secured with small doses of morphine; (c) *heat to the abdomen*, by an electrically heated pad, or hot glycerine compresses; (d) *avoidance of oral or rectal fluids*; (e) *prevention of*

*dehydration* and *plasma-depletion* by intravenous glucose-saline or Hartmann's solution, or by subcutaneous saline with periodic feeds of intravenous glucose-saline.

(ii) *After the first twenty-four hours* attempts should be made to nullify the sympathetic activity, and to encourage the return of peristalsis. For reasons already given (see page 1086), pituitrin and eserine are best avoided. *Morphine* should be continued in small doses ( $\frac{1}{6}$  gr. six-hourly), as, contrary to what is usually taught, it has been shown to increase peristalsis in the small intestine. Many surgeons speak well of the stimulating effect of *raw meat juice*, which is best introduced via the duodenal catheter. *Acetyl-choline* is also a proven peristaltic stimulant, and may be given in 0.1 or 0.2 gm. doses hourly, until flatus and fæces are passed; it is undoubtedly effective, but its action tends to be temporary.

After these remedies have been given a chance to act, an *ox-bile enema* (6 ounces) should be administered slowly, and followed by a *simple* or *turpentine enema*, half an hour later. The enema may be repeated, but only at intervals of not less than twelve hours. While these stimulating measures are being tried, duodenal drainage, heat to the abdomen, and intravenous or subcutaneous fluids must be continued. In most cases this treatment should prove successful, the return of peristalsis being heralded by the discovery of intestinal sounds on auscultation, and by the passage of flatus. The return of peristalsis is soon followed by a striking improvement in the patient's condition.

As soon, however, as it becomes obvious that the intestinal paralysis will not yield to these simple measures, and certainly before the end of the second twenty-four hours, we should resort to that most powerful of peristaltic stimulants—*intravenous hypertonic saline*; 500 cc. of 10 per cent saline are run in slowly, and half to one hour later an enema administered. If this fails, we are driven to our last line of defence, namely, *spinal anaesthesia*. It is true that this only abolishes the sympathetic activity, but this activity is usually the chief cause of the ileus. The danger is not that we might employ spinal anaesthesia too soon, but that we might leave it till too late. It should never be delayed later than the end of the second day, as after this the patient may be too collapsed to stand it, or the intestine may be damaged past recovery.

Not many cases of peritonitic ileus will fail to respond to one or other of the above measures, provided they are employed intelligently,

at the right time, and at suitable intervals. Those that do not respond will almost certainly die, whether we resort to a *second operation* or not. Provided the cause of the peritonitis was adequately dealt with at the first operation, we cannot hope to do any good by re-opening the abdomen because the patient is dying from paralytic ileus. *Enterostomy*, under local anæsthesia, may help when the obstruction is partly mechanical, but it is useless to make an opening into intestine which is paralysed beyond recovery.

#### (5) TREATMENT OF PERITONITIC TOXÆMIA

The rapidly fatal course of fulminating streptococcal peritonitis demonstrates, in a dramatic manner, our helplessness before a really virulent peritonitic toxæmia. Fortunately, other and more common varieties of diffuse peritonitis show some response against the infection, and it is in the direction of furthering this response that lies our chief hope of influencing the battle in the patient's favour.

The most vital thing we can do is to cut short the further supply of infecting organisms, by promptly shutting off the source of the infection. That being done, we can assist further by removing the greater part of the toxic exudate, without injuring the peritoneum by a too vigorous exhibition of our zeal. As already shown, we cannot continue to remove the exudate, since it is impossible to drain the peritoneal cavity for more than a very short period.

In the hope of encouraging the collection of the toxic exudate in the lower part of the abdomen, and thus of avoiding infection of the dangerous subphrenic region, the *Fowler position* was advocated, and has been universally employed for many years. While this position is of undoubted service in that it diminishes the tendency to pulmonary collapse and pneumonia, it is very doubtful if it really serves the purpose for which it was originally adopted. In many cases of diffuse peritonitis the peritoneal cavity soon breaks up into more or less separate compartments, by the agglutination of neighbouring serous surfaces, and changes of position cannot affect the distribution of the exudate; moreover, infection spreads mainly by direct extension along the peritoneum, and therefore the sitting posture cannot prevent involvement of the subphrenic region. Nevertheless, in the presence of a really large and free effusion gravity is bound to play an important part, and in such cases the *Fowler position* is of real benefit.

It cannot be doubted that the copious administration of *fluid*, *intravenously* and *subcutaneously*, besides maintaining the water-balance

and the ionic content of the plasma, also serves the useful purpose of diluting toxins; in bad cases of toxæmia a *blood-transfusion*, provided the bloods are absolutely compatible, may prove of even greater value.

The common practice of attempting to disinfect the peritoneal cavity, or to hinder the absorption of toxins from it, by the employment of antiseptics, is worse than useless. Solutions of iodine, quinine, hypertonic saline, Dakin's solution, and ether, are among the substances which have been employed, but they are all likely to do more harm than good.

Recently, Fraser and Walsh (7), of St. Mary's Hospital have, employed finely *emulsified olive oil* (10 per cent emulsion) as an intravenous remedy for severe toxæmia; the results are at least promising, the emulsion acting by absorbing the toxic substances, and thus rendering them innocuous. I have been given to understand by one of these two workers that this emulsion has been given intravenously to at least one case of pneumococcal peritonitis, with a successful outcome. It is certainly worthy of trial in all cases of peritonitis in which toxæmic manifestations predominate. Furthermore, I am hoping to investigate the action of this emulsion on the peritoneum itself; it would be relatively simple to introduce thin tubes of perforated rubber (long Carel tubes) into various parts of the peritoneal cavity, and to administer the emulsion through a funnel or syringe during the actual operation, and perhaps once more after some hours. Anything in the nature of continuous irrigation would be impossible, as the peritoneum rapidly seals itself off from the tubes, but even this temporary instillation of the emulsion may prove of material benefit.

The *bacteriological treatment* of diffuse peritonitis is frankly disappointing. *Anti-streptococcal* and *anti-pneumococcal sera* sometimes seem effective in peritonitis caused solely by these organisms (mostly primary peritonitis of children); but in the common cases of secondary peritonitis (with mixed infection) serological treatment is of little if any use. *Anti-Welchii* serum would be of real service if the *B. Welchii* played an important part in the morbidities of peritonitis, but the results of accumulated experience seem to show that this is rarely the case, and in the hands of most surgeons its use has proved a complete failure. However, I have seen it produce striking results in two cases of gas gangrene of the abdominal wall, after operations for appendicular peritonitis, and I think it is at least worthy of trial when examination of the peritoneal exudate shows the presence of *B. Welchii*.

The use of *bacteriophage* in peritonitis has already been referred to

(see page 1081). I cannot speak of it from experience, but good results are claimed for its use in America (Smith (8)). It is administered by a catheter through the operation wound, or through the drainage-tube; 30 to 60 cc. of autogenous or mixed anti-coli and anti-streptococcus and anti-staphylococcus bacteriophage is given at the operation, and repeated daily for a day or two.

Sufficient time is rarely available for the employment of *vaccines* (either autogenous or stock) in acute diffuse peritonitis, but they might prove useful in the rare cases which become chronic. The *prophylactic* use of vaccines *before operations* incurring the risk of peritonitis (e.g. colonic resections) is based on common sense, and it is undoubtedly responsible for the low incidence of peritoneal sepsis in the hands of some operators.

### SPECIAL VARIETIES OF DIFFUSE PERITONITIS

Four varieties of diffuse peritonitis are of sufficient importance to merit separate consideration. They are: (i) peritonitis caused by *gastric and duodenal perforations*; (ii) *appendicular peritonitis*; (iii) *post-operative peritonitis*, and (iv) *primary peritonitis* in children.

### PERFORATED GASTRO-DUODENAL ULCER

The great majority of perforated ulcers are found near the pylorus, and it makes little difference whether they are on the gastric or duodenal side of this sphincter. It is well known that ulcers which perforate are mostly *chronic*, but we now believe that acute gastric ulcers perforate more often than was thought to be the case a few years ago. In the great bulk of cases the perforation involves the *anterior wall* of the stomach or duodenum, and it is rarely far from the lesser curvature (fig. 599). *Double perforations* are rare, but the possibility of their occurrence should always be borne in mind. A large majority of the patients are *males* (90 per cent of 74 consecutive cases investigated by the writer at St. Mary's Hospital), and the highest *age incidence* is between 30 and 50.

A perforation may be *acute*, *sub-acute* (leaking ulcer), or *chronic* (penetrating ulcer). *Diffuse peritonitis* is likely to be caused only by an acute perforation; the first reaction is produced by the *irritant* effect of the gastric contents. There is an interval of some hours before bacterial invasion occurs, and operations performed during this interval

have a mortality many times smaller than that of operations in the stage of infective peritonitis. Statistics vary enormously, but recent figures show that the death-rate of cases operated on within six hours of the onset is less than 5 per cent, while that of cases operated on after twelve hours approaches 50 per cent. Of no lesion can it be said with greater truth that the patient's life hangs on prompt diagnosis and immediate operation.

Duodenal perforations (fig. 600) have the higher mortality. One reason is that the ulcer is more difficult to get at and to close; but

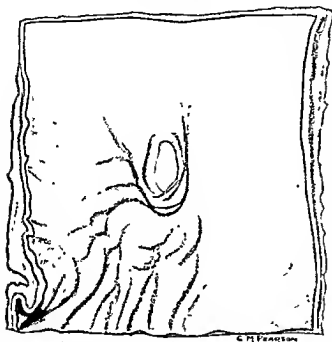


Fig 599—PERFORATED GASTRIC ULCER.  
(St. Mary's Hospital Museum.)

probably a more important one is that duodenal perforations are frequently *sub-acute*, leakage being limited to the right renal pouch, and spreading from this along the external paracolic sulcus into the right iliac fossa and pelvis, or round the liver edge to the sub-diaphragmatic space. In such cases the immediate symptoms lack the dramatic character of the acute perforation which floods the general

cavity of the peritoneum, and the lesion is likely to be missed in its early and, therefore, favourable stage. Not infrequently, a wrong diagnosis of appendicitis is made, perhaps after an interval of a day or two, or the perforation is overlooked completely until a subphrenic or pelvic abscess develops several days or weeks later.

*Diagnosis.* The diagnosis of an acute perforation is rarely difficult. A previous history of gastro-duodenal ulcer is obtainable in more than 90 per cent of cases, while the actual perforation is often caused by over-distension of the stomach, or by mechanical trauma. Typically, the onset is dramatic, with absolutely sudden and agonising epigastric pain, initial vomiting, and shock; but sometimes the pain is of more

## ABDOMEN

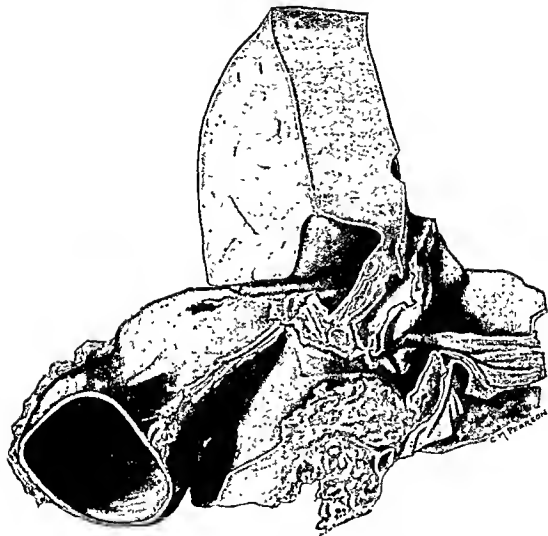


Fig. 600.—DUODENAL ULCER PERFORATING INTO RIGHT SUBHEPATIC SPACE.  
(St. Mary's Hospital Museum.)

gradual development, and initial shock may be trivial. The pulse-rate is often *deceptively slow* for some hours.

After the onset, there is usually a short *interval of recovery* from the initial shock and vomiting, but the pain continues, although even this may abate a little. During this "interval" there is real danger of failing to diagnose the lesion, but the true state of affairs is usually obvious on careful examination of the abdomen. Deep tenderness is present almost from the start, and soon becomes extreme. Board-like *rigidity* is another early sign, and is of the greatest possible diagnostic value; at this stage the abdomen is immobile and retracted.

Diagnosis must be made before the features of infective peritonitis (rising pulse, persistent vomiting, abdominal distension, cessation of flatus) appear.

Differentiation between acute gastric and duodenal perforations is almost impossible, unless it can be made on the past history of the case. This distinction, however, is of little moment: what matters is that the perforation should be diagnosed without delay.

*Treatment.* Very few cases of acute perforation recover without operation. This must be performed as soon as the patient recovers from the initial shock. The pre-operative period of waiting can be shortened, and the general condition improved, by giving an injection of morphine ( $\frac{1}{4}$  gr.), placing a radiant heat cradle over the bed, and administering intravenous glucose-saline. The first and last of these measures should never be omitted.

The abdomen is opened by a right paramedian incision, and any existing doubt is settled by finding free gas, particles of food, or sour-smelling fluid in the peritoneal cavity. The ulcer is readily discovered by lifting up the liver and pulling down the stomach; in eight cases out of ten it is near the pylorus, in the remainder it is further up along the lesser curvature. If the perforation cannot be seen, the lesser sac should be opened through the great omentum or transverse mesocolon and, if soiled contents escape, the ulcer will be found on the posterior wall of the stomach. The fluid exudate is gently mopped up, or preferably removed with a sucker.

The treatment of the perforation itself should be governed by the immediate needs of the case. The essential measure is *closure* of the hole by Lembert sutures, which infold the ulcer. Sometimes the infiltrated and œdematous condition of the margins of the perforation makes the closure extremely difficult, the sutures repeatedly cutting out, and the hole being made progressively larger. This difficulty can



usually be overcome by including sufficient tissue in the sutures ; if the closure of the perforation results in stenosis of the pylorus or duodenum gastro-jejunostomy becomes obligatory, but narrowing of the lumen can often be avoided by placing the sutures at right angles to the long axis of the tube. On two occasions, however, I have been forced to do a primary gastro-jejunostomy, because it proved impossible to close the perforation without markedly narrowing the lumen. Efficient retraction and a good light are essential to success in a difficult case, particularly when the perforation is some way back along the duodenum.

When suturing fails to effect a reliable closure, it should be reinforced by sewing some omentum over it, or by sewing the margins of the ulcer to the gall-bladder or liver. The method of fitting a drainage-tube into the perforation, and surrounding it with a cuff of omentum, is not recommended, except as a last resource ; neither do we favour *resection* of the ulcer and *pyloroplasty*, as this is a difficult operation in the conditions which obtain with a perforation, and it has a high mortality (Williams and Walsh, 9).

In an early case (under ten hours from onset) *drainage* is unnecessary, provided the perforation has been closed securely, and the peritoneum is fairly clean ; if any doubt exists, however, it is wise to drain the right renal pouch, the tube emerging through a stab wound near the loin. A suprapubic drain, with the end of the tube in the pouch of Douglas, is useful only when there is a large free effusion, and even then it ceases to be of service after 12 hours.

Recently I have successfully closed several perforations through a very small abdominal incision, as recommended by A. D. Wright (10). With a "free" ulcer at or very near the pylorus, the operation can be easily performed through an incision of one to two inches, the pylorus being picked up by the fingers and lifted into the wound. When this cannot be done with ease, or the ulcer is adherent, or at some distance from the pylorus, the incision must be enlarged without delay.

*The Question of Primary Gastro-Jejunostomy.* The closure of a perforation is a life-saving operation, but it cannot be expected to cure the ulcer. It is perfectly true that some cases remain quite free from symptoms for a large number of years after simple suture of the perforation, but I cannot agree that this figure is anywhere near 50 per cent (Pannett, 11, and others). Very recently I was able to follow up 73 cases of gastro-duodenal perforation successfully operated on at St. Mary's Hospital, between 1920 and 1932 ; 62 of them were treated

by simple suture, and 11 by suture and primary gastro-jejunostomy. Of the 62 suture cases only 12 (19 per cent) could be considered real cures (free from ulcer symptoms for two years or more); no less than 28 cases (45 per cent) had to have a secondary operation performed later, 16 cases (26 per cent) continued to have ulcer symptoms, or died of the ulcer, while the remaining 6 cases were free from symptoms for a period of less than two years.

The chance of curing the ulcer by simple closure of a perforation is thus about one in five, and this in spite of subsequent medical treatment. I have, therefore, no hesitation in agreeing with Deaver (12), Dineen (13), and others who claim that something more than simple closure is usually required. But I find it difficult to accept the view that this something is primary gastro-jejunostomy: firstly, because gastro-jejunostomy is not necessarily the best operation for peptic ulcer; secondly, because (in spite of published figures to the contrary) in the hands of most surgeons a gastro-jejunostomy at the operation for a perforated ulcer increases the primary mortality; and thirdly, because even one chance of a cure in five is not to be despised. Neither can one subscribe to the views of Judine (14) of Moscow, and others (quoted by Gilmour and Saint, 15), who advocate the heroic procedures of primary partial gastrectomy and gastro-duodenectomy for perforated ulcers; although Judine himself reports a mortality of only 12 per cent in his last 212 cases, outside special gastric clinics such methods would have a terrible mortality.

Our final conclusions are, therefore, as follows:

(i) That a perforated gastric or duodenal ulcer should be treated by simple suture only, unless this so narrows the digestive canal that a primary gastro-jejunostomy becomes obligatory.

(ii) That the case should then be given the benefit of thorough medical treatment, preferably at the hands of a physician.

(iii) That at the *first* reappearance of ulcer symptoms (and *not until then*), an operation should be performed with the object of curing the ulcer. Whether this operation is to be a gastro-jejunostomy, a sleeve resection, or a major gastrectomy or duodenectomy, cannot be argued in this article. What we must stress is that the second operation should not be delayed, once ulcer symptoms reappear, as there is real danger of a second perforation occurring if the ulcer does not remain healed.

## PERITONITIS OF APPENDICULAR ORIGIN

Acute appendicitis is the most common cause of both diffuse and localised peritonitis. The diffuse variety is usually associated with acute perforation, or with rupture of a purulent or gangrenous appendix; but it can also result from spread of infection through the wall of an intact appendix. According to Foss (16), 20,000 fatal cases of appendicitis occur in the United States every year, and diffuse peritonitis is the cause of death in 80 per cent of them. Analysis of statistics from several sources shows that the development of peritonitis increases the mortality of appendicitis by *more than ten times*.

*Delayed operation* is undoubtedly the chief explanation of this high incidence of fatal peritonitis in appendicular disease. Bower (quoted by Foss), analysing 5000 cases of acute appendicitis, showed that of cases operated on in the first 24 hours only one in 39 died; in the second 24 hours one in 17 died; in the third 24 hours one in 13 died; while of those operated on after the third day as many as one in 6 died. Statistics of the results of appendicectomies depend far more on the average time-interval between the onset of symptoms and the operation, than on the skill of the surgeon or the efficiency of the hospital service.

The first object of the surgical treatment of appendicitis should be the prevention of peritonitis, and this can only be assured by operating on all cases, *without exception or delay*. It is to be regretted that just when the hulk of practitioners, and the lay public, were becoming converted to the teaching of "immediate operation for all acute appendices," the unjustly termed *Ochsner-Sherren treatment* was introduced, or, more accurately speaking, re-introduced. Its exponents advocate immediate operation for the first and second day appendix, but a policy of watchful delay for the more difficult and dangerous third and fourth day cases, unless or until peritonitis develops. It is true that the mortality of immediate appendicectomy is bound to be higher in the latter type than in the former, and also that under ideal conditions the conservative policy may appear to give slightly better figures at the hands of *some* surgeons. But the real fact, which in our view cannot be overlooked, is that the waiting policy becomes extremely dangerous when it is adopted by average surgeons under average conditions. We therefore advocate immediate operation for all cases, and agree with Ashurst that "to delay operation in acute appendicitis is to gamble with death."

There can be no question that the taking of *aperients* increases the

incidence of peritonitis in acute inflammations of the appendix. Miller (17) reports that of 239 fatal cases of appendicular peritonitis more than 40 per cent admitted to having taken purgatives.

Peritonitis occurs not only as a complication of appendicitis, but also as a *post-operative complication* after appendectomy. Unfortunately this disaster is sometimes attributable to faulty surgery, particularly to the habit which is growing among younger surgeons of *not draining* the site of the operation, because definite pus is not present. Of course, a clean catarrhal appendix, and even an intensely congested appendix, do not require drainage, even if some serous or fibrinous exudate exists in the vicinity. But a friable or recently adherent appendix, a pus-filled or gangrenous appendix, or an oedematous, friable, and oozing appendix bed, are potential causes of peritoneal infection, and should be regarded as definite indications for drainage. Soiling of the peritoneum by rupture of a pus-filled or gangrenous appendix during its removal, or by tearing of protective adhesions, is another common cause of peritonitis, and is often attributable to carelessness, hurry, or inexperience of the operator. For the same reason we deprecate the opening of appendix abscesses into the uninfected part of the peritoneal cavity.

Occasionally, diffuse peritonitis occurs after appendectomy for no obvious reason, and I have known it to follow two cases of resection of absolutely cold appendices.

Regarding the actual *treatment* of appendicular peritonitis, little need be added to what has already been said. We believe the mortality would be lowered if all such cases were operated on under *spinal anaesthesia*. Complete removal of the appendix is essential, however friable or broken up it may be. Drainage of the appendix bed should never be omitted in the presence of peritonitis. Finally, we must not forget the peculiar tendency these cases show to develop a post-operative paralytic or adhesive ileus, and plan our treatment accordingly. Patients destined to recover soon begin to show signs of improvement, and the worst of the peritonitis is generally over by the end of a week. Also, those who fail to survive rarely live longer than a week.

#### POST-OPERATIVE PERITONITIS

*Etiology and Prophylaxis.* Few things can be more distressing to a surgeon, or more fatal to the patient, than the development of peritonitis after an abdominal operation. Sometimes this complication is unavoidable, or at least we cannot be held responsible for it, but more

often than not it could have been prevented. The surgical errors, both of commission and of omission, which may lead to peritoneal infection are so numerous that we cannot do more in this place than refer to them very briefly.

One of the most common causes is *soiling* of the peritoneum by *intestinal contents*, or by the contents of an *infected viscus* or a *localised abscess*. Such soiling is always avoidable by the exercise of due care. A pus-filled appendix or gall-bladder must be handled with the greatest gentleness, and the peritoneal cavity should be carefully packed off at the start of the operation; sometimes it is wise to empty a suppurating viscus by aspiration, before attempting its removal. A localised abscess, whenever possible, should be opened extra-peritoneally; if, however, the general peritoneal cavity has to be traversed, every precaution must be taken against spillage of the pus, by careful packing, and by complete evacuation of the abscess. The separation of *recent adhesions* round a focus of acute infection is always a risky procedure; as far as possible, therefore, such adhesions should be left alone.

Soiling of the peritoneum by *intestinal contents* is unfortunately far from uncommon, and can only be attributed to gross carelessness; peritonitis resulting from this cause is extremely fatal. The contents of the lower ileum are the most dangerous, particularly in conditions of obstruction. The jejunal contents have a much lower bacterial concentration and are relatively free from danger, especially in the upper jejunum. The contents of the unobstructed stomach and duodenum are practically sterile. The colonic contents, being almost solid, spread much more slowly, and so can be removed with more certainty; spillage during operations on the colon is, nevertheless, a very serious matter.

A common cause of faecal soiling is injury to a coil of intestine with a scalpel, when making the first abdominal incision. This is a very easy thing to do, and, almost before we realise it, the peritoneum becomes hopelessly flooded with highly septic material. I have known this accident to happen to three young surgeons on the threshold of their career; all three patients died, victims of the youthful desire for speed and "showy" operating. Another common reason for spillage is rupture of distended or friable intestine, produced by rough handling; further causes are incomplete emptying of intestinal segments during resection and anastomosis, and slipping of intestinal clamps in this or other operations on the intestine. Infection of the peritoneum from the gut, however, can occur without spillage of contents; excessive handling of obstructed intestine will do it, and for this reason we

deprecate "exploratory" laparotomies for carcinoma of the colon with acute obstruction.

The peritoneum may also become soiled with faecal material *after* an abdominal operation. Most frequently this is due to leakage of an anastomosis, particularly in the large intestine. The causes of such leakage are numerous, the most common ones being: (a) distension of the intestine; (b) poor blood supply; (c) incomplete peritoneal covering; (d) bursting of the stumps in a side-to-side union. The first can be avoided by proximal safety-valve drainage, the second and third by choosing suitable parts of the intestine for the operation, and the fourth by the procedure shown on page 1060. Post-operative peritonitis, especially after operations on the colon, may be caused by escape of organisms from the intestine, without actual leakage of contents; to some extent this may be guarded against by pre-operative treatment with peritonitic vaccine (see page 1125).

Failure to *drain* an infected focus is another potent cause of peritonitis (see page 1119).

Finally, post-operative peritonitis may occur without any soiling whatever, and even after "clean" operations. The introduction of infection from without is, of course, occasionally responsible, but this is a very rare event in a well-conducted and up-to-date hospital. Low resistance on the part of the patient, or prolonged exposure and handling of peritoneal surfaces, may account for other cases. But a cause which is apt to be overlooked is flaring up of an old and dormant focus of infection. For example, I have had a fatal peritonitis supervene on a lumbar sympathectomy, performed on a man who had his appendix removed more than ten years previously; during the operation some adhesions anchoring the cæcum had to be divided, and this was the only possible source of infection found at the autopsy. I also know of another case of fatal peritonitis after lumbar sympathectomy, this time because a cold appendix was removed at the same operation.

*Diagnosis.* The early stages of post-operative peritonitis are by no means easy to diagnose. Peritonitic pain is often masked by morphine, or mistaken for the gas-pains of post-operative stasis; vomiting is put down to the anæsthetic; while abdominal tenderness and rigidity are attributed to the trauma of the operation. It is only when the temperature and especially the pulse-rate begin to show an upward trend, and the general condition of the patient to deteriorate, that the true state of affairs may dawn upon us. This delay can be avoided if the surgeon is constantly on guard, and ready to examine the abdomen

as soon as any suspicious symptoms develop; the discovery of deep tenderness or rigidity in regions away from the operation wound should be taken as definite evidence of peritonitis.

*Treatment.* The abdomen should only be re-opened when a cause of gross infection is suspected, e.g. leakage of an intestinal anastomosis, rupture of distended intestine, necrosis of what was thought to be viable gut, a massive accumulation of blood clot, etc. In such cases life may be saved by prompt treatment of the cause. If drainage has not been instituted at the first operation, it should be done now. When a source of gross infection is unlikely (e.g. after primary soiling of the peritoneum), or a septic focus has already been drained, a second operation cannot be expected to influence the course of the case, and treatment should be directed to the toxæmia and paralytic ileus. In the unlikely event of the patient's recovery, a careful watch must be kept for a residual abscess.

#### PRIMARY PERITONITIS OF CHILDREN

Primary peritonitis of children is an extremely fatal disease. Barrington Ward (18) states that all the 22 streptococcal cases admitted into Great Ormond Street Hospital in the last 15 years died, including 12 who were operated on. But Bruce and Logie (19) report having seen eight streptococcal cases in four years, three of which recovered after operation, while Duncan (20), who collected 66 cases of primary peritonitis, gives a mortality of only 38 per cent.

The term "primary" is given to those cases of peritonitis for which no obvious abdominal cause can be found. Although the disease is mainly one of *early childhood*, it is occasionally seen in adults, particularly *puerperal* women and sufferers from *nephritis*. *Female* patients predominate, and the commonest age is under a year, and between 5 and 8.

Two organisms appear to be almost entirely responsible—the *streptococcus* and the *pneumococcus*, and they are usually found in pure culture. The streptococcal variety is the more serious, its probable mortality being about 80 per cent (Price, 21), while that of pneumococcal peritonitis is about 60 per cent.

There can be no doubt that in most cases the peritonitis is a local manifestation of *septicæmia*, and that the infection reaches the peritoneum by the *blood stream*; this is particularly the case in streptococcal cases. But some pneumococcal cases appear to be produced by

*lymphatic spread*, either from the *female genital organs*, or from the *thorax*. It should not be overlooked that *umbilical sepsis* may be a possible cause in new-born infants. In a recent paper, Lazarus (22) points out that children with *nephritis* are particularly susceptible to streptococcal and pneumococcal infections.

It is a striking fact that while secondary peritonitis usually starts as a localised condition, and becomes generalised, the primary variety begins as a diffuse inflammation, and becomes localised if the patient lives long enough.

*Diagnosis.* The absence of a local cause and the youth of the patient combine to make diagnosis a difficult problem. Abdominal pain and vomiting are the outstanding symptoms, but the latter may overshadow the former, and they are both common symptoms of childhood disorders. Tenderness tends to be most marked in the lower half of the abdomen, but there may be little or no rigidity. The temperature and pulse are usually both raised, and there may be a scarlatiniform rash, or other manifestations of toxæmia. The white cell count varies, but leucocytosis appears to be usual.

It is thus obvious that the clinical picture is somewhat indefinite, particularly in the early stages. In doubtful cases abdominal *paracentesis* has been suggested and practised by several authorities; when carefully performed, it is said to be free from danger. Instead of abdominal paracentesis, Lowe (quoted by Fraser, 23) recommends puncture through the posterior fornix of the vagina as a safer procedure.

*Treatment.* If one could be certain that an attack of peritonitis in a child is "primary," it would doubtless be wiser to defer operation until the condition becomes localised. This is the conclusion one arrives at after study of the mass of literature published recently on this subject; the main thesis of recent workers appears to be that the factor which determines recovery is "peritoneal immunity," that the development of peritonitis means imperfect immunity, and that early operation is likely to interfere with increase of this immunity. Furthermore, there appears to be some truth in the claim that early operation increases the likelihood of fatal pulmonary complications.

But, unfortunately, it is rarely possible to be certain that we are not dealing with a *secondary*, and particularly an appendicular peritonitis, and the policy of non-intervention is too risky to be either advocated or practised. When confronted with a case of peritonitis, our first duty is to *open the abdomen*, whether the patient be an adult or a child.



In the absence of anything to guide us, we should make a small exploratory incision over the right iliac fossa. The failure to find a cause for the peritoneal infection is the surest evidence that we are dealing with a primary peritonitis.

We may then decide to evacuate the toxic exudate and drain the abdomen, or, if we do not wish to interfere with immunisation, we may leave the effusion alone and close the abdomen, deferring drainage to a later date (15th-25th day); personally, I would choose the former alternative.

*Serum-therapy* appears to have given good results, both in pneumococcal and streptococcal cases, but the appropriate serum must be given early and in large quantities. *Blood-transfusion* is alleged to have saved several cases of pneumococcal peritonitis, but it is of doubtful value in streptococcal cases. The use of intravenous *emulsions of olive oil* has already been referred to, also the suggested employment of the emulsion intra-peritoneally.

## CHRONIC DIFFUSE PERITONITIS

### (1) TUBERCULOUS PERITONITIS

Tuberculous peritonitis is a disease of children and young adults, and affects both sexes equally, although women come to operation more often than men. In children the condition tends to run an acute or sub-acute course, but in adults it is usually chronic from the onset. The mortality of untreated cases is very high, but combined medical and surgical treatment appears to cure nearly 50 per cent.

In most cases the disease is secondary to tuberculosis of the appendix, the mesenteric glands, or the Fallopian tube, the peritoneum becoming infected directly, or via the lymphatics. It can also complicate phthisis, the infection in this case being probably hæmatogenous. The frequency of a tubal origin varies in different statistics, but it is probably higher than most of us realise.

The *morbid anatomy* of the lesion is too well described in elementary text-books to require more than a passing reference in this article. Four pathological varieties are recognised: (1) The *wet* or *serous* type, in which there is much effusion and numerous peritoneal tubercles, but little else. (2) The *dry* or *adhesive* type, which is characterised by the formation of numerous adhesions, binding coils of intestine to each other and to the abdominal wall, and dragging them back by fibrous

contraction of the mesentery; this variety leads to chronic small-gut obstruction of a progressive type. (3) The *ulcerative* or *fistulous* variety, in which the outstanding change is the development of fistulous communications between various viscera, and even of external fistule, usually opening to the surface near the navel. (4) The *purulent* type, which in acute cases tends to be diffuse, but in the more chronic ones becomes localised (encysted peritonitis). Most cases partake of the characters of two or more of these four types.

*Diagnosis.* The acute and sub-acute cases in children are fairly typical. There is a high, remittent or intermittent fever, often with chills; also there is anorexia, rapid wasting, abdominal pain, and progressive distension. Diarrhœa is not uncommon, but the child may be constipated, or constipation and diarrhœa may alternate. On examination, the abdomen is tender, often somewhat rigid, and there is obvious ascites. Leucopenia is usual. In male children the fluid may fill a patent funicular process, with the formation of a "congenital" hydrocele.

*Chronic cases* are very variable in their clinical manifestations. The symptoms may resemble the above picture, except that they are less acute; or the case may present itself as a chronic intestinal obstruction, with acute exacerbations; or it may appear as a cystic swelling, or a localised abscess in the abdomen. In the "dry" type several masses may be palpated, and are produced by matting together of coils of intestine, or by "rolling-up" of the omentum. Not infrequently diagnosis is not made until the abdomen is opened.

*Treatment.* The chief justification for surgery, apart from establishing the diagnosis, is that sometimes a local cause can be found and removed. Salpingectomy produces excellent results when the tube is infected, while the removal of a tuberculous appendix is only a little less successful. In ascitic cases the mere opening of the abdomen sometimes produces a dramatic, if temporary, improvement. Surgery may also be needed to deal with complications, the chief of which is obstruction.

All cases should receive very thorough medical treatment. In the acute variety, and when no local cause can be found or removed, hygienic therapy provides the only hope of a cure. Aspiration with oxygen-replacement has its advocates, but the results hardly justify this hazardous procedure.

(II) CHRONIC PYOCOCCAL PERITONITIS

Chronic diffuse peritonitis may be caused by several pyogenic organisms, but three varieties exist which are quite distinct from one another, viz. pneumococcal, gonococcal, and septic.

(a) *Chronic pneumococcal peritonitis* is similar to tuberculous peritonitis, both in its morbid anatomy and in its clinical course. Thus it occurs in ascitic, adhesive, and encysted forms, with corresponding clinical pictures. The main distinction lies in the *prognosis*, for while at most only 50 per cent of tuberculous cases recover, the great bulk of chronic pneumococcal cases resolve completely.

(b) *Chronic gonococcal peritonitis* is a gynaecological disorder, and can receive only the most brief consideration here. It is an extremely common result of gonococcal infection in the female, and is almost always preceded by salpingitis. The peritonitis is in the main *adhesive* in type, and is, of course, almost limited to the pelvis, where the various viscera are matted together almost inextricably. The only complication of interest to the general surgeon is *intestinal obstruction*, from matting, kinking, or compression of the intestine.

(c) *Chronic septic peritonitis* is a residue of acute diffuse peritonitis which has resolved incompletely. The condition is inevitably fatal, although the course may be very prolonged. Clinically, the picture is that of an exhausting illness which supervenes after partial recovery from acute peritonitis. There is irregular fever, night sweats, diarrhoea, and rapid emaciation. At autopsy, many parts of the peritoneal cavity are obliterated by fibrosis, while isolated areas of it are represented by abscess cavities lying in the midst of matted abdominal contents.

(III) ENCAPSULATING CHRONIC PERITONITIS

This is a peculiar condition which has excited much interest in the last two or three years, particularly in Germany and France. The main pathological feature appears to be the development of a thick contractile membrane, which envelops portions of the small intestine and mesentery, and which becomes densely adherent both to the viscera it envelops and also to the parietal peritoneum. A similar membrane may form over the stomach, and perhaps the liver and spleen, but the large intestine is apparently never involved (Devine, 24). Neighbouring coils adhere to one another, and, enclosed by this membrane, form one

or more globular "tumours," which constitute a conspicuous feature when the abdomen is opened. The intestine inside these "tumours," or perhaps more accurately termed "cysts," becomes obstructed, so that dilated gut may be seen entering into them.

Later on, continued formation and thickening of this membrane may lead to obliteration of large parts of the peritoneal cavity. The contractile tendency of the membrane is seen when an incision is made into the intestine for the purpose of performing an anastomosis: the intestinal wall prolapses through the incision in a disconcerting manner, and the intestine may even be turned inside out.

The *etiology* of this interesting though rare condition is unknown, but it is said to be the result of "extinguished" tuberculosis of the peritoneum.

The *clinical features* are not unlike mild appendicitis, which is the usual pre-operative diagnosis. The patient becomes progressively more and more constipated, often complains of dyspepsia, and suffers from recurrent attacks of colicky pain. Sometimes a mass can be felt, and not unnaturally a suspicion of cancer arises.

The *surgical treatment* is far from easy. Some cases are said to undergo spontaneous resolution in time, and operative intervention is therefore only indicated when obstructive symptoms supervene. In such cases the best procedure is *decapsulation* of the contractile membrane, with the object of unravelling the obstructed intestine. Apparently a line of cleavage can be obtained at the mesenteric border of the intestine, and it is essential that the decapsulation should be started here. In late cases this may prove impossible, and a short-circuit by lateral anastomosis seems the only alternative, but this is both difficult and dangerous owing to the persistent tendency of the gut-wall to prolapse through the incisions. To add to the surgeon's troubles, it is often difficult even to find the peritoneal cavity!

Quite recently I operated at St. Mary's Hospital on an elderly man with symptoms of sub-acute intestinal obstruction. After opening the abdomen, the picture at first sight appeared to be one of encapsulating peritonitis. About five feet of ileum were closely matted by adhesions, and there was a globular mass in the immediate vicinity, attached to and partially enveloping the adherent coils of intestine; the gut proximal to the mass was distended. A closer inspection, however, showed one very definite area of tubular thickening of the terminal ileum, some two inches in length, and of almost cartilaginous consistency, and there were other smaller but similar patches more proximally. Moreover, the globular mass was definitely cystic in character, the peritoneal cavity was nowhere obliterated, and there was no evidence of a contractile membrane. Also, several large and firm glands were palpated in the mesentery of the affected intestine.

I could not arrive at a definite diagnosis, but thought that the condition was probably an atypical carcinoma of the ileum. With some difficulty I was able to free the entire mass, and remove it in one piece, which included the matted coils of ileum. The resection was followed by end-to-end anastomosis, and the patient made a complete recovery.

Examination of the resected structures was not very illuminating, but it at least proved that there was no carcinoma; neither was there any real evidence of tubercle. A possible explanation is that this was an atypical case of the lesion recently described as "ileitis plastica."

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## CHAPTER V

### PERITONEAL ABSCESSSES

#### GENERAL CONSIDERATIONS

LOCALISED suppuration in the peritoneum may complicate a local infective lesion in the abdomen (*secondary abscess*), or it may be left behind after the subsidence of a diffuse peritonitis (*residual abscess*). In the first case the suppurative process is localised from the start, by the development around it of protective adhesions which shut off the general cavity of the peritoneum. In residual abscesses the suppuration is generalised at the start, but the larger part of the peritoneum recovers, and the pus becomes limited by adhesions, either to the region of the causative focus, or perhaps to some dependent part of the abdominal cavity. Although a secondary abscess as a rule develops in the vicinity of the original infection, it can also occur in a distant part of the peritoneum, or in the extra-peritoneal cellular tissue. In such cases the infection often travels via the lymphatics.

Clinically, an abdominal abscess manifests itself as a *tumour* or *mass*. Pus occupies the centre of the mass, but the bulk of the tumour is made by adherent omentum, mesentery, intestine, and other viscera, which between them constitute the *abscess-wall*. Almost all abdominal abscesses, at some part of their surface, are in contact with the abdominal parietes, which thus form part of the abscess-wall; most of them are in relation to the posterior abdominal wall, but some are in contact with the pelvic floor, the diaphragm, or the anterior abdominal wall. As it enlarges, a peritoneal abscess may come into relation with a new part of the parietes. Thus, a posterior abscess may come forward and eventually adhere to, and even come through, the anterior abdominal wall; similarly, a pelvic abscess may rise up out of the pelvis and come into contact with the anterior abdominal wall.

Abscesses may form anywhere in the abdomen, but the common sites are: (i) the iliac fossæ; (ii) the pelvis, and (iii) the subphrenic region. The transverse colon and mesocolon separate the subphrenic region from the rest of the abdominal cavity, and abscesses in that

region are discussed under the general heading of *subphrenic abscess* (see page 1153). *Iliac abscesses* are mostly seen in connection with appendicitis, while *pelvic abscesses* usually complicate appendicitis and gynaecological disorders, or occur as residual abscesses.

The *diagnosis* of abdominal abscess is usually quite simple. The patient is already ill with an infective lesion in the abdomen, or is convalescing from one, when he suffers a relapse. Sometimes he becomes worse fairly suddenly, but as a rule the onset of the abscess symptoms is gradual; often it is insidious. The *general symptoms* are those of toxic absorption: fairly high fever, often remittent, a full and rather rapid pulse, malaise, furred tongue, night sweats, a hectic flush, and rapid wasting. Constipation is the rule, and even paralytic ileus may occur, but cases of pelvic abscess frequently have diarrhoea and increased frequency of micturition. The *local symptoms and signs* are those of a localised peritonitis: there is increased pain, usually of a throbbing nature; *local tenderness*, both deep and superficial, and often excruciating; and *localised rigidity*, usually very well marked, of the muscles overlying the abscess. In addition, a *mass* or *tumour* can be felt in most cases. An iliac or central abdominal abscess can be palpated with ease through the anterior wall, in spite of the rigid muscles over it; but a pelvic abscess may be only palpable on rectal, vaginal, or bimanual examination, unless it is very large and rises into the abdomen. A subphrenic abscess is only occasionally palpable (e.g. when it is subhepatic), but indirect evidence of its presence may be obtained by *radiography*, or by feeling a downward-displaced liver.

A strongly confirmatory evidence of pus under tension is the discovery of *leucocytosis* on examination of the blood. In abdominal abscesses figures of 20,000 or more are common.

A diagnostic procedure much employed in the past is *aspiration*. Unfortunately, this entails a definite risk of infection of the peritoneum or pleura, one of which has to be traversed on the way to the abscess. For this reason, the use of the exploring needle for diagnostic purposes should be abandoned, although it may be employed to confirm the presence of pus during the actual operative approach to the abscess.

*Treatment.* A peritoneal abscess should be evacuated and drained as soon as it is diagnosed. It must be admitted that spontaneous cure sometimes occurs by absorption of the abscess, or by its rupture through the abdominal wall, or into a hollow viscus, such as the rectum, vagina, stomach, and intestine. The chance of this, however, is so uncertain that it should never influence our treatment. On the

contrary, this should be determined by, and should anticipate, the ever-present danger of rupture of the abscess into the general cavity of the peritoneum ; the resulting peritonitis has an extremely high mortality. In subphrenic cases there is the additional and very real danger of secondary involvement of the thoracic contents, by spread of infection through the diaphragm. For these reasons we strongly advocate early evacuation and drainage of all abdominal abscesses.

*The operative approach to a peritoneal abscess should be so planned as to avoid contamination of uninvolved parts of the peritoneal cavity.* The common practice of approaching an appendix or other intra-abdominal abscess through the clean peritoneal cavity is surgically unsound, since it exposes the patient to a definite risk of fatal peritonitis. Sometimes the transperitoneal route is employed in cases in which it could quite easily be avoided ; one would have to restrain oneself to describe such a procedure as anything less than criminal. But even in cases in which at first sight a transperitoneal approach appears inevitable, the exercise of a little imagination and ingenuity will nearly always enable us to avoid it.

Not infrequently an abscess can be opened by a *direct* incision over it, without any danger of entering the general abdominal cavity. This is, of course, the case with all abscesses adherent to the anterior or lateral abdominal wall, e.g. late iliac abscesses, and late anterior subphrenic abscesses. The same applies to abscesses in the pouch of Douglas which become adherent to the rectum or vagina ; in such cases the abscess can be evacuated and drained by an incision through the rectal or vaginal wall, with absolute safety and the almost certain prospect of recovery.

Abscesses not adherent to the anterior abdominal wall or rectum must be approached by an indirect *retroperitoneal route*. It is assumed that the inflammatory mass is attached by part of its circumference to the peritoneum of the posterior abdominal wall, as is usually the case. An incision is made in a convenient situation near the abscess, down to but not through the peritoneum. *The peritoneum is then separated from the anterior and posterior parietes until the abscess-wall can be definitely felt.* The practised finger has no difficulty in deciding when the region of safety is reached, i.e. the area in which the part of the peritoneal cavity originally overlying the abscess is obliterated by adhesions. The finger or a sinus forceps is pushed through this area forwards into the abscess, the pus carefully mopped out or removed with a sucker, and a large, fenestrated rubber tube inserted into the cavity. The drainage-tube should be placed in the position which best



facilitates drainage; in many cases it will be necessary to make a counter-incision in a more suitable site, and to insert a second drain into the abscess cavity through it.

In exceptional cases a peritoneal abscess is so deeply or *centrally* situated that it is nowhere in actual contact with the parietes, the pus being contained between coils of intestine, omentum, and mesentery. It is impossible to approach such an abscess by a retroperitoneal route, and the policy of waiting until the abscess reaches the anterior or posterior abdominal wall is too dangerous a one to recommend. There are two practical alternatives for the surgeon's choice.

One alternative is to open the abdomen directly over the mass, and very carefully to pack off the peritoneal cavity all round, leaving a narrow track to the abscess; through this a large aspirating needle is pushed in, all the pus sucked out, and the abscess opened and carefully dried with swabs on holders. The abscess cavity is then wiped out with spirit, and a large non-fenestrated drainage-tube inserted into it, fitting snugly in the hole which has been made in the abscess wall.

The other alternative is to open the peritoneal cavity over the abscess, leave the abscess alone, and insert a large tube through the wound into actual contact with the abscess-wall; the chances are that within 48 hours the abscess will burst into the drainage-tube and thus evacuate itself. The danger of infecting the peritoneum is obviated by the rapid formation of adhesions round the drain which seal off the general peritoneal cavity. If at the end of 48 hours the abscess has not started to discharge, it should be opened by the finger or a sinus forceps pushed through the tube. This is undoubtedly a safer procedure than the one-stage transperitoneal opening of the abscess, and should be employed unless circumstances indicate immediate evacuation.

### (I) ILIAC ABSCESS

(a) *Right Iliac Abscess.* Abscesses in the *right iliac fossa* are an extremely common result of acute suppurative or perforative *appendicitis*. Most frequently they are true *secondary* abscesses, being localised to the region of the appendix from the start. Less often they are *residual* abscesses, left behind after recovery from a diffuse peritonitis. A third group are *post-operative*, the abscess developing after appendicectomy, mostly owing to the omission of drainage when this is necessary.

The exact situation of the abscess varies with the position of the appendix. Thus it may lie mostly behind the caecum, external to it

in the outer paracolic sulcus, in front of the cæcum, or to its inner side. As the abscess enlarges it outgrows its original boundaries, but part of it always maintains its first relations. A retrocæcal or external paracolic abscess is usually adherent to the posterior peritoneum from the first, and can therefore be easily reached by the retroperitoneal route. An antecæcal abscess soon adheres to the peritoneum of the anterior abdominal wall, when it can be opened by a direct incision without trespassing on the peritoneal cavity. But an abscess to the inner side of the cæcum may either remain entirely free of the parietes, or become adherent to the psoas muscle and implicate the psoas sheath.

As an appendix abscess increases, it may spread in any direction, but it mostly does so either outwards into the *loin*, downwards into the *pelvis*, or upwards to the *subphrenic* region. A fourth direction of spread is above the symphysis pubis and in front of the bladder, to the *left iliac fossa*, producing what is known as a *horseshoe abscess*. Finally, it should be remembered that an appendix abscess may be entirely outside the peritoneum, i.e. *retroperitoneal*; this is most likely to be the case when the appendix itself is retrocæcal in position, and also adherent to the posterior abdominal wall, but it may result from spread of infection along the lymphatics or cellular tissue of the meso-appendix to the retroperitoneal tissue. A retroperitoneal appendix abscess may remain in the iliac or lumbar region, but not infrequently it spreads upwards to the posterior extra-peritoneal subphrenic space (see page 1158). It is important to observe that the discovery of pus outside the peritoneum does not exclude the possibility of an intra-peritoneal abscess being present as well.

Appendicitis is not the only lesion which causes a right iliac abscess. Intra-peritoneal abscesses may be produced here by *Meckel's diverticulitis*, *carcinoma of the cæcum*, and *actinomycosis* or *tubercle* of the same organ. Retroperitoneal abscess in the right iliac fossa may result from *suppuration of the iliac glands*, and also from a lateral extension of a *psoas abscess*.

*Diagnosis.* The symptoms and signs have been discussed so fully in an earlier part of this article that little need be added here. The constitutional symptoms of toxæmia, with the discovery of an extremely tender and hard mass in the right iliac fossa, which can be palpated in spite of the local rigidity of the muscles over it, combine to form a characteristic picture. If confirmation is needed, it can be obtained by an examination of the blood, which usually shows very definite leucocytosis.

When an iliac abscess is intra-peritoneal, the tumour, tenderness, and rigidity are discovered in and over the iliac fossa, but with a retroperitoneal abscess the swelling may only be palpable in the flank, and the rigidity and tenderness are most marked posteriorly. In fact, the case closely resembles, and is indeed a variety of, perinephric abscess.

The differential diagnosis between *appendicular* and other types of right iliac abscess mainly depends on the previous history, and on the acute nature of the symptoms. Mistakes, however, are far from uncommon. Thus, an abscess round a *carcinoma* or *actinomycosis* of the cæcum is often misdiagnosed as an appendix abscess, but the chronic nature of the preceding illness should at least suggest alternative possibilities. Suppuration of the *iliac glands* is more deeply situated than most appendix abscesses, and usually presents a less acute picture; moreover, the discovery of a primary focus of infection, and the absence of preceding appendix symptoms, should lead to a correct diagnosis. A *psoas abscess* tracking out under the iliac fascia may resemble an appendix abscess to the inner side of the cæcum; this is particularly the case when the appendix is adherent to the psoas muscle, and associated with the flexion deformity of the hip-joint which characterises irritation of this muscle. But the absence of any signs of spinal or hip disease, and the acute illness of the patient, should suffice to prevent diagnostic errors.

An abscess from *Meckel's diverticulitis* is clinically indistinguishable from an appendix abscess.

(b) *Left Iliac Abscess.* One of the most common causes of suppuration in the left iliac fossa is *appendicitis*. This would be expected in the rare cases of *transposition* of the viscera, but far more often it is produced by spread of infection from a normally placed appendix. The occurrence of *horseshoe abscess* has already been referred to, but a left-sided abscess can also occur when the right iliac fossa is free from pus; this left iliac abscess is of special interest when it develops *after appendicectomy*, in which case the usual explanation is contamination of the peritoneum on the left side during the operation, or spread of infection along the peritoneum or via the lymphatics (often attributable to omission of drainage).

Other causes of left iliac abscess are *carcinoma* of the sigmoid colon, *sigmoid diverticulitis*, *suppurating iliac glands*, and *psoas abscess*. The differential diagnosis is in no sense different from that of right iliac abscesses, except for the relative infrequency of appendix abscess on

this side. In old people a left iliac abscess is most likely to be caused by carcinoma or diverticulitis of the sigmoid.

*Treatment of Iliac Abscesses.* The treatment of an iliac abscess is much the same, whether it is of appendicular origin, or caused by carcinoma, diverticulitis, or some other condition. All acute abscesses should be opened and drained without delay, and every effort must be made to avoid contamination of the clean peritoneal cavity. An abscess which is obviously coming through the anterior abdominal wall, or pointing in the flank or near the iliac crest, can be opened by an incision directly over it, without fear of entering the general cavity of the peritoneum.

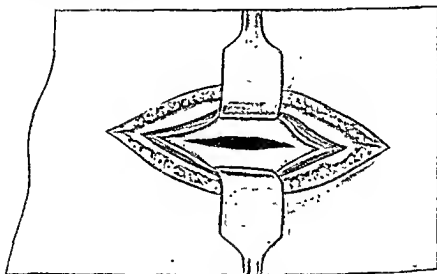


Fig. 601.—AUTHOR'S INCISION FOR DRAINING AN ILIAC ABSCESS.

In all other cases the operation is bound to be exploratory in its first stage. The ideal *anesthesia* is *spinal*, but a regional nerve-block or gas-oxygen-ether can be employed instead. The best general *incision* is a horizontal one extending inwards for two inches from the anterior superior spine (fig. 601); everything is divided down to but not including the peritoneum.

The peritoneum is now carefully palpated, and if it is adherent to the underlying mass we know that the peritoneal cavity is obliterated at this point, and that the abscess can be opened in safety by a direct incision through the adherent peritoneum. But if the peritoneum can be moved over the mass, thus proving that the cavity is still patent, the direct route is abandoned, and the *retroperitoneal* route employed instead. In the lateral angle of the incision the edge of the peritoneal

reflection from the anterior to the posterior wall is sought for, and gradually peeled off the iliac fascia in a medial direction with a finger (see fig. 602), which is insinuated behind it. This separation of the posterior peritoneum is gently continued with the finger, assisted by gauze-swabs on swab-holding forceps, until a definite resistance is encountered, which means that the point at which the abscess is adherent to the posterior parietes has been reached. The finger now definitely feels the infiltrated wall of the abscess, and may even detect its fluctuating contents. A way is next made through the wall into the abscess, either by the finger, or a sinus forceps, and the pus evacuated with the sucker. After this, the abscess cavity, and the space made in the retroperitoneal region to reach it, are swabbed over with spirit,

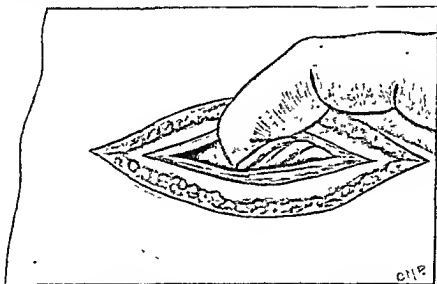


Fig. 602 —ARTHUR'S INCISION FOR DRAINING AN ILIAC ABSCESS.

and a large fenestrated drainage-tube is inserted into the cavity. The tube is brought out of the outer angle of the incision, the remainder of which is approximated with a few loose sutures.

Occasionally, the separation of the peritoneum from the iliac fossa may have to be continued to the edge of the psoas muscle, and even over the muscle itself, before the adherent wall of the abscess is reached. In exceptional cases no such adhesion is reached, and the peritoneum is mobile over the whole abscess, *behind* as well as in front; this is the *central* or deep-seated abscess, which cannot be approached by any but a transperitoneal route, and the treatment of which has already been outlined on page 1145.

Whenever an appendix abscess is opened, search should be made for a possible *extension* of the suppurative process. The most common

direction for such an extension is *towards the loin* ; when this is found, a sinus forceps should be pushed from the original incision into the pocket in the loin, a counter-incision made on to the points of the forceps, and a second drainage-tube inserted through the counter-incision.

Another common extension of an iliac abscess is into the pelvis ; if this is discovered during the iliac operation, an assistant should perform a rectal or vaginal examination. Should the pelvic extension be already adherent to the rectal or vaginal wall, the iliac incision is drained and sutured, the patient placed in the lithotomy position, and a counter-opening for drainage made through the rectal or vaginal wall (see page 1152). When the pelvic extension is still free in the pelvic cavity, however, it is safer to evacuate and drain it from the iliac incision.

Under no circumstances is it permissible to attempt the removal of the appendix when operating for an appendix abscess, unless this organ is lying free in the abscess cavity. It is particularly dangerous to essay the removal of an appendix adherent to the abscess-wall, as this is more than likely to open up the uninfected peritoneal cavity. For the same reason any adhesions encountered during the operation must be most strictly respected. Appendicectomy should be left over and performed in a cold interval.

When it appears likely that an iliac abscess is entirely retroperitoneal (c.g. suppurating iliac glands, or acute psoas abscess), a better incision than the above is an oblique one just above and parallel to the outer half of Poupart's ligament. The possibility of a direct approach to the abscess does not arise, and through this incision it is particularly easy to peel the peritoneum off the iliacus and psoas muscles, and so to reach a collection of pus behind it.

## (11) PELVIC PERITONEAL ABSCESS

In this place only those pelvic abscesses will be considered which are intra-peritoneal, i.e. in the pouch of Douglas. Their commonest cause is again the appendix, but we must distinguish between the appendix abscess which is pelvic from the start, and is produced by a "pelvic" appendix, and the abscess which comes down from above, and is therefore associated with an iliac appendicitis or an iliac abscess. In women an abscess in the pouch of Douglas is often caused by disease of the pelvic organs, especially *salpingitis*. Other causes of pelvic peritoneal abscess are : *carcinoma* of the pelvic colon, rectum,

bladder, or uterus; *Meckel's diverticulitis*; *sigmoid diverticulitis*; and other pelvic disorders. But an abscess in Douglas's pouch may also be *residual*, and caused by a local accumulation of pus after recovery from a diffuse peritonitis (e.g. in gastro-duodenal perforation).

The *diagnosis* of pelvic abscess is based on a combination of four factors:

(a) The presence of a *likely causative disease*, e.g. appendicitis, salpingitis, etc.

(b) The symptoms of *toxic absorption*, such as high remittent fever, night sweats, loss of weight, and leucocytosis (for further details see page 1143).

(c) Special symptoms produced by irritation of the *pelvic viscera*: particularly *menorrhagia*, *dysmenorrhœa*, *increased frequency of micturition*, and *diarrhœa* (not all of which, however, are necessarily present).

(d) Palpation of an exquisitely *tender mass* in the pouch of Douglas, by either *rectal* or *vaginal examination*, or by both.<sup>57</sup>

In an early stage the mass may be at some depth, and may be fully palpable only on manual examination; the rectum or vagina are still quite free from the tumour, and their wall can be moved independently from it. Later on, however, the abscess reaches and becomes adherent to the rectal or vaginal wall, which can no longer be moved apart from the mass.

*Treatment.* The tendency of abscesses in the pouch of Douglas to adhere to the rectum or vagina, with obliteration of the intervening part of the peritoneal cavity, provides us with the opportunity of evacuating and draining the abscess, without either transgressing the uninfected peritoneum, or having to embark upon a tedious and uncertain retroperitoneal approach. This opportunity of opening and draining the abscess into the rectum or vagina is one we cannot afford to ignore, but the operation is really safe only when the mass has become adherent to the wall of one or other viscus. A premature evacuation through this route, i.e. while the rectal or vaginal wall can still be moved freely over the abscess, entails a definite risk of pelvic cellulitis or peritonitis. Therefore, if for some reason (e.g. extreme toxæmia) immediate drainage is necessary, it is probably wiser to approach the abscess through an iliac route.

In the great bulk of cases, however, the evacuation of a pelvic abscess is not an urgent matter. Certainly far less risk is run by

waiting until the abscess adheres to the pelvic floor, than by intervening prematurely.

Assuming, therefore, that such adhesion has taken place, the technique of evacuation is as follows :

(1) *Rectal route* (fig. 603). The bladder is first emptied, and the patient is placed in the lithotomy position. The only really painful

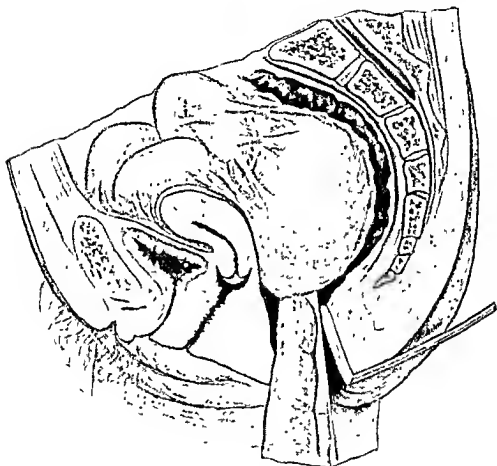


Fig. 603.—RECTAL APPROACH FOR ABSCESS IN POUCH OF DOUGLAS.  
(Modified after Kirschner)

step in the operation is the stretching of the anal sphincter, which must be done thoroughly in all cases. For this reason spinal or gas-oxygen-ether anaesthesia is preferable to the local application or injection of novocaine. The bulge in the anterior wall of the rectum can be seen after stretching the rectum with special retractors or a large speculum. The finger palpates the bulge and discovers the spot where the rectal wall is fixed to the mass. A long aspirating needle is pushed along the finger into the mass, and as soon as pus is seen a scalpel is made to



follow the needle, and the abscess is opened (see fig. 603). The opening is enlarged with a long curved Kocher forceps, and a large drainage-tube introduced into the abscess, and fixed with a stout catgut suture to the rectal wall. The drain should be left undisturbed for several days.

Occasionally troublesome hæmorrhage is said to occur from an artery divided in making the rectal incision, when this vessel will require ligature. Although I have opened many pelvic abscesses by the rectal route, I have not yet experienced serious bleeding.

(2) *Vaginal route.* This should be preferred to the rectal evacuation only when the abscess adheres to the vaginal wall first. There is no difference in the actual technique, but it must be remembered that the bulge is to be looked for on the *posterior* vaginal wall. An anæsthetic is often superfluous, as it may not be necessary to stretch the vaginal orifice before introducing the retractors or speculum, whilst the actual opening of the abscess is only momentarily painful.

### (III) SUBPHRENIC ABSCESS

Although less common than either iliac or pelvic abscess, subphrenic abscess is the most interesting of the localised suppurations in the abdomen. *Ætiologically*, it is related to most of the major surgical affections of the abdomen, while its pathology is complicated by the intricate arrangement and numerous anatomical relations and connections of the sub-diaphragmatic region. Diagnosis is made difficult by the inaccessible position and great depth of the abscess. Finally, the treatment is associated with special difficulties of surgical approach, and particularly with the grave danger of traversing uninfected pleura or peritoneum.

The mortality of subphrenic abscess is extremely high. In the absence of surgical treatment more than 90 per cent of cases die, while the death-rate of operated cases was, until recently, in the neighbourhood of 50 per cent. The reasons for this high mortality are not far to seek. Firstly, in nine cases out of ten the patient is already debilitated by a previous abdominal lesion, e.g. appendicitis, gastro-duodenal perforation, or an infection of the liver or biliary apparatus. Secondly, the inaccessible position of the abscess is responsible for serious delay in diagnosis and treatment. Thirdly, the close proximity of the thoracic contents leads to their implication in the later stages of the

infective process. Fourthly, and lastly, accidental or deliberate contamination of the pleura or peritoneum accounts for many if not most fatal results after operation.

The last twenty years have witnessed a very real improvement in the surgical treatment of subphrenic abscess, and a drop in the mortality of operated cases to less than 20 per cent. This improvement is a reflection of the enormous amount of interest and work recently attracted to the subject. From the publication of Barnard's (1) papers in 1908, with their lucid description of the complicated anatomy of the subphrenic region, numerous papers have been written, and a great deal has been done to facilitate earlier diagnosis and treatment, and particularly to produce methods of surgical approach which are free from the danger of pleural or peritoneal contamination.

#### ANATOMY OF SUB-DIAPHRAGMATIC SPACES

A knowledge of the anatomy of this region is very necessary, both for a clear conception of the ætiology and pathology of subphrenic abscess, and for an appreciation of the essential facts of its treatment.

The subphrenic space is that part of the peritoneal cavity which lies below the diaphragm and above the shelf made by the transverse colon and mesocolon. It is largely occupied by the liver, which divides it into suprahepatic (superior) and infrahepatic (inferior) compartments. Each of these compartments is subdivided into right and left "halves" by the ligaments which "suspend" the liver: the suprahepatic space by the coronary ligament (suspending liver to diaphragm), and the infrahepatic space by the suspensory or falciform ligament (suspending liver to anterior abdominal wall).

The left suprahepatic space is single, but the right suprahepatic compartment is further subdivided by the right lateral ligament into a right antero-superior and a right postero-superior space. Below the liver, this arrangement is reversed, for whilst the right inferior space is single, the left inferior compartment is subdivided by the stomach and omentum into a left antero-inferior and a left postero-inferior space.

There are thus six *intra-peritoneal* subphrenic spaces: three above the liver (left superior, right antero-superior, and right postero-superior), and three below the liver (right inferior, left antero-inferior, and left postero-inferior). There is also an *extra-peritoneal* space between the diaphragm and the peritoneum lining it. According to Vegni (2), this extra-peritoneal space can be divided into an upper and a lower part, by the attachment of the coronary ligament.

A study of figures 604 and 605 shows that the intra-peritoneal spaces are in free communication with one another, and with other regions in the abdomen. Thus, the right postero-superior space joins the right inferior space round the edge of the liver, and both join the right external paracolic sulcus to the outer side of the hepatic flexure. Similarly, the left superior and the left antero-inferior spaces communicate round the left lobe of the liver. In view of these communications, it is not surprising that infection may spread from one space to another, or that it may reach one of the spaces from another part of the abdomen. Usually, however, once a compartment is infected, it

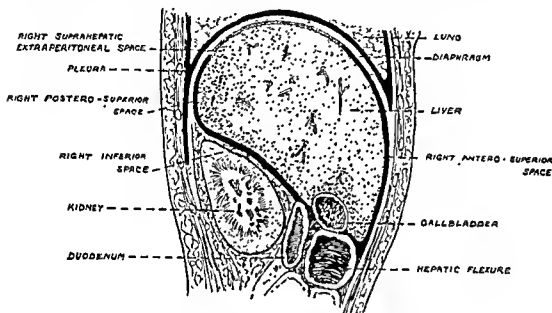


Fig. 604.—SEMI-DIAGRAMMATIC SECTION SHOWING RIGHT SUPRAHEPATIC SPACES.  
(After Woolsey.)

tends to become shut off from its fellows by adhesion of neighbouring peritoneal surfaces, and so the abscess is likely to remain localised to one space only.

We now proceed to a description of the individual spaces, and of their communications.

(1) *Right postero-superior intra-peritoneal space.* This is the most important of the spaces, since it is by far the commonest site of subphrenic abscess. It lies between the diaphragm and that part of the right lobe of the liver which is behind and below the right lateral ligament (see fig. 604). To its left the coronary ligament separates it from the left superior space. Round the free edge of the lateral ligament it communicates with the right antero-superior space, while its

lower end opens into the junction of the right inferior space (renal pouch) and external paracolic sulcus (see fig. 598). This last communication is of great importance, since it represents a common route by which infection reaches the space. Along the paracolic sulcus pus or peritoneal inflammation spreads from the cæcum and appendix, or even from the pelvis, whilst by its junction with the renal pouch the postero-superior space becomes infected from the pyloro-duodenal region or from the gall-bladder.

(2) *Right antero-superior intra-peritoneal space.* This lies in front of the right lateral ligament and to the right of the suspensory or falciform ligament. Round the lateral ligament it joins the right postero-superior space, whilst below it communicates freely, round the sharp edge of the liver, with the right inferior space. Infection of this space mostly comes from the anterior surface of the pyloro-duodenal region and from the gall-bladder.

(3) *Right inferior intra-peritoneal space.* This space corresponds to the right renal pouch, and therefore has the upper part of the kidney and the crural fibres of the diaphragm behind it. Above and in front, it has the right lobe of the liver and the gall-bladder, whilst below is the hepatic flexure of the colon. To the right of the hepatic flexure is the important three-way junction (see fig. 598) at which the inferior space receives the lower end of the postero-superior space, and the upper end of the external paracolic sulcus. To the left of the space is the falciform ligament, the pyloro-duodenal region, the edge of the lesser omentum, and, behind this, the foramen of Winslow, and the ligament of the ductus venosus. Through the foramen the right inferior space normally communicates with the left postero-inferior space (or lessersac).

The sources of possible infection of the right inferior space are thus very numerous. Perforations of the pylorus or duodenum, and infections of the gall-bladder or biliary ducts, may involve the space by rupture or by direct spread into it; also suppuration may spread to it from the right iliac fossa or from the right postero-superior space. It is somewhat of a surprise, therefore, that this is one of the least common sites of subphrenic abscess; Ochsner and Graves (3), for instance, collected 464 cases of abscess in the right postero-superior space, and only 142 in the right inferior space. The explanation is either that infection in this space is overcome without suppuration more frequently than in other spaces, or that the space often becomes obliterated by adhesions before an abscess forms.

(4) *Left superior intra-peritoneal space* (fig. 605). This lies between the left lobe of the liver and the left half of the diaphragm. Behind, it has the left lateral ligament, which separates it from the left postero-inferior space (lesser sac). Below and in front it communicates, round the edge of the left lobe of the liver, with the left antero-inferior space. To the right, it is separated from the right antero-superior space by the suspensory or falciform ligament. The left superior space is only infrequently occupied by a subphrenic abscess.

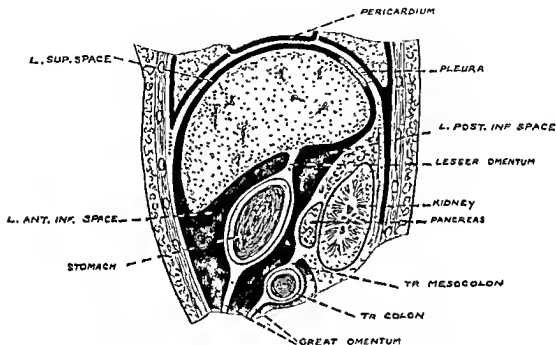


Fig. 605.—SEMI-DIAGRAMMATIC SECTION SHOWING LEFT SUBPHRENIC SPACES.

(5) *Left antero-inferior intra-peritoneal space* (fig. 605). Lying beneath the left lobe of the liver, this space is in front of the lesser omentum, stomach, and greater omentum, which structures intervene between it and the left postero-inferior space, or lesser sac. The falciform ligament separates it from the right inferior space. It is a common site of subphrenic abscess, occupying the second place in Ochsner and Graves' list (3), (309 cases); the usual cause of infection is, of course, a perforated ulcer of the front wall of the stomach.

(6) *Left postero-inferior intra-peritoneal space*. This is the lesser sac of peritoneum (see fig. 605), and is a closed space except for its communication with the right inferior space through Winslow's foramen. In the presence of infection, however, this foramen is very soon obliterated by adhesions, and then the sac becomes completely isolated

from the rest of the peritoneal cavity. The boundaries of the space are: the left lobe of the liver (behind the portal fissure) above; the lesser omentum and stomach in front; the left crus, left suprarenal and kidney, the body of the pancreas, and the transverse colon and mesocolon, behind; and the spleen to the left (see fig. 605). The space also extends for a variable distance downwards into the great omentum, where it forms the omental bursa, and upwards behind the Spigelian lobe of the liver. The lesser sac is rarely occupied by a subphrenic abscess, and the most likely causes of infection are perforation of the back of the stomach, and pancreatitis or operations on the pancreas; it may also be infected by leakage from a retro-colic gastro-intestinal anastomosis, or from a perforated gastro-jejunal ulcer.

(7) *The retroperitoneal spaces.* There is no true space outside the peritoneum, either in the subphrenic region or elsewhere. Instead, there is a layer of areolar fatty tissue, variable in thickness; this layer intervenes between the parietal layer of peritoneum and the abdominal parietes, and also occupies the space between the two leaves of the mesentery, mesocolon, and omenta. The retroperitoneal space in the subphrenic region may be divided into two areas (Vegni, 2) a posterior and an anterior, by the attachment of the upper leaf of the coronary ligament to the diaphragm. This attachment is too firm to be stripped up, and so serves as a definite barrier to the spread of pus from one area to the other.

The *posterior retroperitoneal space* (phrenico-lumbar space) is in direct continuity with the retroperitoneal tissue in the loin, perinephric region, and iliac fossa. It is a common site of subphrenic abscess, the infection spreading up from such causes as a retro-cæcal appendix, perinephric abscess, pancreatitis, diverticulitis, or suppurating lumbar or iliac glands. The space extends up as far as the upper leaf of the coronary ligament, and so comes into direct relation with the "bare areas" on the back of the liver and the corresponding part of the diaphragm; it is thus evident that infection may also reach this space from the liver, and even from the thorax.

The *anterior retroperitoneal space*, in front of the coronary ligament, is in direct continuity only with the extra-peritoneal tissues of the anterior abdominal wall. It is, therefore, but rarely the seat of a subphrenic abscess, the most likely source of infection being the lymphatics of the round ligament (coming from the umbilicus), and the lymphatics in the rectus sheath.

## PATHOLOGY OF SUBPHRENIC SUPPURATION

In the study of the pathology of subphrenic abscess two things stand out: one is the *routes* by which infection can reach the spaces beneath the diaphragm, and the other is the *causes* of such infection. Before considering these, however, brief reference may be made to other aspects of the pathology of the lesion.

Firstly, it should be noted that not all cases of subphrenic infection proceed to abscess formation. A subphrenic peritonitis must be a common complication of acute infections of the upper abdomen, e.g. gastro-duodenal perforation and cholecystitis, but a subphrenic abscess develops in only a small proportion of such lesions. Similarly, an ascending infection of the peritoneum, or retroperitoneal tissues, probably occurs in a large proportion of cases of acute appendicitis, but in most of them the infection subsides without abscess formation; the actual incidence of subphrenic abscess after infections of the appendix is not much more than 1 per cent. Many cases of subphrenic infection without abscess undoubtedly remain undiagnosed; but even of diagnosed cases only 30 per cent proceed to suppuration (Ochsner and Graves). It would appear, from these facts, that the peritoneum in the upper abdomen is gifted with exceptional powers to cope with and overcome infection by bacteria.

The *bacteriology* of subphrenic abscess does not differ to any material degree from that of diffuse peritonitis. The *B. coli* and the *streptococcus* are the organisms most commonly found, while the *staphylococcus* appears to be less frequent. The infection is often mixed.

The *morbid anatomy* is that of any acute localised suppuration. The size of the abscess is determined by the limits of expansion of the space in which it forms. In spite of the free inter-communication of the various spaces, the abscess tends to limit itself to one of them, since the communications are shut off by adhesions quite early in the infective process. The only common example of suppuration in more than one space is seen in the combined involvement of the right postero-superior and the right inferior intra-peritoneal spaces in infections of gastro-duodenal or appendicular origin. Lastly, it should be observed that an abscess does not necessarily occupy the whole of an infected space; frequently the suppurative process is limited to one part of the space, the remainder of it being shut off by adhesions.

The *fate* of untreated subphrenic abscesses varies. A very small

percentage *resolve* completely, except for the formation of local adhesions. A much more common termination, however, is *rupture* of the abscess. Suprahepatic abscesses tend to rupture through the diaphragm into the *pleura*, and thus to terminate in a septic pleurisy; not infrequently the diaphragmatic pleura becomes obliterated by adhesions as the abscess approaches it, and so the latter bursts into the *lung*, with the production of a fatal pulmonary abscess, or gangrene of the lung. Occasionally, a suprahepatic abscess ruptures into the pericardium. Infrahepatic abscesses, on the other hand, generally burst into the main peritoneal cavity, with the production of a rapidly fatal *peritonitis*; less often they rupture into a hollow viscus, or even track through the abdominal wall and burst externally, in both cases possibly resulting in a natural cure.

It should be noted that *thoracic complications* may occur without rupture of the abscess through the diaphragm. An irritative serous pleurisy is a common and relatively early complication of abscess in one of the suprahepatic spaces, but the other and more serious complications (empyema, pericarditis, pneumonia, pulmonary abscess or gangrene, broncho-pleural fistula) all occur late in the course of the disease, and are therefore partly attributable to delayed diagnosis and treatment.

*Incidence of Abscess in the various subphrenic spaces.* Most published series of cases show that the right postero-superior intra-peritoneal space is the commonest site of subphrenic abscess, that the left antero-inferior and the retroperitoneal spaces come next in order of frequency, and that the left postero-inferior space, or lesser sac, is the least common site. The relative frequency of involvement of the various spaces is well shown in the following table, which is based on the 1564 cases, collected by Ochsner and Graves (3):

Right postero-superior space . . . . .	464 cases.	(30%)
Left antero-inferior space . . . . .	309 "	(20%)
Retroperitoneal space . . . . .	221 "	(14%)
Right antero-superior space. . . . .	197 "	(11.5%)
Right inferior (renal pouch) space . . . . .	142 "	(9%)
Left superior space . . . . .	56 "	(3.5%)
Left postero-inferior (lesser sac) space . . . . .	47 "	(3%)
Combined . . . . .	56 "	
Not determined . . . . .	72 "	
<b>Total . . . . .</b>	<b>1564 cases.</b>	



*Routes of Infection.* The following are the routes by which infection may reach the subphrenic region :

(i) *Direct infection* from a viscus in the immediate vicinity of one of the spaces. E.g. a gastric ulcer may perforate into the left antero-inferior space, a duodenal ulcer or the gall-bladder may infect the right inferior space, while a liver abscess may burst into one of the suprahepatic spaces.

(ii) *Intra peritoneal spread* from distal foci of suppuration. This is a common route in subphrenic abscess of appendicular or pelvic origin, the purulent effusion spreading up along the external paracolic sulcus (see page 1100) from the right iliac fossa or pelvis. Reaching the junction of the sulcus with the right inferior and right postero-superior spaces (see fig. 598), the pus may infect one (usually the postero-superior) or both of them. Similarly, an effusion from a perforated duodenal ulcer, although at first located in the inferior space, may spread round the edge of the liver, and so infect either the antero-superior or posterior-superior space. It is obvious that this type of spread is limited to cases in which a purulent effusion is already present in some part of the peritoneal cavity, and also that it may possibly be favoured by the adoption of the horizontal position. We must observe, however, that infection can also spread by direct extension along the peritoneal membrane, quite independently of the movement of fluids, and that such spread cannot be controlled by alterations of posture.

(iii) *Lymphatic spread* from distal or proximal foci of infection (not necessarily suppurative). That bacterial infection may extend along the peritoneal and extra-peritoneal lymphatics may be regarded as an established fact. Starting in an intra-peritoneal viscus, such as the appendix, the organisms invade the lymphatics in the mesentery of the viscus, and so reach the lymphatic plexus immediately outside the parietal peritoneum. Along this plexus the infection passes to the subphrenic region, where it may cause either an intra-peritoneal or a retroperitoneal abscess.

The lymphatic route probably accounts for most subphrenic abscesses which complicate non-purulent appendicitis or cholecystitis, and particularly for the cases in which an *interval* of some length intervenes between the causative disease and the development of the abscess. It also provides a reasonable explanation for cases in which the abscess is associated with a causative lesion at some distance, the intervening part of the peritoneum being apparently healthy, as well as for some

cases of post-operative abscess, particularly those which follow a "cold" appendicectomy. Furthermore, a trans-diaphragmatic lymphatic route of infection is probably responsible for most subphrenic abscesses which complicate intra-thoracic suppuration.

(iv) *Spread along retroperitoneal cellular tissues.* Most retroperitoneal subphrenic abscesses are produced by a spread of infection along the loose areolar tissue behind the peritoneum of the loin and iliac fossa. The original focus of infection may itself be retroperitoneal, e.g. perinephric abscess, posterior duodenal ulcer, pancreatitis, etc., or it may reach the extra-peritoneal tissues from an inflamed viscus adherent to the parietal peritoneum, e.g. a retro-cæcal appendix; or, finally, the infection may extend from an intra-peritoneal viscus along the cellular tissue of its mesentery, and so reach the retroperitoneal plane.

All the above conditions produce the common *posterior* retroperitoneal subphrenic abscess, i.e. the one limited above by the attachment of the upper leaf of the coronary ligament to the diaphragm. The rare instances of abscess in the *anterior* retroperitoneal subphrenic space are explained by spread of infection along the cellular tissue or lymphatics of the round ligament or rectus sheath, from septic lesions of the umbilicus and anterior abdominal wall.

(v) *Spread via the blood stream.* Subphrenic abscess of hæmogenous origin is definitely rare. Cases have been observed in which infection spreads along the *portal vein* (pylephlebitis) causing, in the first place, a liver abscess, which subsequently bursts into and infects one of the subphrenic spaces. Other cases have been reported as occurring in the course of a *systemic pyæmia*.

*Causes of Subphrenic Abscess.* About 90 per cent of subphrenic abscesses follow infective lesions in the abdomen, of which appendicitis, gastro-duodenal lesions, and infections of the liver and biliary apparatus are by far the most common. The *appendix* is responsible for about a third of all subphrenic abscesses. Fifield and Love (4) give a figure of 35.7 per cent, Perry (5) of 25.3 per cent, and Ochsner and Graves (3) of 31 per cent. *Gastro-duodenal perforations* represent a cause very nearly as common (28 per cent in Fifield and Love's cases, and 29 per cent of the cases collected by Ochsner and Graves). Infection of the *liver* and *bile passages* is the next most common cause, accounting for about 10 per cent of cases.

Other *intra-peritoneal* causes are: carcinoma of the stomach or

intestine, operations on the stomach or intestine, pelvic disorders, and infective lesions of the stomach, intestine, and spleen. Among *extra-peritoneal* sources in the abdomen, the most common are the abdominal wall, the kidney, the perinephric region, the pancreas, and the skeleton (vertebræ, pelvis, and ribs). Infective lesions in the *thorax* were responsible for 2·5 per cent, and blood-borne infection for 3·5 per cent, of the 3000 odd cases of subphrenic abscess collected by Ochsner and Graves.

(1) *Appendicular origin.* A subphrenic abscess of appendicular origin occurs mostly in the course of an acute suppurative or perforative appendicitis, or follows an "acute" appendicectomy. Less often it complicates a sub-acute or catarrhal appendix, and it may even develop in cases of chronic appendicitis or after a "cold" appendicectomy. The development of an appendix abscess, in the iliac fossa or pelvis, greatly enhances the danger of involvement of the subphrenic spaces; whilst other predisposing causes are: a high cæcum, an upward-pointing appendix, a retro-cæcal appendix, and adhesions to the posterior peritoneum. But undoubtedly the chief predisposing cause is *delay in removing an inflamed appendix*, whilst perhaps the most common reason for the development of subphrenic abscess after appendicectomy is the *failure to drain an infected appendix bed*.

Although infection of the subphrenic region must be a fairly common event in appendicular disease, the actual incidence of subphrenic abscess in appendicitis is not more than between 1 and 1·5 per cent. The proportion of subphrenic abscess in residual abscesses of appendicular origin varies in the literature between 6·6 and 37 per cent, but is probably not more than 8 per cent (Ochsner, 6).

One route by which the infection extends from the appendix is along the *external paracolic sulcus*. In many cases there is an actual spread of pus up the sulcus, which reaches the right inferior space (renal pouch), and either stays there, or more often passes on to the right postero-superior space; not infrequently both spaces are infected simultaneously, or one from the other. In other cases the infection spreads by direct extension along the peritoneal membrane, without any actual movement of fluid. The *lymphatic route* is probably responsible for cases following catarrhal appendicitis, especially when there is a clear interval before subphrenic symptoms appear, and also for abscesses which occur after non-purulent appendicectomies. But perhaps the commonest route of infection is along the *retroperitoneal* cellular tissues, resulting in the production of a posterior retroperitoneal

subphrenic abscess; such an abscess cannot spread up beyond the coronary ligament, but it may burst above it into the right antero-superior space.

It certainly seems that the retroperitoneal route is responsible for most cases of actual abscess. The explanation is that whilst the peritoneum has a high resistance and is able more often than not to abort the infective process before actual suppuration occurs, the cellular tissue outside it does not possess this resistance and so is less able to overcome the tendency to abscess-formation.

(2) *Gastro-duodenal origin.* Acute or sub-acute perforations of gastric and duodenal ulcers are responsible for the large majority of subphrenic abscesses of gastro-duodenal origin. Less common causes are operations on the stomach, carcinoma, and perigastritis from a chronic or "leaking" perforation. The abscess is peculiar in that it may contain free gas. The site of the abscess is determined by the position of the ulcer and the disposition of perigastric adhesions.

Abscesses of *gastric* origin involve the left subphrenic spaces. Since perforating ulcers are nearly always anterior, the most common space to be affected is the left antero-inferior, but occasionally the suppuration extends to the left superior space. Perforating ulcers of the posterior wall of the stomach are unusual, and if subphrenic abscess results, it will be limited to the left postero-inferior space, or lesser sac.

Abscesses of *pyloric* and *duodenal* origin occupy the right subphrenic spaces. In the first place, the right inferior space (or renal pouch) will be contaminated, and an abscess may occur here, and become localised by the formation of adhesions between the liver and parietes. More often, the pus spreads up to one of the right suprahepatic spaces, and abscesses form both below and above the liver. Not infrequently, however, the inferior space is obliterated by adhesions between the liver and gall-bladder, and the abscess will then be entirely suprahepatic (antero-superior or postero-superior space).

(3) *Hepato-biliary origin.* These abscesses tend to be less acute than those of appendicular or gastro-duodenal origin. Marked differences exist between subphrenic suppuration of hepatic and biliary origin.

(a) The *hepatic* causes of subphrenic suppuration are amœbic abscess of the liver, pyæmic abscess, and infected hydatid cyst. Although the natural tendency for all these lesions is to spread upwards towards the suprahepatic spaces, a subphrenic abscess results but rarely,

because these spaces are usually obliterated by an associated perihepatitis or by perihepatic adhesions. The consequence is that the liver abscess tends to go through the diaphragm into the thorax, rather than to burst into the peritoneal spaces above the liver.

A subphrenic abscess of hepatic origin is almost always suprahepatic, involving particularly the right postero-superior and antero-superior spaces. It may be distinguished by the presence of chocolate-coloured pus or by hydatid hooklets, but it *does not contain bile*.

(b) A *biliary* origin is much more common. Rupture, perforation, or gangrene of the inflamed gall-bladder are the usual causes, but infection may occur by migration of organisms through the wall of an inflamed gall-bladder or bile-duct, and also via the lymphatics, or along inflammatory adhesions. Furthermore, a subphrenic abscess may occur as a post-operative complication, after operations on the gall-bladder or bile-ducts.

Primarily, an abscess of biliary origin tends to form in the right inferior space, but it may extend to the right superior spaces, unless the communications are obliterated by adhesions. It should be observed that the suprahepatic spaces may be attacked directly, the infection in this case travelling along the lymphatics. Abscesses of biliary origin are characterised by *containing fætid bile*.

#### DIAGNOSIS OF SUBPHRENIC ABSCESS

The early diagnosis of subphrenic abscess is not easy. One reason is the deep and inaccessible position of the abscess; another is that the clinical picture varies within wide limits, the differences depending on the onset and course of the disease, and also on the particular space involved. Furthermore, the symptoms of the causative lesion often mask the initial features of the abscess, and the picture may be confused still more by the presence of thoracic symptoms and signs.

Cases with an insidious onset and indefinite symptoms may remain undiagnosed for long periods, particularly if they follow, as they often do, a somewhat obscure abdominal lesion. I have operated upon a large abscess which remained undiagnosed for more than two months, and cases are recorded in the literature in which diagnosis was not made until many months after the onset of the abscess (Lockwood, 7 and Russell, 8). Unfortunately, delayed diagnosis necessarily implies delayed treatment, which is beyond question an important cause of the high mortality of the disease, mainly because the longer the abscess remains unopened the greater is the chance of thoracic complications

developing. Whilst it must be admitted that early diagnosis is often hedged in with difficulties, we submit that much of the delay which is usually experienced can be prevented. Timely diagnosis may be made in all cases, provided the clinician has a clear conception of the various clinical pictures presented by the lesion, and also is ready to regard with suspicion every abdominal case which fails to improve as it should, or which seems to relapse after an interval of complete or partial recovery. Careful and, if necessary, repeated examinations will very soon confirm or disprove his suspicions.

*Clinical Varieties.* The great bulk of cases of subphrenic abscess, as seen nowadays, fall into one of three clinical groups.

(i) Cases with *sudden onset, acute symptoms, and rapid course.* This group usually follows lesions which cause severe contamination of the peritoneum, e.g. perforated gastro-duodenal ulcer or perforative appendicitis or cholecystitis. Less than a third of all cases of subphrenic abscess come under this heading, and they are, of course, the easiest to diagnose early. As a rule the picture is at first one of acute diffuse peritonitis, and then, without any interval, this partially subsides, and the picture changes to that of a circumscribed abscess.

(ii) Cases with *insidious onset, sub-acute symptoms, and more gradual course.* This group follows non-perforative infective lesions in the abdomen, particularly those not associated with suppurative peritonitis. Very often the infection reaches the subphrenic region from a distal focus, such as the appendix, by means of the lymphatic or retro-peritoneal cellular route; therefore, in many cases of this group, there is a clear *interval* between the subsidence of the original infection and the onset of subphrenic symptoms. In other cases, however, no such interval exists, the symptoms of subphrenic infection being grafted imperceptibly on to the symptoms of the causative lesion, which are often somewhat obscure. This group is probably a little more numerous than the last, and is the one in which delayed diagnosis is most common.

(iii) Cases of *post-operative* subphrenic abscess, i.e. those following operation for a suppurative or other infective lesion in the abdomen. In these days this is undoubtedly the largest group, accounting for 40 per cent or more of all cases. As a rule an *interval* of some days intervenes between the operation and the commencement of subphrenic symptoms, but the onset is not necessarily insidious, nor are the symptoms always sub-acute. In some cases the subphrenic region is already infected (although not obviously) before the operation is

embarked upon, and the fact that the subphrenic abscess is post-operative is a mere coincidence. In other cases, however, the operative procedure is to blame, in part at least, for the subsequent development of the abscess.

*Clinical Features.* The onset varies, and there may or may not be an interval between the symptoms of the causative lesion and those of subphrenic infection. Abscesses with an *acute* onset, e.g. after gastroduodenal perforation, tend to follow the causative lesion without a clear interval.

A distinction must be made between subphrenic *infection* and subphrenic *abscess*, since, as already stated, most cases of the former subside without an actual abscess resulting; moreover, it would be a serious error to operate on a case before pus had formed.

(1) *Subphrenic Infection (Subphrenitis).* The first symptoms of subphrenic infection are usually constitutional. The patient is recovering, or has just recovered from an abdominal infection (e.g. appendicitis or perforated ulcer), or has recently been operated upon for such a condition. Then things begin to go wrong. The temperature, which had been coming down, or had even reached normal, begins to go up again, and the chart shows a definite *pyrexia*. The patient feels ill, the tongue becomes furred, and there may be a rise in the pulse-rate.

The first local symptom is pain, perhaps with a sensation of pressure, both of which are increased by and may interfere with *deep breathing*. Sometimes the pain is intense from the start, but more often it is slight at first and increases later. The situation of the pain varies with the space. In the common right postero-superior space infection it is mostly felt in the back and side of the lower right chest, but it may also be referred to the right shoulder. In retroperitoneal infection the pain may start in the loin, while in subhepatic intra-peritoneal infections it is mostly complained of along the respective costal margin. Another common symptom is a painful dry cough, whilst a little later there may be actual *dyspnœa*.

On examination one sign is always present, and this is *local tenderness*. The site of the tenderness again varies with the space involved. Vegni refers to three "tender points": (i) the *anterior phrenic spot*, where the lower border of the tenth rib crosses the mid-clavicular line (anterior suprahepatic and subhepatic spaces); (ii) the *posterior phrenic spot*, at the posterior end of the last intercostal space (posterior suprahepatic and retroperitoneal); (iii) the *superior phrenic spot*, just

above and between the two beads of origin of the sterno-mastoid muscle (involvement of diaphragm itself). Although these definite "spots" of tenderness are sometimes encountered, more often the tenderness is elicited over somewhat larger areas. Thus, persistent tenderness over the *right twelfth rib* is almost diagnostic of right postero-superior space infection, tenderness in the *right flank* suggests retro-peritoneal infection, whilst deep tenderness along the *costal margin* indicates infection of the antero-superior or inferior spaces on the right side, and the superior or antero-inferior spaces on the left.

In the absence of an actual abscess, *examination of the chest* may reveal nothing abnormal, except perhaps a diminished respiratory excursion and poor air-entry on the suspected side. Similarly, *radio-graphy* is only likely to show relative loss of movement in the affected half of the diaphragm.

(2) *Subphrenic Abscess*. The above symptoms and signs enable us to diagnose an infection in one of the subphrenic spaces (subphrenitis), and although they may subside spontaneously, they should warn us to anticipate the additional features which indicate that an abscess is definitely forming. If the case resolves without them, so much the better, but it is only by detecting the symptoms of suppuration at their first appearance that we can hope to intervene with the best prospects of success.

With the formation and accumulation of pus, the *fever* becomes higher and more *remittent* in type. The *pulse-rate* is more rapid, night-sweats and a hectic flush may appear, and the patient begins to lose flesh. An extremely important and significant event is the discovery of *leucocytosis* on examination of the blood. *Pain* increases, *respiration* becomes more restricted and dyspnoic, and the "diaphragmatic" *cough* gets more worrying; indeed, some cases present a picture sufficiently "thoracic" to suggest an acute pleuro-pneumonia.

*Examination of the abdomen* shows increased tenderness over the appropriate spots or regions. Suprahepatic abscesses may push the liver down, increasing the *hepatic dullness*, and even bringing the liver edge well below the costal margin, where it can be felt. Intrahepatic abscesses usually cause *rigidity* of the corresponding upper rectus, and may even show a slight hudge at or below the costal margin; while posterior retroperitoneal suppuration may produce a swelling of the loin. In a small number of cases an accumulation of *free gas* in the abscess may be shown by resonance on percussion, perhaps with obliteration of the liver dullness.



*Examination of the chest* reveals variable signs, but true thoracic complications are late in development and should never be waited for. An accumulation of fluid in the homolateral pleura, however, may be detected quite early, and is not necessarily of serious significance.

*Radiographic examination* is of the greatest possible value in the detection of a subphrenic abscess. Radiograms should be taken in the upright position, and antero-posterior and lateral views obtained on full inspiration and expiration. *Limitation of movement* of one half of the diaphragm is the first abnormality to be detected; as already explained, this suggests subphrenic inflammation, but not necessarily an abscess. *Elevation* of the affected half of the diaphragm, however, is definite evidence of abscess, and is shown in nearly 90 per cent of cases. In the common right suprahepatic abscess the antero-posterior view shows obliteration of the costo-phrenic angle, while the lateral view shows obliteration of the posterior costo-phrenic angle. A gas-containing cavity is seen only in a small proportion of late cases, and is of little diagnostic value.

*Aspiration* should be condemned as both dangerous and unnecessary. It incurs a definite risk of infection of the clean pleura and peritoneum, and more than one case of fatal empyema or peritonitis has been directly attributable to it. If practised at all, it should be done only as an immediate preliminary to operation, and the needle must on no account traverse the pleura or peritoneal cavity (e.g. insert needle from beneath the twelfth rib). In doubtful cases operative exploration of a suspected space is a safer procedure than aspiration.

Before proceeding to operation, it is necessary not only to diagnose the presence of a subphrenic abscess, but also to localise it to a particular space, or at least to decide whether it is right or left, and whether it is anterior or posterior. In the interests of brevity the symptoms and signs of individual space infections are summarised in the form of a table (overleaf).

#### TREATMENT OF SUBPHRENIC ABSCESS

*Prevention.* Prompt and efficient treatment of causative lesions would undoubtedly prevent or abort most cases of subphrenic suppuration. The immediate removal of inflamed appendices is particularly important in this connection, and we certainly cannot accept the view that the conservative treatment of appendicitis diminishes the danger of subphrenic infection (Fifield and Love, 4). On the contrary, it is more than probable that subphrenic abscess of appendicular origin

## DIFFERENTIAL DIAGNOSIS OF INDIVIDUAL SPACE INFECTIONS

Space.	Causative Lesions.	Pain.	Tenderness	Thoracic Symptoms (cough, limited breathing, etc.).	Thoracic Signs (dullness, poor air-entry, etc.).	Rigidity.	Swelling.	Liver.
R. post.-sup.	Appendicitis, liver abscess, etc.	Lower back and side of right chest, perhaps right shoulder.	Right twelfth rib and last space.	Yes.	Yes (right base).			Pushed down, palpable edge, dullness increased.
R. ant.-sup.	Perf. duod. ulcer. Cholecystitis.	Right hypochondrium.	Right costal margin.	Yes.	Yes (right base).			Pushed down still more.
R. inf.	Perf. ulcer. Cholecystitis. Appendicitis.	Right costal region.	Right costal margin and below.			Upper right rectus.	Below right costal margin.	
L. sup.	Perf. gastric ulcer (ant. wall).	Left hypochondrium, perhaps left shoulder.	Left costal margin.	Yes.	Yes (left base).			
L. ant.-inf.	Perf. gastric ulcer (ant. wall).	Left costal margin.	Left costal margin and below.			Upper left rectus.	Below left costal margin.	
L. post.-inf.	Perf. ulcer (post. wall). Pancreatitis.	Back of lower left ribs.	Left twelfth rib and last space.					
Retros. peritoneal.	Appendicitis. Perinephric abscess, etc.	Loin and lower back thorax.	Loin and last rib.	Often present.	Often present.	Loan and flank.	Loin and flank.	May be pushed down.

would vanish completely if every acute appendix was removed within twelve hours of its onset. Similarly, immediate closure of perforated gastric or duodenal ulcers would materially diminish the incidence of sub-diaphragmatic peritonitis. A further protection is afforded by drainage of all foci of abdominal sepsis; this should never be omitted if there is the least chance of infection of the peritoneum.

The adoption of Fowler's position after operations for peritonitis or septic abdominal lesions is of much less value than was formerly thought. It may prove of some service in the presence of an abundant purulent exudate, but it does not prevent direct or lymphatic spread of infection. To be effective, at least 60 degrees of elevation is necessary.

*Conservative Treatment.* Unless symptoms or signs are present which definitely indicate or suggest an abscess (high or remittent fever, leucocytosis, increased pain and tenderness, elevation of diaphragm, etc.), the case should at first be treated on conservative lines. The causative lesion, if not already removed, must be promptly dealt with and, if necessary, drained. The suspected half of the subphrenic region should be immobilised, as far as this can be done, by firmly strapping the lower ribs and adjacent abdominal wall with adhesive plaster. Local application of heat is of value, particularly in the form of an electrically heated pad. Bacteriological therapy may be tried if causative organisms have been isolated, and everything possible should be done to maintain the patient's nutrition and strength.

In most cases of subphrenic inflammation these measures will succeed and an abscess may thus be aborted; the fever subsides in two or three days, the pain and tenderness diminish, and the convalescence from the original lesion or operation is resumed. A careful watch must be kept, however, for the signs of suppuration, and a thorough examination carried out at least once daily.

*Operative Treatment.* As soon as an abscess is diagnosed, or strongly suspected, operation becomes imperative, and must be undertaken without delay. The object of the operation is to open, evacuate, and drain the abscess.

Statistics show that cases operated on early do not develop *thoracic complications*, but that such complications are common in late cases; thus of 31 presumably late cases recently reported by Beyer (9) no less than 23 had intra-thoracic lesions, 15 showing an actual perforation of the diaphragm! The significance of thoracic

complications lies in the fact that they more than double the mortality of subphrenic abscess; Ochsner and Graves show that the death-rate of cases with pleuro-pulmonary lesions is about 50 per cent, whilst that of cases without them is under 25 per cent.

Apart from the *time-factor*, the other vital matter concerned with operative treatment is the *approach to the abscess*. The situation of the abscess deeply within the thoracic cage makes access to it difficult; figure 606 illustrates the various routes by which the abscess can

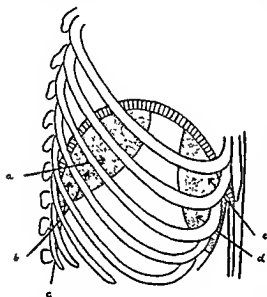


Fig 606.—ALTERNATIVE ROUTES OF APPROACH TO ANTERIOR AND POSTERIOR SUPRAPHRENIC SPACES.

- a=TRANSPLEURAL.
- b=EXTRA PLEURAL.
- c=POSTERIOR EXTRA PERITONEAL.
- d=ANTERIOR TRANSPERITONEAL.
- e=ANTERIOR EXTRA-PERITONEAL.

be reached, i.e. transpleurally, transperitoneally, extra-pleurally, and extra-peritoneally.

For many years it has been customary to approach the abscess by a *trans-serous* (trans-pleural or transperitoneal) route, and surgeons have adhered to it slavishly in spite of a very high mortality. While it is true that the trans-serous route affords the most direct approach to the majority of subphrenic abscesses, it carries with it a very real risk of infection to uninvolved parts of the pleura and peritoneum, and therefore a grave danger of causing a suppurative pleurisy or peritonitis. In view of these obvious and serious drawbacks,

the persistent employment of the trans serous route shows a curious lack of understanding; also it can often be attributed to failure on the part of the surgeon to localise the abscess, and to plan his approach before embarking on the operation.

The *transpleural route* has the doubtful privilege of being "classical." Its advocates attempted to minimise the dangers of pleural contamination by ingenious devices, but unfortunately without appreciable difference to the mortality. Thus, Trendelenburg (10), in 1883, suggested that the part of the pleural cavity which has to be transversed in the transpleural approach may be isolated, after the resection of one or more ribs, by a circle of sutures stitching the costal and diaphragmatic layers of the pleura together; this procedure has been copied

from text-book to text-book for nearly fifty years, but experience has proved it to be worthless. A few years later, 1889, Boeckel (11) pleaded that a low transthoracic approach could miss the phrenico-costal sulcus of pleura, as this is pulled up out of the way by the elevation of the diaphragm; this comforting suggestion has been used as an excuse for the transthoracic approach by numerous surgeons, but recent work proves that it has no foundation in fact. Realising the dangers of the transpleural route, its advocates finally brought forward a *two-stage procedure*, the first stage ending with the resection of two or more ribs; unless the pleura over the abscess has already been obliterated by adhesions, these are encouraged to form by packing iodoform gauze against the pleura. A week or so later the pleural cavity in the floor of the wound will be obliterated, and an incision can be made through it and the diaphragm into the abscess (second stage). We cannot speak of this procedure from experience, but apart from other considerations, the delay of a week condemns it.

The *transperitoneal route* (for anterior subphrenic abscesses) is subject to the risk of infecting the peritoneal cavity. Carefully packing off the approach to the abscess, and complete evacuation of the pus with a sucker, diminish this danger, but do not remove it entirely. Neither is the danger of peritonitis completely obviated by a two-stage transperitoneal operation.

It is our firm conviction that the transpleural and transperitoneal routes should be abandoned as surgically unsound and dangerous, especially in early cases. In neglected cases the danger is not so great, since there is a good prospect of the pleura or peritoneum over the abscess being obliterated by adhesions. But as we can never be certain of this, and we must above all provide for *early intervention*, only the safer *extra-serous* operations will be described in this article.

*The Extra-serous Approach.* The advantages of evacuating a subphrenic abscess without contamination of healthy serous membrane are obvious, and it is not surprising that extra-pleural and extra-peritoneal methods of approach show a lower mortality than do trans-serous methods. Thus, Ochsner and Graves collected 305 cases of transpleural evacuation with a death-rate of 39 per cent, 307 cases of transperitoneal evacuation with a death-rate of 35.5 per cent, and 189 cases of extra-serous evacuation with a death-rate of 21 per cent. These figures are made more significant by the inclusion among the trans-serous groups of a large number of cases in which the serous cavity over the abscess was already obliterated by adhesions. When the free

pleura or peritoneum is opened the mortality must be expected to exceed 50 per cent.

Of the two extra-serous procedures, the *extra-peritoneal* should be preferred to the extra-pleural, for the very simple reason that it is usually easier to separate the peritoneum from the diaphragm than the pleura; furthermore, it has proved the safer of the two methods. I have employed the extra-peritoneal route in four cases, with no mortality, while Ochsner and Graves report 31 personal cases with the very low death-rate of 9.7 per cent.

There are two methods of extra-peritoneal approach: (i) the *posterior approach*, through which the right postero-superior, right

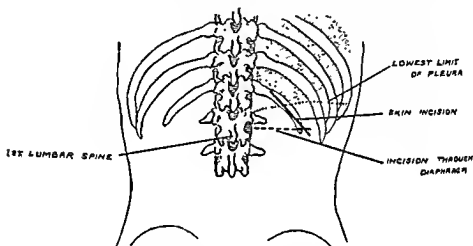


Fig. 607.—DIAGRAM ILLUSTRATING POSTERIOR EXTRA-PERITONEAL APPROACH FOR RIGHT POSTERO-SUPERIOR SUBPHRENIC ABSCESS.

inferior, left postero-inferior, and retroperitoneal spaces can be reached; and (ii) the *anterior approach*, through which the right antero-superior, the left superior, and the left antero-inferior spaces can be reached.

(1) *The Posterior Extra-peritoneal Operation.* On the right side this is the ideal approach for abscesses in the right postero-superior space, the retroperitoneal space and the right inferior space, which, between them, constitute the site of about 70 per cent of subphrenic abscesses. On the left side it is the best approach for the rare abscess in the left postero-inferior space, or lesser sac.

The technique of the operation, assuming that it is performed for a right-sided abscess, is as follows: the patient lies on the left side, with the table angulated in the "renal position," or with a sand-bag under the loin. Under gas-oxygen-ether, or paravertebral anaesthesia, an incision is made along and down to the right last rib (see fig. 607).

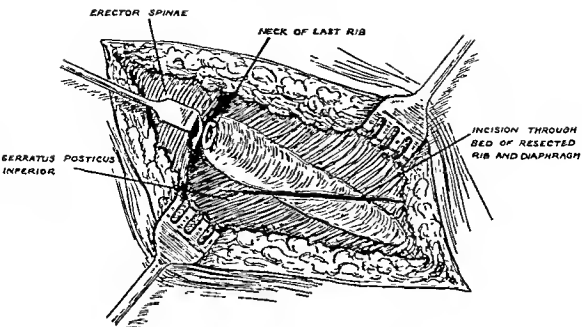


Fig. 608.—POSTERIOR EXTRA-PERITONEAL APPROACH FOR RIGHT SUBPHRENIC ABSCESS (FIRST STAGE). (After Ochsner and Graves.)

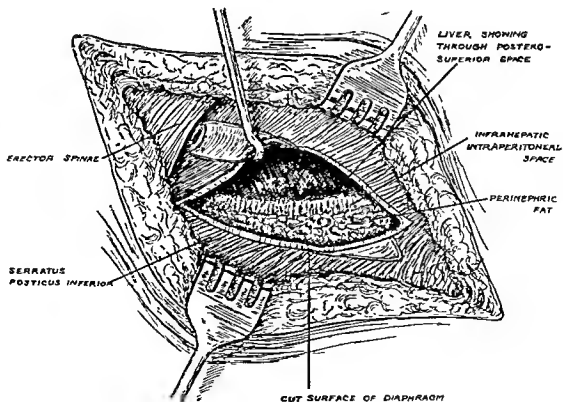


Fig. 609.—POSTERIOR EXTRA-PERITONEAL APPROACH FOR RIGHT SUBPHRENIC ABSCESS (SECOND STAGE). (After Ochsner and Graves.)

The last rib is resected subperiosteally; Doherty and Rowlands (12) advise that the resection should not include the neck of the rib, to avoid injury to the pleura, which usually comes down below it, but this danger can also be obviated by making the resection strictly a subperiosteal one. The next step of the operation is the most important one: after retracting the erector spinæ, a *transverse* incision is made across (not parallel to) the bed of the resected rib, dividing the spinal attachment of the *diaphragm* at the level of the first lumbar spine (see

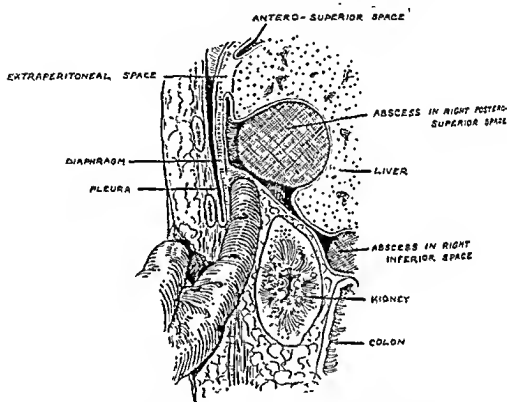


FIG. 610.—ILLUSTRATING SEPARATION OF PERITONEUM FROM DIAPHRAGM IN POSTERIOR EXTRA PERITONEAL APPROACH FOR SUBPHRENIC ABSCESS. (Modified from Ochsner and Graves.)

fig. 608). By making the incision strictly horizontal, and by keeping it at the level of the first lumbar spine, injury to the pleura can be avoided with absolute certainty.

After dividing the diaphragm (which may here be only a thin sheet of muscle-fibres) the perinephric fascia is seen. This is displaced downwards, with the kidney, until the lower edge of the liver with the subhepatic space of peritoneum is seen and felt (fig. 609). The subhepatic (right inferior) space is carefully palpated for a possible abscess, and any doubt is settled by aspiration; if an abscess is found here it is opened with the finger or a sinus forceps and drained. A retroperi-



toneal collection of pus will also show itself by now, or in the early part of the next stage of the operation. This stage consists in peeling off the peritoneum from the under-surface of the diaphragm, with the object of reaching an abscess in the right postero-superior space. This separation can be effected with the finger (see fig. 610), and is made easier by the œdematous condition of the peritoneum when an abscess is present near it. To facilitate the separation of the peritoneum or pleura from the diaphragm, I have employed a small rubber balloon, which is introduced collapsed between them, and then blown up by reversing an electrically driven sucker. The separation is continued

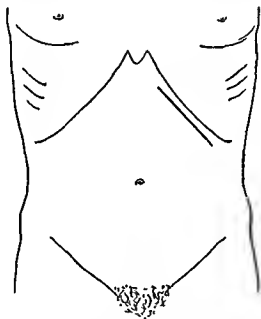


Fig. 611.—INCISION IN EXTRA-PERITONEAL APPROACH FOR LEFT ANTERIOR SUPRACOSTAL ABSCESS.

until an indurated mass is felt, which is the abscess. Any doubt is settled by aspirating the mass, after which the abscess is opened by pushing the finger through its wall, and the pus emptied with the sucker. A large drainage-tube is inserted into the abscess cavity and fixed to the skin, and the wound loosely sutured.

(2) *The Anterior Extra-peritoneal Operation.* On the right side this is the best approach for abscesses in the right antero-superior space; it can also be employed for a right inferior abscess if one can feel certain that the right postero-superior space is not also involved, but it is less suitable for drainage than the posterior approach. On the left side it is the ideal approach for abscesses in the left superior and the left antero-inferior spaces.

An incision is made immediately below and parallel to the costal margin on the affected side (fig. 611). Everything is divided in the line of the incision, down to, *but not including*, the peritoneum. The peritoneum is then peeled off from the diaphragm (see fig. 606, *e*) until the abscess can be felt. After confirmatory aspiration, this is opened by pushing the finger or a sinus forceps through its wall, evacuated, and drained with a large rubber tube.

If, during the performance of an extra-peritoneal operation, it is found that the abscess is already adherent to the parietal peritoneum, and that the peritoneal cavity is completely obliterated over the abscess, this can be opened directly by pushing a finger or forceps through the adherent abscess wall.

The only occasion on which an extra-peritoneal approach is contra-indicated is the neglected case with a complicating empyema. Here it is probable that the diaphragm is already perforated, and the procedure is governed by the necessity to drain the empyema. Therefore the transpleural route is inevitable. After confirming the empyema by aspiration, large portions of at least two of the lower ribs are removed in the postero-lateral region, the phrenico-costal pleura deliberately opened, and the empyema evacuated. A finger searches for a perforation in the diaphragm, which is enlarged, and the sub-phrenic abscess is emptied. It will be necessary to drain the empyema cavity, as well as the abscess cavity below the diaphragm.

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SECTION 13

ILEUS

by  
the late

H. TYRRELL-GRAY

## SECTION 13

### ILEUS

THE origin of the term "ileus" is obscure; but, nevertheless, for decades it has presented to every clinician a picture regarded with dread and anxiety. The picture is one which conjures up those doubts as to the wisdom of past actions and decisions (as well as that uncertainty as to future action) which are prominent among those burdens of our profession which are all too little appreciated either by patients or the public at large. It is questionable to the writer how far the estimate of a possible post-operative ileus is considered by those who are responsible for advising for or against operation.

In the past the atmosphere of mystery or the lack of a physiological approach to the subject has formed a basis for much speculation, as well as for empirical and largely experimental treatment. Further, the loose application of the term "ileus" to all forms of intestinal distension has, in the writer's view, militated against a clear conception of the pathology, which alone can guide us in its prevention and cure. At the outset it may be stated categorically that there are *two clear-cut types of intestinal distension*, the pathology of which is *totally different*, and the clinical management therefore proportionately diverse.

These two types are :

A. *Intestinal Obstruction.* This term is strictly to be limited to those agencies which mechanically impede or prevent the passage of contents along the intestinal lumen, the mechanism of intestinal movement being unimpaired.

The result of such impediment is to throw additional work on to the intestinal muscle, which responds to the demand by "hypertrophy and over-action" (1).

Such a condition is evidenced clinically by :

- (1) *Inspection*, for visible peristalsis is evident.
- (2) *Palpation*. The hardening of distended and hypertrophied coils during peristalsis can often be felt on examination.
- (3) *Auscultation*. The periodic intestinal movements can further be appreciated by the routine use of the stethoscope on the abdomen.
- (4) The initial absence of those toxic signs, so characteristic of true ileus (to which attention will be directed later). Such a picture may, according to the rapidity of onset, be :
  - (a) *Acute*. Of this the impacted gall-stone is a classical example.
  - (b) *Chronic*. This is best illustrated by the terminal symptoms of the "ring carcinoma" of the large and (rarely) small intestine.

B. *Primary ileus*. In this class distension follows upon the *inability of the intestinal muscle to contract*. Such distension is obviously in complete contrast to that of the over-action and hypertrophy which result from a mechanical obstruction to the intestinal lumen, an obstruction which only imposes a greater burden and evokes a more willing response up to a certain point.

On an accurate appreciation of such a distinction depends the true conception of this condition. The clinician must next ask himself this question: What are the factors which can prevent intestinal muscle from contracting in its normal manner, since the greater the obstruction the greater the effort to overcome it? No less an authority than Barnard (2) once wrote: "Ileus is an ancient term incapable of precise definition, and therefore should not be used." The present writer, however, has defined ileus as "*distension from intestinal inertia*" (3), a definition which presents the picture of a *motionless intestine incapable of muscular action*—in marked contrast to the picture presented in chronic obstruction.

Having defined ileus we may now pass on to a consideration of those factors which can prevent the normal contraction of intestinal musculature, and so contribute to "intestinal inertia."

A brief review of the normal mechanism of gastro-intestinal activity is, however, an essential preliminary to a study of those factors.

The gradual change from voluntary control to involuntary action

of the muscular mechanism of the alimentary tract (*pari passu* with the normal gradual abolition of sensation) is signalled by the entry of a dual nerve control (4, 5) antagonistic in action, the one exciting, the other inhibiting both muscular activity and secretion. This is termed the autonomic system.

Clinical and radiological evidence, and pathological considerations, all support the suggestion that the fundus of the stomach is to be regarded as a dilated pouch of the lower end of the *œsophagus*

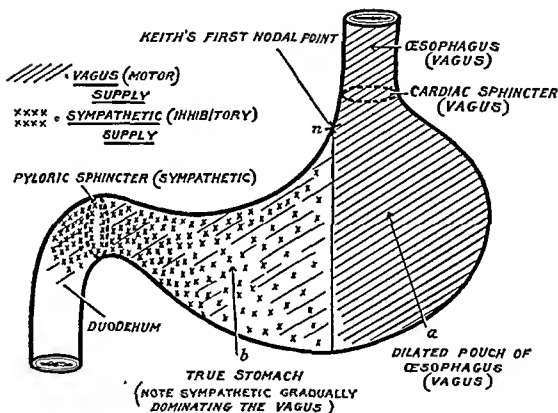


DIAGRAM OF SUGGESTED NERVE-SUPPLY OF THE HUMAN STOMACH.

Fig. 612.

(fig. 612) (5). The presence of rhythmic waves, continued from the *œsophagus*, the absence (under normal conditions) of true peristaltic waves, the continuation of the *longitudinal* fibres of the *œsophagus* into the *oblique* fibres of the fundus: all these considerations, together with the relative immunity of the fundus of the stomach from the gross primary disease—i.e. “peptic ulcer, primary carcinoma, etc.”—are sufficiently striking features to justify the view that the fundus of the stomach is a dilated pouch of the *œsophagus*, controlled by one nerve supply, the vagus (a, fig. 612), which begins at the vertical line (n, fig.

612), being the site of the first nodal point (in Auerbach's plexus) Keith (6).

It is at this point that irritation (whether by the pathologic stimulus of an ulcer, etc., or by experimental mechanical stimulation) is most prominently associated with spasmodic contraction in the opposite border of the viscus. The "spasmodic hour-glass" stomach so produced, becomes less marked (pathologically and experimentally) as the lesions occur more distally. Evident in the duodenum, and to a lesser degree in the jejunum, such a feature is entirely absent in inflammatory lesions of the small intestine (e.g. tuberculous and typhoid ulceration, etc.).

The same is true of operative manipulation, where inhibition is the rule. Experience, it may be recalled, has shown that, in the ordinary forms of stimulation of vagus and sympathetic together, the sympathetic always dominates the vagus (7). The gradual entry and ultimate domination of the inhibitory element of the dual nerve supply account for the gradual disappearances of the "nitch and notch" phenomenon with the more distal situation of the lesion.

It is probable, therefore, that at *n* (fig. 612) the voluntary system gives way finally and completely to the commencement of the true autonomic system with its dual antagonistic nerve control.

Here it is that the *true peristaltic wave* is first seen under normal conditions. Under X-ray examination a wave consisting (in contrast to the "rhythmic wave") of a contracting and synchronously inhibited segment travels as a combined dual entity down the intestinal tract. The mechanism of this has never been established. But, since an appreciation of the factors involved is so essential to a clinical study of the various derangements of this mechanism, the writer can only offer his own views.

In the first place it must be accepted as a postulate that the mucosa, musculature, visceral peritoneum, etc., of the intestinal wall are all entirely devoid of sensation.

All sensation in the visceral tract, within the limits of the true autonomic system, originates in the *mesentery* (10), which is the sense-organ of the viscera; and the mesentery is consequently the basis of its protective mechanism. There is, therefore, no pain (which is a *pathological* symptom) originating in the intestine proper; and the physiological "pain" of impending defecation arises both in that portion of the hind-gut where the gradual return to voluntary control is becoming evident, and from the association of this act with retro-peritoneal nerves in the region of voluntary control.

*The peristaltic wave.* This consists of two segments :

- (1) A contracting segment, and
- (2) An inhibited segment, immediately distal to it.

These two travel together synchronously down the intestinal tract. Now, Starling (8) showed many years ago that this mechanism of intestinal propulsion could be effective "in vitro," with the intestine entirely divorced from any central influence whatever. This proved that the intestine was an automatic, rhythmic, and independent organ capable of individual initiative in the performance of at least one of its duties, i.e. the propulsion of the intestinal contents in the right direction. This complex wave is probably excited locally (in contrast to the automatic rhythmic wave which passes down the intestine at regular intervals). The rhythmic wave is to be regarded as a "feeler," designed to sense any contents by pressure upon them, this pressure exciting the peristaltic wave locally (4, 5).

It is clear that such an independent mechanism must be normally under the control of central influences, in order that it may be adapted to the varying needs of the whole body from time to time.

It will be simpler to consider first the local mechanism, and subsequently those agencies through which this mechanism can be impeded or deranged.

Four different structures are concerned, namely :

- (1) Outer longitudinal muscular layer.
- (2) Auerbach's plexus.
- (3) Circular muscle layer.
- (4) Meissner's plexus (fig. 613).

The two important structures, then, to be considered are Auerbach's and Meissner's plexuses.

Though unstriated muscle has the inherent power of responding to distension by contracting, these two nervous elements—the one (Auerbach) lying between the two muscular coats, the other (Meissner) in the submucous layer—play a most important part.

The writer conceives the local mechanism of the peristaltic wave as follows (the reader is referred to figure 613) :

The regularly recurring rhythmic wave, on its passage down the intestine, senses, by pressure upon them, the intestinal contents at (1),



when the reverse pressure upon Auerbach's plexus (B) provides the stimulus to these cells (6).<sup>1</sup>

From these proceed (lined arrows) impulses to the two muscle coats stimulating them to contraction at (2). This constitutes the *contracting segment of the true peristaltic wave*.

While, however, the pressure at (1) so stimulates Auerbach's (or the motor) plexus, the same stimulus is clearly applied to Meissner's

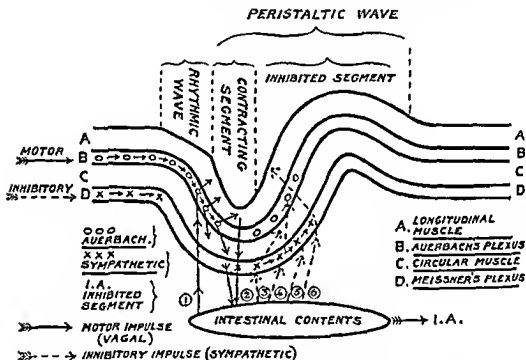


Fig 613.—DIAGRAM ILLUSTRATING THE MECHANISM OF THE LOCAL EXCITATION OF THE PERISTALTIC WAVE, BY THE RECURRING RHYTHMIC WAVE.

submucous plexus at (1). (For clarity this is represented as being most marked at (2) in the diagram.)

From Meissner's plexus proceed impulses (dotted arrows) along fibres directed obliquely downwards to the muscle coats immediately distal (3, 4, 5, 6, fig. 613) which inhibit all tone in the affected zone. This constitutes the *inhibited segment of the peristaltic wave*.

The effect of this combined mechanism is a powerful contraction of the circular muscle upon the intestinal contents (2, fig. 613), together with a drawing back of the gut proximal to these by the synchronous contraction of the longitudinal muscle. This forces the contents into the inhibited area (I, A), i.e. the line of least resistance.

<sup>1</sup> Keith has shown that mechanical stimulation in certain situations can induce contraction.

The whole arrangement would appear to be meticulously designed to ensure the passage of the intestinal contents in the desired direction—down the intestinal tract (5).

It is not difficult to envisage such a “dual” wave, excited similarly and successively by local stimulation, passing down the intestinal tract as one entity.

Figure 614 represents diagrammatically a peristaltic wave as seen by the writer many years ago during an operation on an infant under

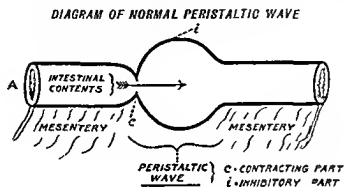


Fig. 614.

spinal anaesthesia. This contraction was, in fact, followed by a momentary intussusception, and was in marked contrast to the rhythmic wave so often seen during operations under this form of anaesthesia. The picture presented formed the basis of the writer's present conception of the mechanism.

#### DERANGEMENT OF THE NORMAL MECHANISM

It is clear that there are many ways in which such a delicate mechanism may be impaired, either temporarily or permanently, and the effects, symptoms and signs should, if our knowledge were perfect, enable us to detect clinically the actual process involved in any given instance. However, though our knowledge is imperfect, much may be learned from the scientific application of clinical experience, particularly in the study of “ileus.”

(1) *Intra-mural lesions (a).* There is no necessity to labour the well-known truth that no muscle—and certainly no neuro-muscular tissue—is capable of performing its function adequately if invaded by inflammatory or other disease.

A hypothetical case will best illustrate the theme if the reader will turn once again to figure 614 and conceive some intra-mural defect,

however small, which must derange the delicate mechanism just referred to (whether this can be detected clinically or not). Such a section of intestinal wall, completely thrown out of gear, will affect the mechanism just described as shown in figure 615. Such areas I have described as "inert areas" (4, 5, 9). At such points the gut is quite incapable of responding to stimulation of any kind (motor or inhibitory).

These are, therefore, treated as foreign bodies within the intestinal lumen and forced down the intestinal tract. (cf. fig. 614 with fig. 615.) The mesentery being the "sense organ" of the intestine (4, 5, 9), and in this instance the "anchorage" of the intestine, tension upon it results (fig. 615), and leads to pain (10, 12). Should the mesentery be firm and contain a due proportion of fat, etc.—as obtains in adult life—*pain, coincident with the peristaltic wave alone results, without any*

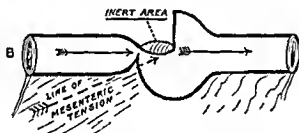


FIG. 615.—DIAGRAM ILLUSTRATING THE DERANGEMENT OF THE NORMAL PERISTALTIC WAVE BY AN "INERT AREA" IN THE INTESTINAL NEURO-MUSCULATURE—LEADING TO INTESTINAL COLIC, INTUSSUSCEPTION, ETC.

*other obvious clinical interference with intestinal function.* Should the mesentery be thin, elastic, and extensible (as in infancy), intussusception may result. *This is the mechanism of intussusception; and intestinal colic is only attempted intussusception.*

The point to be emphasised is that such lesions cause *colicky pains with no other symptoms whatever*; and these are frequently overlooked in consequence. Examples are:—acute and chronic rheumatism, acute tonsillitis, chronic tonsillar infections, severe urticaria, scarlet fever, etc., etc. (4, 5, 9, 11).

In exaggerated forms such lesions are well recognised. "Henoch's purpura," for instance, is a clinical entity *only because the lesion can be felt*, and its size entails a rupture of the mucosa, so that *hæmorrhage from the bowel can be observed*. It is curious that in a smaller degree, unrecognisable by clinical examination, the existence of such lesions as a clinical entity is still generally unrecognised by the profession. The result is often the removal on chance of many innocent appendices!

*Intra-mural lesions (b).* If now such lesions be envisaged as involving the whole circumference of the bowel (e.g. fibrous stricture, malignant stricture, inflammatory infiltration from perforating diverticulitis, etc.), the symptoms are but exaggerated and progressive. With the relative immobility of the "*inert area*" the main weight of the peristaltic wave is exerted upon the redundant mucosa proximal to the lesion. Venous congestion, œdema, and increased mobility and detachment result, so that the lumen of the gut at the site of the lesion is gradually narrowed. The bowel responds to the *increased distension and work by muscular hypertrophy and increased effort* (1). Venous congestion is correspondingly increased, and the abdominal muscles gradually relax to prevent an increase in the normal intra-abdominal pressure. The whole represents a partial compensatory process to suit altered conditions.

This, however, presents a typical picture of *chronic obstruction* as indicated in A at the commencement of this chapter (see page 1181).

It is characterised by those *four infallible clinical signs* which label it as an *active process from the start*. The final picture in all such cases is that the vigorous efforts of the intestine finally succeed in driving the swollen and hyperæmic mucosa (with or without rupture, i.e. hæmorrhage) into the narrowed lumen, and the obstruction becomes complete. Continued over-action only exaggerates the picture; compensation in every direction continues (and toxic symptoms are absent—this will be referred to later), until finally the whole mechanism breaks down and the intestine passes gradually into complete "*inertia*," or "*ileus*." This is a "*secondary ileus*," that is to say a complete "*inertia*" from so-called "*paralysis*" of the intestinal musculature.

This condition is much the same as "*primary ileus*," which is our next consideration. But it is clear that *it is a late sequel to an unrelieved obstruction, which never ought to occur clinically*.

(2) "*Acute Ileus*." Up to the present we have only considered the gastro-intestinal tract as a *local mechanism*. We have now to consider those outside agencies which normally guide and control its activities to suit the continually varying needs of the body at any given moment.

The immense importance of the mesentery (and the need for it to receive every possible protection from injurious agencies) is evidenced by a consideration of those structures which reach the intestine within its protective envelope (12).

(a) *Innervation.* (i) The *motor nerve supply* is the parasympathetic, which activates the plexus of Auerbach, and is represented by the vagus and pelvic visceral nerves—the vagus functioning (it is believed) to a point somewhere short of the splenic flexure, the duties therefrom being assumed by its companion. *It also stimulates secretion.*

(ii) *The inhibitory nerve supply* is vested entirely in the sympathetic system which activates the plexus of Meissner. *It is also inhibitory to secretion.*

The normal activities of these are balanced to control intestinal activity. Thus—though the intestine may be compared to a horse, which is capable of doing all the work independently—the central nervous and vascular systems should be regarded as the guiding of the human brain and hand; and the nerves and blood-vessels, etc., as the reins which are the medium of control (7).

As an example of the motor side may be mentioned the onset of gastro-intestinal activity, as the result of hunger, or the sight or smell of food. Hunger is initiated by the degree of engorgement of the digestive glands (gastro-intestinal, pancreatic, etc.). From these proceed efferent vagal impulses evoking the motor and secretory response (5, 9). The secretions are ready, as are the motor activities, for the active process of digestion and transit. The distressing audibility of this "signal" in some unfortunates should never be accepted by a hostess as a rebuke for the lateness of a meal! This is but the mechanism restarting the activity of the rhythmic waves throughout the gastro-intestinal tract. On the other side of the picture let us consider a little more fully an example of the inhibitory function of central origin, and its simultaneous widespread distribution over the whole body. The reader is reminded of the statement earlier in this chapter, namely, that under normal conditions the ordinary stimuli to the sympathetic and parasympathetic together are always associated with the domination of the sympathetic response.

While inhibiting gastro-intestinal activity, the sympathetic system also stimulates the arteries to vasoconstriction, and, by raising the blood-pressure and improving the work of the heart, increases the blood supply of the cerebral and skeletal tissues. Accordingly we see an elaborate mechanism for ensuring that, during the ordinary daily activities of brain and muscle, the digestive processes are relatively in abeyance; while during the period of greatest rest (i.e. sleep) all the digestive processes attain the zenith of their activity unhampered by inhibitory stimuli.

It must be obvious that any derangements of balance in the action of these two, of central origin, may (indeed do) have far-reaching effects. These form the basis of some of the most difficult problems in medicine and surgery, i.e. the decision as to the primary origin of the disturbance under consideration. This aspect will be subsequently referred to.

(b) *Blood supply*—Arterial and Venous. The variations in intestinal activity in relation to the alteration in the arterial supply have been briefly touched upon. But it has always been a matter of astonishment to the writer how little emphasis is laid upon the one essential to the normal function of gastro-intestinal musculature, namely, the integrity of the venous returns. Well recognised in voluntary muscle, venous stasis is never sufficiently stressed as a possible cause (even in

its incipient stages) of a slow and progressive intestinal failure of central origin (whether primarily cardiac or pulmonary), which may have far-reaching effects of the utmost significance to the clinician (3, 12).

(c) *Lymphatic supply.* The importance of the integrity of this hardly needs more than mention.

*Is it any wonder that a structure such as the mesentery* (responsible for the safe transit of all the structures *essential* to the nutrition and life of the animal) should be provided with organs of sensation, powerful anti-bacterial defence, and protection against mechanical and other injury by adequate reflexes—voluntary and involuntary (2)? Further, is it surprising that the mesentery, provided with such elaborate means of protection (12), should itself be constituted the main protective agency or guardian of the integrity of the intestines?

In passing on now to a practical study of *Intestinal Inertia* ("ileus") it has to be remembered that there are four possible causes:

- (1) *Intestinal obstruction.* This has been shown never to cause primary ileus.
- (2) The effect of bacterial toxins on the intestinal wall. The observations of Nothnagel (13) would appear definitely to exclude this as a cause, since he (and subsequently, no doubt, many others) observed active intestinal movements after death from "ileus."

This leaves us with two types of "Intestinal Inertia."

- (3) "*Active Ileus.*" This is *physiological* and *protective* in nature.
- (4) "*Paralytic Ileus.*" This is *pathological* and one of the most dreaded complications of abdominal surgery.

"*Active Ileus.*" In its simplest form this is seen every time the abdomen is opened under general anæsthesia, when (with the exception of the fundus and proximal stomach, and a few spasmodic contractions of the antrum) a completely motionless intestine is the rule.

[*This dramatic cessation of all movements on the "rude invasion of the surgeon" has been picturesquely compared by Meltzer with the sudden hush which descends upon a roomful of chattering friends on the sudden entry of a complete stranger.*]

The stimulation of the somatic afferent fibres by the incision of the abdominal wall, the impulse from which is continued down the *efferent*

*sympathetic* (as a local reflex), inhibits all movement and induces simultaneous vasoconstriction in the area involved. This is a protective measure against hæmorrhage and possible damage to the intestine itself.

[The reverse reflex, from the mesentery—clinically again a protective measure—should be borne in mind for future reference.]

Spinal anæsthesia (or experimental section of the cord) abolishes this reflex and allows free and unchecked intestinal movements.

The simplest clinical type of active "protective ileus" is best

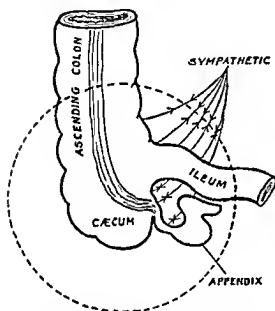


Fig. 616.—DIAGRAM OF IRRITATED AREA IN "ACUTE (2-5 O'CLOCK) APPENDICITIS." BOTH AFFERENT AND EFFERENT IMPULSES ARE INDICATED BY ARROWHEADS.

illustrated by the concrete example of acute inflammation of the appendix in the 2-5 o'clock position, i.e. adjacent to the ileal mesentery. Such an inflammatory lesion stimulates the lower ileal mesentery by hyperemia, cedema, etc. (as can be done experimentally by trauma) (10); and the resultant sympathetic stimulation, following the rule of sympathetic domination (7), has a twofold result:

(1) *Efferent sympathetic impulses*, proceeding from the area of inflammation and local hyperæmia, inhibit and render motionless the area of intestine within the dotted area (fig. 616).

(2) *Afferent sympathetic*. The appendix being itself insensitive, the initial discomfort or pain will be referred from the stimulus to the mesentery. Discomfort primarily, followed by pain, is therefore referred in the first instance to the solar plexus, i.e. it is *epigastric*.

Subsequent symptoms due to afferent stimulation follow in a succession, the rapidity of which varies with the severity of the lesion.

Where the sympathetic system has not yet entered into its normal antagonism with the parasympathetic, the synchronous afferent vagus stimulus will evoke an unchecked vagus response. The reader

is referred to figure 612. Accordingly, though the *efferent sympathetic response closes the pylorus (14)*, the fundus, unhindered by opposition, is able to contract.

With the extension of the inflammatory process the stimulation of the Paccinian corpuscles in the mesentery results in a local somatic response which immobilises the abdominal wall over the segment involved.

*[This is but a pathological mimicry of the normal reflex which takes place on the assumption of the upright position in the normal human being. When the upright position is suddenly assumed from recumbency, the viscera fall by gravity until they are supported by the mesentery. The mesentery contains far too many vital structures for it to be submitted to any mechanical duties ; accordingly it is provided with the Paccinian (" touch ") corpuscles, from which travel impulses to the somatic abdominal nerves. As a result the abdominal wall contracts and affords the necessary support, i.e. if the reflex and muscles are intact.]*

Let us now correlate the clinical symptoms with the mechanism just described :

(a) The mechanism of inhibition of all movement in the dotted area (fig. 616) (together with a certain degree of resultant immobility distal to it) is to be regarded as a protective mechanism designed to provide "*rest to the inflamed part.*"

(b) The initial afferent sympathetic stimulus from the mesentery is responsible for the initial epigastric discomfort or pain ; this is soon masked by the *local symptoms*. These afferent stimuli lead to widespread efferent sympathetic response, proportionate to the extent and intensity of the stimulus.

(c) *Efferent sympathetic response.* Relative immobility of the whole intestinal tract still further prevents the spread of the infective focus.

*Constipation* of varying degree is the result of (a), (b), and (c).

(d) The reflex contraction of the fundus of the stomach against a closed pylorus leads to *vomiting*.

(e) With the progress of the inflammatory process, hyperæmia, œdema, etc., the increased local stimulus causes the *local pain* to dominate all other discomforts. Pain is subsequently, therefore, referred to the right iliac fossa. Similarly, the well-known "*guarding,*" by the reflex contraction of the abdominal muscles in the affected area, is another aid to complete immobility.



This, the commonest type of intra-abdominal inflammation, is taken as an illustration of the most elaborate defensive mechanism, designed to provide *rest to the inflamed part*, by the rejection of food and the immobility of the affected parts.

The writer has termed this form of intestinal immobility and distension "active ileus," because it is a normal physiological response to injury. *It is to be encouraged and fostered clinically by every known means.*

These principles form the essential basis of the "Fowler treatment" of acute intra-abdominal inflammation.

"*Paralytic Ileus.*" In order to understand the transition from intestinal obstruction, or the active ileus just described, to true "paralytic ileus," it is essential first to remember the physical law: *that increase of pressure within any bent tube (of whatever material) tends to straighten the tube proportionately.*

Now, the gastro-intestinal tract is composed of a series of bent tubes, the straightening out of which from over-distension is prevented (in nearly all its extent) by the anchorage of a mesentery infinitely shorter than the viscus itself.

*The first effects of any mesenteric tension are felt by the most collapsible structures, i.e. the veins.* This is shown in figure 617.

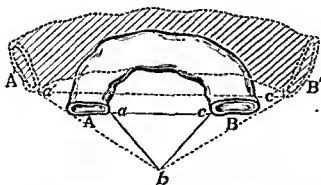


Fig. 617.—ILLUSTRATES DIAGRAMMATICALLY THE TENSION ON THE MESENTERY WHICH RESULTS FROM INTESTINAL DISTENSION BOTH IN THE VERTICAL AND HORIZONTAL DIRECTIONS. THUS, WHEN A B BECOMES A' B', THE ANCHORAGE AT ab, bc TENDS TO BECOME a'b, b'c VERTICALLY; AND ac TENDS TO BECOME a'c' HORIZONTALLY.

The relative incapacity of any muscle to contract in the presence of venous stasis is only proportionate to the degree of circulatory obstruction, and this is clearly proportionate to the degree of distension and resulting tension on the mesentery.

*Mesenteric tension* has been recognised as a factor of importance by Keith (15), Nothnagel (13), and Barnard (16) amongst others, while the latter particularly emphasised the importance of the circulatory factor.

The acutest and most dramatic example of "paralytic ileus" is "*acute intestinal strangulation*" (as opposed to obstruction, already discussed). This is, at the outset, the clinical analogy of the experimental ligation of the intestinal veins with the arteries intact.

The clinical results are :

- (1) Severe stimulation of the Paccinian corpuscles (and other sensory nerve-endings), leading to shock (10), vomiting, and initial immobility of the abdominal wall.
- (2) As soon as the pressure in the obstructed vein exceeds that in the intestinal lumen, gases will pass, by osmotic pressure, into the intestine, the intestinal musculature being paralysed by venous stasis.
- (3) In due course, however, a time arrives when this pressure within the intestinal lumen again exceeds that in the adjacent veins. Intestinal gases then pass in the reverse direction, i.e. *from the intestinal lumen into the blood stream*. These gases are then rapidly exhaled by the lungs, and give that characteristic odour to the breath which is *one of the most reliable diagnostic signs of intestinal venous stasis*. Whether the process is acute or chronic is only a question of degree.

"Intestinal breath," as a symptom of intra-abdominal inflammation or strangulation, is stressed too little. For example, it rapidly appears even in early acute appendicitis, when the adjacent coils of gut are motionless and distended, and will often help to differentiate intra-abdominal crises from disease of pulmonary or other origin.

- (4) As venous distension progressively extends upwards and downwards fluids pass from the veins :
  - (a) Into the intestinal musculature, causing œdema.
  - (b) Into the peritoneum causing ascites.
  - (c) Into the intestinal lumen, accentuating the distension and those highly infected fluid contents of the intestine which are so characteristic.
  - (d) Into the ileo-colic sphincter.

Under normal conditions the large bowel is anatomically adapted to repeated attacks of intermittent obstruction arising from the voluntary control of defæcation. This is an adaptation not shared by the small intestine, which is accordingly protected by the ileo-colic sphincter.

*[That this is true is shown by those profound changes in the small intestine following colectomy—changes which I have demonstrated to be absent when the ileo-colic sphincter is preserved and transplanted into the large intestine (3).]*

Elliott (17) showed that sympathetic stimulation inhibited the intestines, but closed the ileo-colic sphincter.

In active ileus, therefore, the ileo-colic sphincter is closed—again an obvious protective measure.

In passive or paralytic ileus, the venous congestion renders the sphincter inactive and functionless. Contents of the large bowel now have free access to the small, and the highly increased infectivity of the latter is, at any rate partly, accounted for.

(c) Into the pyloric sphincter. Similarly, with the progressive upward spread of the venous stasis, the pyloric sphincter becomes incompetent, and allows of the regurgitation of highly odorous and infective fluid contents from the intestine into the stomach. Regurgitant stercoral vomiting results. This vomiting never really rids the stomach of its contents, because its contractions also are impaired by venous stasis. The result is gradual overloading, overstretching, and ultimately a picture analogous to that of acute dilatation of the stomach.

(f) Into the abdominal wall. Increase in intra-abdominal pressure, by further compression of the larger veins, again tends to aggravate intestinal venous stasis, and, since there is less elasticity in those with a fat mesentery, “paralytic ileus” is more apt to develop in this type of subject than in thin people with lax abdominal walls.

The writer has seen alarming degrees of distension follow Cæsarean section quite unassociated with any marked discomfort to the patient.

## CLINICAL CONSIDERATIONS

The exciting factor in the onset of ileus, whether *active* or *paralytic*, may be :

(1) Extra-abdominal, or (2) Intra-abdominal.

(1) *Extra-abdominal causes.* The following are examples :

(a) *Active ileus.*

(i) The psychic factor has long been recognised as playing an important part in intestinal derangement, for excessive or prolonged generalised stimulation of the sympathetic system inhibits both motor and secretory activity. *In the acute form* the cessation of intestinal movement has been noted by Cannon (18), Langdon Brown (20), Leonard Hill (19), and many others.

In milder form gastro-intestinal derangements arise from prolonged mental strain, fear, apprehension, etc., and, if long continued, may cause sufficient permanent sequelæ to make the recognition of the primary origin of the disorder a matter of extreme clinical difficulty.

(ii) The renal factor. In some acute renal crises the intense sympathetic stimulus gives rise to widespread intestinal inhibition and distension. This is *not* a paralytic ileus—there is no primary venous congestion—there is, therefore, no “*intestinal breath*.”

[The following is one example. Some years ago the writer operated on a man of 51 years with a history of progressive constipation culminating in an acute attack of abdominal pain, absolute constipation, and repeated vomiting. The distension was enormous, but no movement could be seen in the distended coils. No gross lesion could be discovered in the alimentary tract, but a hæmorrhage into a left hypernephroma was discovered. Cæcostomy was performed, and the acute symptoms subsided. A nephrectomy was subsequently performed.]

(b) *Passive ileus.*

(i) The pulmonary factor. Extreme abdominal distension in pneumonia, empyema, etc., is a common clinical experience. Mannaberg (21) believed it to be due to peritonitis, but it is almost certainly circulatory in origin.

[An extreme instance is that of an infant of three months, ill for two days with a history of sudden abdominal distension of a very marked degree, great distress, and respiratory embarrassment. This was thought to be due to pressure upon the diaphragm. Careful examination failed to discover any pulmonary disease, and the abdomen was explored. Extreme distension of all the intestines without discoverable cause led to immediate closure of the abdomen. At the post-mortem examination an acute empyema was found on both sides.]

- (ii) The cardiac factor. Heart failure has long been recognised as a cause of intestinal distension from venous stasis.

In the last two types the breath does not exhibit that characteristic intestinal odour already referred to, for two reasons :

1. It is either terminal, and heralds death, or
2. It is a slow process which, though progressive, is accompanied by a compensatory adaptation of local conditions.

(2) *Intra-abdominal causes :*

*Post-operative ileus.* This, with its attendant anxieties, most closely concerns the surgeon in its acute form.

A. *Active (inhibitory) Ileus.* This is to be regarded as a protective mechanism designed to provide rest to the damaged or inflamed parts. It should therefore be promoted by every means possible.

*Starvation.* This ensures the minimum of work for the intestinal tract and promotes rest. There are some who carry this principle to an extreme. Such a course entails the misery of thirst and restlessness, which in itself violates the principle of bodily and mental rest. On the other hand, the free administration of large quantities of fluids sometimes leads to flatulence, discomfort, and vomiting. Common sense and moderation will always indicate, in any given instance, to what extent the principle of starvation is to be applied.

A medicine glass of any fruit juice, placed by the bedside and sipped at will, supplies glucose, fluid, and cleans the mouth. Very often the patient will prefer to clean the mouth by chewing some fruit and spitting out the solids.

*Rectal Saline* (with 5 per cent glucose) is often more easily retained and less irritating than water, though perhaps it may be more slowly absorbed. Given at four-hourly intervals this is sometimes less disturbing

and more effective than continuous proctoclysis. By this means fluid and carbohydrate deficiency are easily compensated, and the elimination of toxic products (anæsthetic, etc.) is promoted.

*Fowler position.* There are two important reasons for adopting this position :

- (i) Relaxation of the abdominal wall promotes comfort, and tends to reduce any tendency towards compression of the veins.

[This, as well as the frequent discomfort and distress resulting, is one reason why tight bandaging is to be deplored.]

- (ii) To promote the gravitation of infected peritoneal fluids to known and less dangerous regions (Box, 22).

*Sedatives.* Local and general rest are provided by *sleep*, during which time the elimination of poisons (i.e. anæsthetics, etc.) and waste products can be proceeded with, unhampered by the addition of the superadded products of muscular action, mental activity, etc., entailed by movements, anxiety, apprehension, pain, etc. The writer has demonstrated (jointly, 10) that the effect of strong emotions (fear, apprehension, extreme anxiety, etc.) upon the pulse and blood-pressure reproduces a chart so closely resembling that seen in the initial stages of shock, that for these reasons, for bodily comfort and rest, for mental peace (all of which are important items in the picture of rest), *the liberal administration of sedatives is part of the essential routine.*

Accordingly, the writer's practice is to administer the first saline-glucose per rectum immediately the patient is back in bed, and to add aspirin 30 grs. with pot. brom. 60 grs. This is repeated eight-hourly for three doses. In the meantime injections of morphia, heroin, or omnopon are given as and when required. *These should never be stinted.*

*Radiant Heat.* If the onset of "paralytic ileus" is characterised by venous stasis it is clear that maintenance of the integrity of the blood supply from the start is advisable, and that, immediately the inhibitory stimulus of trauma or infection has passed off, intestinal movements will begin to be heard and felt by the patient, and will be followed by the passage of flatus.

The routine application of a radiant heat cradle to the abdomen for twenty minutes two or three times a day has formed an integral part of the writer's routine for many years.

The withdrawal of blood to the surface tends to militate against deep engorgement, is a preventive measure against paralytic ileus, and is also a source of great comfort to the patient with very few exceptions.

*Purgation and Feeding.* Nearly everyone has his own routine as to time and method, and the variations are very wide, since they are based on empiricism. Actually there is *no one time in any given instance at which to start purgation*. As soon as the *protective (active) ileus* starts to pass off, *intestinal movements will be heard with a stethoscope* (the routine use of which is to be strongly advocated).

*This is the signal* to encourage the bowels to act by any routine favoured by the individual in charge. The writer gives inj. pituitrin 1 cc. followed by a turpentine wash-out. Then a laxative or purgative follows.

The first satisfactory action of the bowels is the first indication for a start to be made towards a gradual return to a normal diet—this, of course, always provided that the temperature, pulse, and local conditions exclude any residual local inflammation.

The return of intestinal movements (coinciding with the cessation of the inhibitory stimuli) must obviously vary in each individual case with the extent of the operative trauma, the degree of active infection persisting afterwards, and the irritation of drainage-tubes, etc., etc. The only safe rule which can guide us clinically is the estimation of the cessation of protective mesenteric irritation as evidenced by even the slightest return of intestinal movements.

*Before this is evident purgation is useless, harmful, and on occasions disastrous*, as it may be contributory to the transition from *active ileus* to *paralytic ileus*.

**B. Paralytic Ileus.** It must be clear to every reader that, if pressure upon the veins, together with any cause (intra- or extra-abdominal) which may accentuate venous obstruction, is the first step towards the commencement of a "paralytic ileus," there may at times be a *very narrow margin* dividing the transition from active to early paralytic ileus. Thus cardiac or pulmonary complications may easily be the deciding factor in a case which would otherwise be straightforward. Tight bandaging has already been mentioned as bad, for it allows of no compensatory relaxation of the abdominal wall, as well as being liable to embarrass the expansion of the lower lobes of the lungs.

The measures for prevention are largely the measures for cure. In

addition to the steps already outlined, the following should be watched and treated :

- (a) The persistence of a dirty tongue, with no evidence of cleaning at the tip or edges, should call for extra vigilance.
- (b) Gradual rise of pulse-rate, or failure to fall from a high level.
- (c) Absence of a corresponding rise of temperature at first.
- (d) Distension, marked with a soft well-moving abdomen.
- (e) Persistent flatulence or hiccough.
- (f) Visible coils without visible peristalsis.
- (g) Absence of pain, particularly colicky pain.
- (h) Absence of true movements of the intestine (together with the presence of "tinkling" sounds, as evidenced by the stethoscope).
- (i) The odour of the breath.
- (j) The partial response, by flatus only, after two or three days to pituitrin and turpentine, this response only partially relieving the distension which rapidly returns.
- (k) Perhaps one vomit with relief, followed by a return of flatulence later.

Such symptoms call for a steady persistence in the measures already outlined, the administration of radiant heat and sedatives, and, at intervals, the partial relief afforded by inj. pituitrin and enema turbinth.

Cardiac and pulmonary integrity also must be most carefully guarded.

*Progressive "Paralytic Ileus."* Should the condition become steadily progressive in spite of these measures, or should vomiting commence, more active steps must be taken.

*Gastric lavage.* This may be carried out either by lavage at intervals, or by continuous syphonage with a Ryle tube. The emptying of the stomach must considerably reduce intra-abdominal pressure, and may restart the gradual spread of a more normal circulation from above downwards leading to partial intestinal movement.

*Purgation.* The resort to purgatives is a *fatal mistake*. As an irritant causes more fluid to be poured into the intestine, hyperæmia is increased and pressure is still further raised. *Purgation is always harmful until there is evidence of intestinal activity.*



*Injections and Enemata.*

- (a) *Pituitrin* has been referred to, and is usually effective. In occasional cases it fails, or, having proved efficient, loses its value by repeated use.
- (b) *Acetyl-choline*. This has been advocated strongly in recent times by several writers, notably by Abel (23) in this country.

When pituitrin fails to act on intestinal muscle, it is always conceivable that acetyl-choline may succeed in promoting a true peristaltic wave by activating the sluggish Auerbach's plexus. Personally, the writer has been very disappointed with its results in advanced cases of true paralytic ileus, but it is always worth a trial, should pituitrin fail, in earlier cases.

*Enterostomy.* In those rare cases when all these measures have failed, the advisability of jejunostomy or high ileostomy must be faced. *This should never be left until the patient's condition is really serious*—it is much better to do it early (even should it possibly have been superfluous) than to leave it late, for the full benefit is usually not seen until between twenty-four to forty-eight hours.

By the Witzel valvular method, when a large catheter is inserted, the sinus usually closes automatically after four to five days, and practically never gives any subsequent trouble.

*Subsequent progress.* The initial profuse discharge of stercoral fluid from the catheter directly it is inserted soon dwindles with the collapse of the adjacent coils, until it finally ceases in many instances. Nothing could be more harmful or useless than to "milk" the intestine, as is often recommended; and it denotes a failure to comprehend the mode of recovery from ileus.

Often there will be no further discharge for twenty-four to thirty-six hours, and distension persists—a condition which often gives rise to needless alarm. *If the catheter is ascertained to be patent and the intestine is still motionless*, the same treatment by radiant heat, fluids, and sedatives should be persisted in, and *purgatives should be avoided at all costs*. The latter the writer has seen lead to a recurrence (mistaken for obstruction) which only yielded to morphia and radiant heat.

The object of the ileostomy (or jejunostomy) is the release of tension in distended coils high up, the restoration of the circulation in these, and the re-establishment of movements. It will be evident that this object will be attained almost at once in the neighbourhood of the

enterostomy, and evacuation commences during the completion of the operation. Thereafter, however, the restoration of circulation (and the consequent return of intestinal movements) can only spread by stages to the peripheral coils from loop to loop above and below, during which period there may be no further discharge.

It is obvious that until this end is achieved, it is as harmful to give purgatives as it is before operating. A patient persistence in the same line of treatment will be amply rewarded, if operation has not been postponed until too late.

Last must be mentioned (though it is well recognised by all clinicians) the one essential feature of the treatment of any acute abdominal crisis associated with ileus, i.e. the ensurance of a steady absorption of saline solution until normal conditions are established. If, as may be the case, saline cannot be retained in the rectum, it must be administered (preferably with 2 per cent glucose) under the skin or into a vein. In either case this is better achieved by the continuous drip method. In more urgent cases recent experience has shown that the intravenous drip method is the more effective. For this special double needles can be obtained, which can be maintained in position for a considerable time.

*Ileus from peritonitis.* Whether pre-operative during expectant treatment, or whether after operation, the same principles hold good.

Dependent on the extent and severity will be the degree of inhibitory ileus and the danger of the onset of paralytic ileus.

The practical point at issue here is similar to that already discussed in the previous section. Thus, while the infective agent may be causing a widespread inhibitory action and protective ileus, a secondary paralytic ileus may be commencing or threatening.

In addition to the ordinary surgical procedure which may be indicated (or in some instances be the sole procedure possible) the question of performing the simple operation of jejunostomy or high ileostomy may occasionally demand serious consideration.

In the more severe types it may well be regarded as a "safety-valve," to tide over that period during which active ileus is providing the necessary rest to widely infected viscera, and to minimise intra-abdominal tension during this critical time.

*Ileus in acute obstruction* (as opposed to strangulation). Mention has been made of the comparative absence of classical constitutional

symptoms in acute intestinal *obstruction* in contrast to the dramatic picture seen in *strangulation*.

The explanation should now be clear. Thus the response to obstruction is hypertrophy, distension, and over-action, followed by gradual relaxation of the abdominal wall, and gradual compensatory circulatory changes in the intestine and mesentery.

The onset of distension, tendency to increased venous congestion, etc., are sufficiently gradual to meet with compensatory accommodation, both cardiac and local. Accordingly, constitutional symptoms are lacking until compensation fails, which it does rapidly when once started. Then the state of paralytic ileus from venous obstruction is initiated, and the same clinical picture (even if not so dramatic) as in those types already discussed has to be faced.

Clinically, the recognition of the motionless intestine, the state of the tongue, the odour of the breath, etc. (following the story of progressive distension, with severe *colicky pain* and increasing constipation, etc.), should indicate that compensation has broken down, and that paralytic ileus has supervened on obstruction.

Here the problem is not quite the same as in the preceding types, for, in addition to the superimposition of paralytic ileus, the treatment of obstruction from the primary gross lesion has to be considered. Such cases often present some of the hardest problems for decision. But there can be no hard and fast rules, and each case forms its own particular study. *In all cases the less that can be done to tide over the emergency (whether the ileus be treated or the obstruction) the better will be the outlook for the patient.*

Nevertheless, the decision may be a momentous one, for, in certain cases, the relief of the mechanical obstruction (e.g. by colostomy) may not relieve the ileus which has supervened or prevent a fatal ending.

*Intermittent and Chronic Ileus.* Up to the present ileus has only been considered as an acute crisis. But it must be clear that similar exciting factors, present in mild or intermittent degrees (whether progressive or not), may institute similar symptoms in more chronic and less dramatic form.

#### CHRONIC INHIBITORY IMPULSES

It is impossible here to enter fully into every aspect of this type of disease, which may be more succinctly illustrated by a specific case.

Thus, if the symptoms of acute inflammation of the "two o'clock

to five o'clock" appendix (i.e. the appendix adjacent to the ileal mesentery) be once more referred to, and if they be modified in severity to harmonise with a mild or recurrent inflammatory or mechanical irritation of the mesentery, it is not difficult to see in the latter a mild but faithful mimicry of the former—both being attributable to the same mechanism.

The following table illustrates the comparison and the reader is again referred to fig. 616 :

<i>Stimulus.</i>	<i>Result Acute.</i>	<i>Symptoms Acute.</i>	<i>Result Chronic.</i>	<i>Symptoms Chronic.</i>
Efferent sympathetic.	Local immobility.	Complete constipation.	Impeded contractions.	Incomplete action. { Constipation.
General efferent (reflex).	General impairment to complete immobility.	Complete constipation.	Impeded general actions.	Distension, variable actions, etc. { Variable actions. Discomfort and Distension.
(1) General efferent (reflex).	Pyloric closure.	(a) Vomiting reflex.	Pyloric spasm. Exaggerated fundus contractions	Increased gastric distension and discomfort. Fulness. Flatulence and belching, especially after food.
(2) Reflex vagus.	Contraction of fundus.	(b) Vomiting food.		
Sympathetic afferent to solar plexus.	Stimulation of solar plexus.	Epigastric pain.	Solar plexus irritation.	Weight and discomfort in epigastrium.
Local Paccinian corpuscles.	Stimulation of mesenteric nerve-endings.	Acute local pain and tenderness.	Mild or intermittent mesenteric irritation.	Discomfort and occasional aching in right iliac fossa.

Another example of such reflex disturbances is the flatulence and dyspepsia arising from chronic gall-bladder infections, etc. The examples might be multiplied.

#### THE CIRCULATORY FACTOR IN CHRONIC DISEASE

If it be borne in mind that the portal system is devoid of valves, it will be more fully realised that the combined efficiency of all those factors concerned in the return of venous blood from the gastrointestinal tract is of vital importance.

The effect of extreme venous stasis in producing paralysis of intestinal muscle, outpouring of fluids into the lumen, etc., has been emphasised. In more chronic general stasis, compensation may prevent complete paralysis, but other symptoms (e.g. intermittent diarrhoea, distension, hæmorrhoids, etc.) take their place until compensation fails.

The main points to be carefully noted and kept in the foreground are :

- (a) Cardiac efficiency.
- (b) The integrity of the vasomotor system.
- (c) The quality of the abdominal muscles.
- (d) The integrity of the "mesenteric-abdominal reflex."
- (e) The type and efficiency of the respiratory excursions.

(a) and (b) are obvious and require no comment.

(c) The influence of the abdominal muscles on intestinal circulation in the upright position has long been recognised.

In discussing the *flabby abdominal wall*, Leonard Hill and Barnard (24) referred to the "abdominal vessels . . . similar in nature to an easily distensible bag." Indeed, the need of adequate muscular support to valveless veins hardly requires elaboration.

(d) *The mesenteric-abdominal reflex* requires one word of explanation. Normally, on assuming the upright from the recumbent position, the viscera fall by gravity until mechanical tension on the mesentery stimulates the Pacinian corpuscles (and possibly other nerve-endings), from whose afferent impulses the efferent somatic response stimulates the abdominal muscles to contract and to :

(i) close the inguinal rings,

(ii) protect the mesentery from further strain,

and (iii) *provide the necessary support for the intra-abdominal veins.*

It is clear that abdominal muscles may, as the result of voluntary effort and training, be perfect ; but that, if this reflex be defective (as is sometimes the case), though of the greatest value for all purposes under voluntary control, *they may be of no value whatever for the purpose under discussion.*

It is for this reason that the writer deprecates as a useless waste of time and money all the multifarious, expensive, and often useless forms of exercises, training, remedial processes, etc., advocated (usually on no scientific basis) by every type of person of every grade of professional status—often no status at all. *All such exercises should be of a type*

which can be automatic and independent of voluntary effort. This will be referred to in the next section.

(e) *Respiratory Excursions.* The negative pressure in the large thoracic veins, which takes place during inspiration, is not only an important factor in the venous return from the abdomen, but is proportionate to the character and extent of the inspiratory incursion.

Imperfect inspiration (either in type or extent) means some degree of intestinal venous stasis.

Keith (25) demonstrated the importance of the combined action of the abdominal and thoracic muscles in inspiration thirty years ago. In that paper, while discussing the "abdominal type of respiration" (in contra-distinction to the normal "thoracic type" with efficient abdominal muscles), he described the diaphragm as an "abdominal piston." In the absence of the combined contraction of the abdominal muscles, this describes fairly accurately the effect of diaphragmatic contraction, and the result is that the pushing down of the viscera is substituted for the expansion of the bases of the lungs.

Not only do the muscles suffer from deficient oxygenation as a result, but the defective negative thoracic pressure contributes to intestinal venous stasis.

The importance of the circulatory factor on the tone of the muscles, the intestinal circulation, and, in a word, the general health of the individual, has been recognised by Cortlandt MacMabon (26). He has applied this principle practically by designing a series of breathing exercises which have merits not possessed by other methods in the following respect :

- (i) Full expansion of the lungs induces adequate oxygenation and development of the muscles.
  - (ii) The abdominal muscles themselves are exercised to the full during the exercises under the best conditions.
  - (iii) The fullest degree of negative pressure in the large thoracic veins maintains a normal abdominal circulation.
- and (iv) *Stress must be laid on the fact that, by constant practice, the correct method of breathing becomes automatic and independent of voluntary control ; therefore, the beneficial effect is not spasmodic, but continuous.*

The foregoing remarks apply significantly to the next section.

## INHIBITORY AND CIRCULATORY FACTORS COMBINED IN CHRONIC DISEASE

Reference has already been made to those numerous factors, extra- and intra-abdominal, which by their inhibitory stimuli may contribute to a derangement of normal gastro-intestinal functions both motor and secretory. In no circumstances do these inhibitory factors exercise more far-reaching effects than in subjects of *visceroptosis*. They are primarily responsible in many instances for initiating those secondary circulatory changes which, though gradual and intermittent in their establishment, induce the condition of chronic progressive ileus (distension, flatulence, constipation, constant discomfort, etc.) which is such a characteristic feature of sufferers from *visceroptosis*.

The constant absorption of gases from the intestine into the blood stream is responsible for the bad breath and dirty tongue; and it is not difficult to see in this picture, together with the classical symptoms in advanced cases, a mild and chronic, but reasonably faithful, mimicry of the dramatic symptoms of acute paralytic ileus.

In principle the immediate causation is identical.

## RIGHT COLOPTOSIS

It is possible to do no more than touch upon the fringe of so large a subject in the space available, though no discussion on ileus could be complete without reference to so important a type.

The mechanism is best explained by a concrete illustration, and the most convenient is the case of the mobile right colon.

It is from this point that the whole series of troubles in *visceroptosis* so commonly starts, but it is only fair to state at once that *it is not the actual position of the colon which matters—this means nothing whatever*. What is so important to realise is that it is only when the mesentery becomes a supporting structure in the upright position (and is submitted to mechanical tension) that pathological features begin to make their appearance. *The mesentery is not (and was never designed to be) a supporting structure*.

Whether the local condition is primary, and secondary extra-abdominal causes aggravate the picture (i.e. psychological), or whether the local condition merely accentuates a syndrome initiated primarily by extra-abdominal factors (either inhibitory, circulatory, or both), in either event the importance of mechanical tension on the mesentery as a pathological feature cannot be overrated.

Thus gradual sequelæ are as follows :

- (1) Inhibition from direct efferent sympathetic stimulation induces a mild degree of *local intermittent active ileus*.
- (2) Efferent reflex sympathetic stimuli induce *mild general active ileus*, intestinal, gastric, gall-bladder, etc., etc.
- (3) Gradually inefficient action and overloading lead to accentuated mesenteric strain and are followed by :
- (4) *Venous Stasis*. The veins are the most collapsible structures and suffer first. This leads to the dirty tongue, bad breath, etc.

Attacks of distension often lead to a mistaken diagnosis of chronic appendicitis, and appendicectomy does not stay the steady progress.

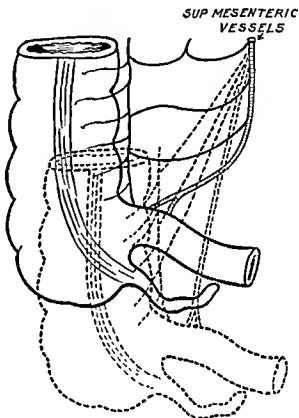


Fig. 618.—REPRESENTS DIAGRAMMATICALLY THE EFFECT OF MESENTERIC TENSION IN THE MOBILE RIGHT COLON WHERE A WEAK ABDOMINAL WALL, FAILURE OF THE MESENTERIC-ABDOMINAL REFLEX, WIDENING OF THE PELVIS, ETC., AFTER CHILDBIRTH (OR ONE OF THE MANY OTHER POSSIBLE FACTORS), LEAD TO FAILURE OF VISCERAL SUPPORT.

- (5) Strain on the lymphatics may lead to some degree of wasting, both local and general. Attenuation of the mesentery still further aggravates the susceptibility to mechanical strain.
- (6) The drag of the overloaded colon leads to nephroptosis and aching in the loin, while, in more pronounced cases, tension on the renal veins may lead to venous congestion of the kidney. This is occasionally evidenced by increased frequency of micturition, as well as the passage of a large quantity of urine—a feature which is absent during rest.



- (7) Constipation or irregular action of the bowels, flatulence, indigestion and anorexia gradually follow, so that it is easy to see how a vicious circle is established. Purgatives are as useless as they are harmful, for the same reasons as were given in discussing acute paralytic ileus.

It is interesting to note the lack of discomfort, flatulence, etc., together with the maintenance of robust health, in those with well fixed colons who are the subjects of habitual constipation of two and three days' duration. *The absence of symptoms is clearly attributable to the absence of inhibitory stimuli and to the absorption from venous stasis due to mesenteric tension.*

Again, attention should be drawn to the interesting parallel, already referred to, in the analogous acute condition.

The absence of "toxæmia" in acute obstruction is analogous to the maintenance of good health with marked constipation on the one hand; while, on the other hand, the chronic toxæmia in sufferers from visceroptosis is again a faithful but mild mimicry of the acute symptoms of acute paralytic ileus. These features are illustrated diagrammatically in figure 618.

If, as is the case, these troubles so often commence from mesenteric tension in a right mobile colon (7, 12), *a fixation of the right colon retroperitoneally* (in imitation of the normal process of development) can be expected to break this vicious circle and be followed by a gradual return to normal conditions without fear of relapse under appropriate treatment (of which breathing exercises are the most important).

It is the *promiscuous performance of such operations* (often most imperfectly carried out), or operations performed when the damage to the bowel wall is beyond repair, which has been responsible for a certain amount of disapproval.

The writer's twenty-six years of continuous experience of this type of procedure, with a progressively diminishing proportion of failures (owing to more careful selection), has convinced him more than ever of its value in curing or arresting the progress of these distressing symptoms.

#### SUMMARY

To sum up, it may fairly be stated that there is no aspect of gastrointestinal disease in which ileus does not, at some time or other, play an important part.

The assessment of its significance in any given instance depends upon the clear conception of three points :

- (1) An appreciation of the abdominal viscera as an *independent and automatic living organ*, capable of performing its functions independently of external influences, by virtue of its own normal neuro-muscular mechanism.
- (2) *Central control* by means of two types of stimuli :
  - (a) Parasympathetic (motor) to Auerhach's plexus.
  - (b) Sympathetic (inhibitory) to Meissner's plexus.

Both these antagonistic impulses (together with their synergic hormones—choline and adrenalin respectively) balance each other normally in controlling and adapting intestinal activity to the needs of the moment.

- (3) *The integrity of the intestinal circulation.*

On the unhampcred combination of these three systems depends the normal functioning of the gastro-intestinal tract, since the same systems control the secretory functions in an analogous manner.

It follows, therefore, that there are only three ways in which derangement can occur, whether in acute or chronic diseases :

- (1) *Mechanical interferences* :
  - (a) With movements of the viscera. This is rarely of importance.
  - (b) With the passage of contents down the lumen of the intestine, i.e. obstruction.
- (2) *Alteration in the normal balance of motor inhibitory impulses*, thus establishing undue domination of the one or the other. Examples of sympathetic domination have largely formed the subject of this chapter. (Examples of domination of the parasympathetic are a clinical entity of equal importance, e.g. enterospasm, duodenal ulcer, etc.) (9).

[*This is an entirely opposite type of case, outside the scope of our immediate study, which is often responsible for confusion in accurate diagnosis.*]

- (3) *Interference with the circulation* This requires no further elaboration.

An attempt has been made to show that, if these three factors are appraised at their proper value and applied to clinical work, empiricism will give way gradually to a scientific attitude both in diagnosis and treatment.

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SECTION 14

COMPLICATIONS FOLLOWING ABDOMINAL  
OPERATIONS

by  
RODNEY MAINGOT

CHAPTER I

Infected Abdominal Wounds

CHAPTER II

Burst Abdomen

CHAPTER III

Fæcal Fistula

CHAPTER IV

Acute Suppurative Parotitis

CHAPTER V

Post-operative Retention of Urine

CHAPTER VI

Bedsore

CHAPTER VII

Post-operative Thirst

CHAPTER VIII

Post-operative Pain

CHAPTER IX

Post-operative Phlebitis

by  
A. DICKSON WRIGHT

## SECTION 14

# COMPLICATIONS FOLLOWING ABDOMINAL OPERATIONS

## CHAPTER I

### INFECTED ABDOMINAL WOUNDS

*An infected wound is not necessarily one in which there is perceptible pus.* Infection of the wound following abdominal operations is a far too frequent event, and occurs more often than is generally admitted.

Eliason and McLaughlin state that a review of the literature indicates that imperfect wounds occur in approximately 10 per cent of all clean cases. Carraway reports 10 per cent, McKim 8 per cent, Goff 12.1 per cent, Roberts and Roberts 12.8 per cent, Thorek 7.17 per cent, Coley 12.8 per cent, and MacFarlane 7 per cent. In this day of aseptic surgery with rigorous pre-operative preparation of the surgical field and highly developed technique of closure and post-operative care, the high incidence of wound complications in the various surgical clinics is surprising. (*Annals of Surg.*, pp. 1159-60, Dec., 1934.)

The incision and the closure and management of the wound are important factors in any abdominal operation, and are worthy of more care and scrupulous attention to detail than is usually bestowed upon them.

The following factors may predispose to wound sepsis :

(1) Rough handling and excessive trauma of the tissues ; merciless, relentless stretching of the layers of the abdominal wall by means of retractors ; grasping and fraying of the abdominal muscles by tissue forceps ; and forceful pulling on the edges of the parietal peritoneum prior to its closure.

(2) *Inadequate preparation of the skin.* Prior to any abdominal operation, whatever its nature, the skin preparation should include the *whole area* of the abdominal wall, and at least the lower half of the chest and the upper third of the thighs, in addition to the flanks. We still too often see only a small portion prepared over the organ to be operated upon. For instance, in operations for chronic appendicitis only the lower half of the abdomen or the region of the right iliac fossa may have received attention; whereas at operation it may be necessary to make an incision elsewhere, or to make an additional incision when disease is discovered farther afield. The preparation of the skin is often perfunctorily performed, consisting merely of dabbing on a little iodine.

(3) *Inadequate protection of the wound.* Tetra-cloths or suitable towels should always be carefully clipped to the edge of the wound and maintained in position until its closure is almost complete. Thin handkerchiefs or mere strips of gauze which serve no useful purpose are often used as tetra-cloths.

During operation all the layers of the abdominal wall must be well protected by mackintosh sheeting, and all the exposed viscera must be covered with warm Cripps' or other suitable abdominal pads. No portion of any of the abdominal contents should be allowed to come in contact with the abdominal wound, except, of course, where it is necessary to anchor the gut to, or to deliver it through the abdominal wall for purposes of drainage, feeding, etc., e.g. colostomy, enterostomy. Additional protection will be required when a hollow viscus is opened, when a portion of the intestinal tract requires excision, or when an abscess or cyst is being drained.

(4) *Imperfect hæmostasis.* This is a frequent cause of stitch abscess and wound infection. A hæmatoma is very prone to become infected, and even if this does not occur it produces a painful tumour which may take many days or weeks to absorb. The blood forms a suitable nidus for the ingeneration of pathogenic bacteria. No dead spaces should be left in the wound, as serum or blood will collect in such cavities and favour the production of sepsis.

(5) *The nature of the operation.* Suppuration of abdominal wounds is particularly prone to occur after operations upon the stomach, duodenum, biliary system, or colon, and in all cases of peritonitis, whether localised or generalised.

It should always be assumed that the contents of the stomach,

duodenum, and small intestine are of a septic nature, and extra precautions should therefore be taken by packing off the viscera and the abdominal wound with additional layers of gauze or waterproof sheeting.

In cases of general peritonitis it is impossible to protect the wound efficiently against the infective exudate. In such cases, although drainage of the abdominal cavity may be indicated, it is essential also to make some provision for the drainage of the muscular planes and subcutaneous tissues in the region of the wound. This systematic protection of the wound and viscera forms an essential part of the technique of all abdominal operations.

(6) Unsuitable ligatures and sutures. Where there is frank sepsis, it is a bad practice to use silk, thread, thick chromic catgut, or such-like material for ligaturing the blood-vessels in the subcutaneous tissues as it will act as a foreign body and produce stitch abscess, and will eventually be discharged from the wound, or will have to be extracted should a persistent sinus be produced.

Even in clean cases, the use of stout ligature material is unnecessary and may lead to the formation of local suppuration. Only the points of the oozing blood-vessels should be clipped, and these should be tied with the finest plain catgut. Care should be taken not to strangle the tissues while sewing up the individual layers of the abdominal wall. Tension sutures should be tied firmly, but not in such a manner as to lead to strangulation and necrosis of the tissues.

The use of *very thick* chromic catgut for closing the peritoneum and other layers of the abdominal wall is unnecessary, as No. 1, or even No. 0, 20-day chromic catgut (double-stranded, if required), well applied, has been proved at numerous clinics to be wholly adequate and productive of good and uniform results. Silk, used both for ligaturing and suturing in abdominal work, has its definite limitations as well as its even more definite indications. In obese patients the fat is liable to infection, and here only the finest catgut thread must be used for ligaturing the blood-vessels.

(7) Wet towels, and cleaning the abdominal wall with saline at the completion of the operation. The use of hot moist towels instead of dry tetra-cloths for the abdominal wound is an often unrealised source of sepsis. These hot towels produce a great deal of sweating, and this sweat, which is always mildly infective, may be swept into the wound with the moisture that drips from the towels.

Another disadvantage of these wet towels is that they lose their

warmth in a very short while and become cold and clammy, leading to chilling of the skin. In addition to this, if the towels are not thoroughly wrung out, a quantity of the fluid contained in them may trickle away down the flanks, wet the towels under the back, and even pool up in this part.

At the completion of an abdominal operation it is a common sight to see the nurse clean up the surface of the abdomen and remove the blood-stains with a swab soaked in warm saline, and for the wound itself then to receive copious ablutions with the same swab. The area of skin around the wound must be considered as contaminated, particularly in any operation that lasts more than half an hour. The swab and the saline used for cleansing purposes carry infection—usually of a mild grade—into the wound, and are a definite cause of infection.

(8) Unnecessary and prolonged exposure of the suture and ligature material; its inadequate protection from contamination prior to use; allowing the threads to trail, or other carelessness in passing them to the surgeon.

(9) Laxity with regard to the antiseptic and aseptic principles entailed in the surgical ritual of cleansing the hands, donning gown, cap, mask and gloves, both by the surgeon, his assistants, and the nurses.

(10) A crowded operating theatre, or the casual incursion of spectators who have not previously equipped themselves in the recognised theatre outfit.

(11) Streptococcic carriers. "Meleney and Stevens have called attention to the role played by streptococcic carriers in the production of wound infections. In investigating a series of streptococcic infections developing within a short period of time, these authors found that one-third of the operating personnel were harbouring the hæmolytic streptococcus in their throats. These authors suggest that both the nose and mouth of all the operating team be adequately covered to protect against this source of wound contamination."

(12) The scalpel. "It is well recognised that various refinements in operative technique may do much to reduce superficial wound infections. The frequency with which a scalpel may carry organisms from the skin into the deeper layers of the wound has been referred to by Sutton, Thorek, Cox, Van Alstyne, and Carraway. The last-named



author cultured a series of 562 blades used to make the skin incisions and found that 117 or 20 per cent of them showed a positive culture." (Eliason and McLaughlin.)

#### TREATMENT

There are many degrees of wound infection, but the two commonest types are stitch abscess or localised suppuration in the wound, and generalised infection. A number of infected wounds do not suppurate, but they cause a great deal of local discomfort or pain, and give rise to a certain amount of toxæmia. The appearance of these mild septic wounds is quite typical. As a result of the surrounding inflammatory oedema the stitches appear to be buried in the skin, the edges are covered here and there with inspissated blood, there may be some oozing of serum between the sutures, or a faint red blush may be discernible in the region of the line of incision. By removing a few alternate stitches and applying hot fomentations or cold eusol or mag. sulph. compresses, these wounds may, in most cases, be induced to heal without further complication.

*Stitch abscess* usually develops about the tenth day, but may occur earlier than this, before any of the stitches are removed, or even some days or weeks after the wound has apparently healed quite soundly. It may be deep-seated or superficial. When the former, an indurated tender lump may be felt in the depths of the wound; when the latter, the typical red or mauve coloured, fluctuating, circumscribed blister or swelling appears somewhere in the line of incision.

When this swelling is incised, a little blood-stained pus is evacuated, and by applying pressure to both sides of the wound the offending stitch may be expelled. Usually, however, the cavity will have to be curetted out, or if a fair-sized suture or ligature is visible it should be picked up with a hæmostat and gently withdrawn. This cavity is then packed with a little gauze that has been soaked in paraffin and flavine, eusol, or pure peroxide, or preferably irrigated with a 2 per cent aqueous mercurochrome solution.

Another type of infection is the small localised hæmatoma, which can be successfully treated by probing the wound and gently separating its surfaces with a pair of sinus forceps or a small hæmostat to give free vent to the discharge.

Where there is a large collection of blood under the skin, or where there is much fat in the abdominal wall which has become infected during the process of operation, *infection with bacillus coli communis*

is very prone to occur with the formation of a large abscess. Such wounds have a dusky, mottled appearance, are swollen, tender, and associated with a great deal of constitutional disturbance. The pulse is usually quick, the temperature is raised, the tongue is dry, and the patient is often gravely ill. Owing to the tenderness of these wounds it may be impossible to elicit the sign of fluctuation, but the appearance should guide the surgeon to remove a stitch or two, and to probe the depths of the wound for deep-seated pus. Pus, when located, is usually under great pressure and may pour forth in a stream or spout with jet-like force. The discharge is evil-smelling, mud-coloured, and creamy with globules of oil. Gas may bubble up through the wound from time to time when the discharge is profuse, and sloughs may be expelled through the process of irrigation.

Resolution and healing rapidly occur when such wounds are drained with a small rubber tube and frequently irrigated with hydrogen peroxide or 2 per cent mercurichrome. Prior to drainage, however, much serious damage may have been inflicted upon the muscles, their sheaths and other structures, and considerable necrosis may have occurred. If the necrosis involves a large blood-vessel in the abdominal wall, such as the inferior epigastric artery, there may be signs of a copious hæmorrhage which even packing or compression of the wound may fail to control.

These cases of *secondary hæmorrhage due to a suppurative myositis* are very serious problems, and are best dealt with by completely opening up the superficial portions of the wound under a general or spinal anæsthetic, locating the bleeding area, and applying ligatures a short distance above and below the bleeding points. This may be exceedingly difficult to perform as the tissues will be cheesy with inflammation, the sutures will readily cut out when applied, and the vessels responsible for the hæmorrhage may almost defy detection and isolation.

When proximal and distal ligature is impossible, a fresh incision will have to be made in healthy tissue to isolate and ligature securely the artery which is deemed to be the primary cause of the hæmorrhage.

When bleeding is controlled, the wound is packed with gauze soaked in peroxide of hydrogen, and the major portion left unsutured to permit of frequent irrigation, repacking, and subsequent healing by granulation tissue.

In other cases of *generalised infection* of the wound it will not be necessary to remove all the skin sutures. A few stitches are allowed to remain, and between these tubes are inserted into the depths of the wound for the purpose of drainage and irrigation. Such wounds

should be thickly coated with zinc and castor oil ointment in order to protect the skin from blistering through the frequent application of hot fomentations.

A *persistent abdominal sinus* is always a cause for anxiety, as it usually implies that a foreign body, e.g. unabsorbable ligature or suture material, a portion of a drainage-tube, a swab, a faecal concretion, etc., is either imbedded in the wound or deep to it in the abdominal cavity, or that one of the hollow viscera is in direct communication with the surface of the abdominal wall by means of a fistulous tract.

## CHAPTER II

### BURST ABDOMEN

THE following are some of the many alternative titles used to describe the condition known as burst abdomen :

- (1) Separation of the abdominal wound.
- (2) Post-operative evisceration.
- (3) Post-operative eventration.
- (4) Prolapse of the intestine.
- (5) Disruption of the abdominal wound.
- (6) Deliscence of the abdominal wound.

Burst abdomen is a tragic and serious complication which may follow any abdominal operation, and it has been computed to occur after approximately 1 per cent of all abdominal operations. When it occurs it presents many serious problems in the management of the patient.

#### CAUSES

(1) Failure of the Tissues to Heal Soundly. This may be attributable to :

(a) *The general state of the patient.* Where the patient is obese, aged, decrepit, toxæmic, diabetic, anæmic, nephritic, or alcoholic, the regenerative powers of the tissues are in abeyance.

(b) *The nature of the disease.*

(i) Diseases of the liver, gall-bladder, and bile-ducts. The incidence after operations upon these viscera is about 1.5 per cent, jaundice and colæmia, when present, being contributory factors.

(ii) Visceral cancer. The comparatively high incidence of burst abdomen following operations for cancer of the abdominal viscera (approximately 2-3 per cent) is due in a large measure to the toxæmia, anæmia, and debility often present in such cases.

(iii) Intra-peritoneal suppuration (diffused or localised).

(iv) Acute hæmorrhagic pancreatitis, the dehiscence in such cases being due to the action of escaping ferments.

(v) Ascites (from any cause).

(vi) Appendix operations.

(vii) Uterine fibroids. Here also there is often anæmia, weakness, and general debility.

It should be stressed that any intra-abdominal lesion associated with toxæmia, anæmia, cachexia, and lowered resistance of the patient, will predispose to burst abdomen.

(c) *Details of operative procedure.*

(i) The position of the incision. About two-thirds of all cases occur after operations upon the upper abdomen, and most of these are right muscle-split (transrectus) incisions.

(ii) The magnitude or character of the operative procedure and the time taken over the operation. These are both small factors, for burst abdomen may occur after a simple exploration lasting only a few minutes or after a more serious operation occupying one or two hours.

(iii) The viscus operated upon. Burst abdomen is commoner after operations upon the stomach, duodenum and gall-bladder than after operations upon any of the other abdominal viscera. The incidence is not dependent so much upon the actual technical procedure as upon the poor condition of the patient as a result of the disease from which he is suffering, e.g. cancer of the stomach, gall-stones with jaundice, etc.

(iv) Contamination of the wound. Contamination frequently occurs after operations for general peritonitis, perforation of a hollow viscus, resection of intestine, and operations for acute hæmorrhagic pancreatitis. Suppuration of the wound, with its attendant evils such as friability of the tissues and a tendency to more rapid digestion of catgut sutures, is prone to occur after operations for such conditions.

"There are cases of early dehiscence in which the chromic gut appears to have completely dissolved. Whether this dissolution is dependent upon an idiosyncrasy of the individual to digest catgut quickly, or whether it is due to an inferior grade of catgut, are open questions." (Ralph Colp.)

(v) The necessity for drainage. Burst abdomen is undoubtedly commoner where drainage has been instituted, but is due not so much to the presence of the tube itself in the wound as to the underlying pathological condition which necessitated its employment.

(vi) Operations performed through previous scars. These wounds have the reputation of being more prone to dehiscence, but it is very doubtful whether this assumption is correct, particularly as patients who have stood one operation well are likely to stand a second operation equally successfully, especially if care is taken to preserve as much tissue as possible and to carry out a well-planned plastic repair of the abdominal wall.

(vii) The choice of anæsthetic. This has no bearing upon the problem, as it has been conclusively proved that burst abdomen occurs with equal frequency after local, spinal and inhalation anæsthesia. Stormy anæsthesia means stormy convalescence.

(2) The Occurrence of Certain Post-Operative Complications. The following may be contributory factors :

(a) *Persistent cough.* Pulmonary complications interfere with the splinting of the abdominal wall and thus predispose to burst abdomen. Persistent coughing, although very distressing to the patient and subjecting the wound to considerable strain, is not, of itself, usually sufficient to cause disruption of the wound unless there is some underlying primary defect in the regenerative powers of the tissues. A large number of patients develop chest complications after abdominal operations, but in very few of such cases does the wound actually break down.

(b) *Infection of the wound.*

(c) *Distension.*

(d) *Repeated gastric lavage.*

(e) *Intractable hiccough.*

(f) *Vomiting.*

(g) *Undue strain* such as may be occasioned by restlessness, the patient getting out of bed, or difficulty in defæcation or micturition.

(h) *The action of digestive ferments*, as occurs after operations for acute pancreatitis and perforated peptic ulcer.

As all these post-operative complications are aggravating factors in wound disruption, every effort should be made to reduce their incidence, and, when present, to diminish their severity by a skilfully applied regime of post-operative management.

(3) *Errors in Operative Technique.* Whilst it is generally admitted that disease, leading to poor and ineffectual healing, is the primary cause of burst abdomen, a certain number of cases are attributable to errors in operative technique. The surgeon is responsible for :

- (a) *A sterile technique.*
- (b) *The choice and type of incision.*
- (c) *Damage to the tissues.*
- (d) *The choice of suture and ligature material.*
- (e) *The type and position of the drainage material employed.*
- (f) *The method of closure of the abdominal wound, and the employment of additional safeguards against undue tension in the wound.*

The wise surgeon, experienced in abdominal surgery, will recognise where extra precaution in the closure of the wound is called for, and by adapting his technique to such cases he will materially reduce the occurrence of this complication in his series of cases.

The following points relating to choice of incision and to closure of the abdominal wound are worthy of particular note :

(i) The position and length of the incision. Disruption is at least two-thirds commoner in wounds in the upper half than in the lower half of the abdomen, and is commonest after the employment of the rectus muscle-split incision, this being the incision most frequently used for operations upon the stomach, gall-bladder, and for other upper abdominal procedures. Wounds situated in this region are also subjected to much greater abdominal pressure or strain as the result of coughing, distension, etc., than those in the lower half of the abdomen.

It is stated that disruption of abdominal wounds is rarer with transverse or oblique incisions than with vertical incisions. The main objection, however, to the routine employment of transverse incisions is that the area afforded for exploration is somewhat limited. I have found Kocher's oblique sub-costal incision very suitable for unequivocal cases of disease of the gall-bladder or biliary passages, and have not seen a case of burst abdomen or post-operative ventral hernia follow its use.

In this incision, however, whilst the eighth dorsal nerve may be sacrificed, the larger ninth must be isolated and protected from harm during operation. Incisions which are either unnecessarily large or unnecessarily small are to be deprecated. A large incision in an unsuitable subject predisposes to wound disruption, and a small

incision increases operative trauma and considerably interferes with the necessary intra-abdominal manipulations.

If a sub-umbilical incision has been made for suspected disease of the pelvic organs or appendix and some other lesion in the upper abdomen is revealed, it is far better to close the primary incision and to make another in the epigastrium than to prolong the primary incision into this area, and vice versa.

(ii) *Suture material.* The employment of unsuitable material, such as excessively heavy catgut, hasty and careless methods of suturing, the introduction of sutures in such a manner as to produce strangulation of the tissues, and the omission of retention sutures in debilitated patients are all recognisable factors in the production of disruption of the wound. The methods advised for the closure of abdominal wounds are fully discussed elsewhere.

(iii) *Drainage-tubes.* It is probable that drains are used *too often*, even in cases of peritonitis. In cases of established infection of the peritoneal cavity, however, there are very few surgeons who would have the courage to dispense with the use of a drainage-tube, however dubious they might be as to its actual benefits. In all cases of peritonitis it is good surgical practice to provide drainage of the subcutaneous tissues, and even down to, though not necessarily always through, the line of peritoneal closure.

When drainage of the peritoneal cavity is employed, the tissues should be approximated snugly round the tube, not so tightly compressed as partially to block the lumen of the tube, or so loosely as to allow a knuckle of gut or a portion of omentum to protrude and become strangulated or to cause an omental wedge to bulge through the interstices of the incision, work its way to the surface, and so predispose to the initial stages of burst abdomen.

Although published statistics show a higher number of undrained than of drained cases, it must be borne in mind that the total number of drained abdominal wounds is much lower than the number of undrained. It seems almost self-evident, however, that burst abdomen will occur in a higher percentage of drained than of undrained cases, the very necessity for drainage indicating that the patient is suffering from intra-peritoneal suppuration with its attendant toxæmia, a combination of factors well recognised as being associated with a comparatively high incidence of wound dehiscence. It has been shown, also, that where there is a large amount of exudation from the wound the absorption of catgut is much more rapid.



## FREQUENCY OF BURST ABDOMEN AND ITS MORTALITY

Sokolov (1932) analysed the replies which he received from over 1000 questionnaires circularised to surgeons in various parts of Europe, and came to the conclusion that the incidence of burst abdomen ranged from 2-3 per cent of all abdominal operations. There were 730 cases with a mortality of over 30 per cent. Ralph Colp (1933) in 2,750 abdominal operations reported 29 cases with an incidence of 1.12 per cent for males and 0.75 per cent for females, with a total mortality of 28 per cent. C. G. Heyd (1933) in 2,145 abdominal operations records only four cases of burst abdomen, i.e. one case in 536. There was one death, a mortality of 25 per cent. The figures for the New York Post-Graduate Hospital (1933) are very similar, showing that out of 1000 cases operated upon there were four cases of wound dehiscence, i.e. one in 250. There was again one death—a mortality of 25 per cent.

Ralph Colp, who has made an extensive study of the subject, offers the following conclusions :

“Wound rupture as such probably contributes very little directly to a lethal outcome. Dehiscence itself is fairly innocuous, but it tokens the presence of fundamental defects and represents the result of serious complications.”

Again : “While no disease affecting the peritoneum or its contents is free from the complication, it appeared to be more frequent in certain diseases, especially in carcinoma, certain gynaecological conditions, notably uterine fibroids, and chronic infections of the biliary system, in fact, those maladies characterised by chronic toxæmia and accompanied by anæmia, cachexia, weakness and general debility. The crux of the problem seemed to rest in the failure of the regenerative powers of the tissues to promote firm healing. It was aided and abetted by certain precipitating factors as pulmonary infections, gastric dilatation, and meteorism.”

## DIAGNOSIS

The diagnosis is evident when there is prolapse of the intestines through the wound (fig. 619), and where the intestines are literally extruded into the bed. The less obvious cases are those in which : (a) All the layers of the abdominal wall have given way except the skin, and the coils of gut which lie subcutaneously produce a tumour which may resemble a giant hæmatoma, or may be felt as an elastic doughy mass. In such cases there is usually a visible impulse on

coughing, and there is increase in the size of the tumour when the patient strains. The skin incision may be perfectly healed, but more often there is a point here and there through which pink blood-stained serum continuously dribbles. (b) The wound has given way, partially or completely, and in its depths unrecognisable, fleshy, plum-coloured, anatomically indefinable structures appear. It is easy to deceive oneself into thinking that this is only muscle, but more often than not it will prove to be omentum, a portion of the colon, or coils of intestine, matted and covered with granulation tissue.

Disruption most commonly occurs on the day that the skin sutures

are removed, i.e. about the seventh or eighth post-operative day. Early recognition of the condition is important, as the prognosis is more favourable when treatment is instituted without delay. A persistent, pink, watery discharge of peritoneal fluid or blood-stained serum through points in an apparently healthy wound is always an omen of grave import, suggesting that a slow but gradual disruption is taking place in the depths of the wound.

"Disruption probably occurs acutely in most cases. In some patients the condition is slower and the final breaking down of

the wound may disclose abdominal contents already adherent to the wound edges and to each other. It is probable that the very early ventral herniæ noticed in the follow-up clinic may be examples of this group of slow disruption in which the skin and subcutaneous tissues healed without disclosing the deeper wound defect." (R. V. Grace.)

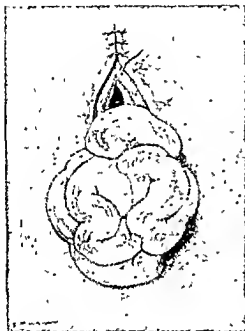


Fig 619.—BURST ABDOMEN.

#### GENERAL PROPHYLACTIC MEASURES

Burst abdomen will never be a wholly avoidable complication until more reliable tests and methods are available whereby to foresee with any degree of accuracy what the recuperative powers of the patient following abdominal operation will be. We have at the present time

to rely largely upon clinical examination of the patient, and to a lesser degree upon laboratory tests, which, although decidedly helpful, are incapable of determining the vital capacity of the patient.

Much, however, can be done to improve the patient's condition whilst he is awaiting operation, to increase his resistance, and to diminish or prevent the risk of serious post-operative complications. Obese patients should be dieted and scientifically treated to reduce their weight, whilst blood-transfusions, drugs (e.g. iron and arsenic), the liberal administration of water, dextrose, vitamins, and nutritious and assimilable diet are indicated for anæmic, debilitated, and cachectic patients. Sunlight treatment is also a useful pre-operative adjunct.

The routine application of the following surgical principles in the closure of wounds tends to diminish the incidence of burst abdomen :

- (1) Avoidance of undue trauma of the tissues.
- (2) Complete hæmostasis.
- (3) Ensuring complete relaxation of the abdominal wall during the process of closure of the wound.
- (4) The obliteration of dead spaces.
- (5) Accurate and precise coaptation of the peritoneum and other layers of the wound.
- (6) An impeccable aseptic technique.
- (7) The insertion of supporting sutures where necessary.

#### TREATMENT

There are two methods of treatment :

- (1) *Re suture*.
- (2) The tampon method, i.e. packing and strapping of the wound.

When it is realised that the abdominal wound has broken down, a decision will have to be made as to whether the condition should be treated by means of packing and strapping or whether re-suture should be undertaken. It is sometimes very difficult to be sure which is the best course to pursue, but in the majority of cases re suture by one of the methods about to be described is the procedure of choice.

*Re suture.* As soon as the condition is recognised the abdomen should be entirely covered with a sterile turkish towel or large

abdominal pads wrung out in warm normal saline solution, and a many-tailed bandage loosely applied. After reassuring the patient and impressing upon him the necessity of not coughing or making any physical effort, an injection of morphia,  $\frac{1}{4}$  gr., or omnopon,  $\frac{1}{3}$  gr., with  $\frac{1}{150}$  gr. of scopolamine should be injected to soothe him and to produce a condition of hypnosis before removing him to the operating theatre. No attempt at replacing the protruded viscera should be made while the patient is still in bed, or until a hypnotic or some form of anæsthetic has been administered. If re suture of the wound is attempted without an anæsthetic the patient may strain or cough and even further the evisceration. Replacement will then, of necessity, prove more difficult, give rise to a great deal of shock, and the subsequent suturing, even if possible, will have to be done hurriedly and often in a most unsatisfactory manner.

After the patient has been anæsthetised (on the operating table) the abdominal binder and the dressings are removed. The skin of the abdominal wall and the prolapsed gut are drenched with warm normal saline solution. When the prolapsed viscera have been replaced further protrusion is prevented by packing a damp Cripps' pad or large abdominal swab snugly into the abdominal cavity. The wound is mopped dry, loose pieces of catgut or silk-worm-gut are extracted, and the surrounding skin is painted with spirit. If only a small portion of the wound has gaped this part only will need to be re-sutured. If, however, more than half of the original incision has burst asunder it is more satisfactory to open the remainder of the wound, remove the



Fig. 620.—HOOK RETRACTOR.

previous stitches, and undertake a complete re suture. This is performed in the following manner: A Cripps pad or large abdominal swab, if placed in correct position, will effectually prevent the intestines from bulging into the wound. Hook retractors are then placed at each extremity of the wound and elevated so as to lift the abdominal wall away from the underlying abdominal pack (fig. 620). A series of through-and-through No. 8 plaited silk sutures are inserted about one inch from the margins of the wound and one inch apart (fig. 621). As

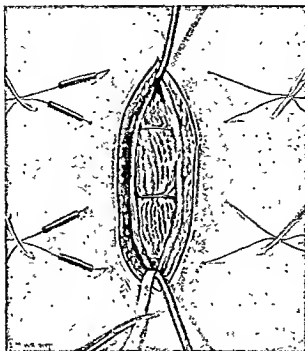


Fig. 621.—METHOD OF RE SUTURE. HOOK RETRACTORS AND ABDOMINAL PAD IN POSITION. FOUR THROUGH AND THROUGH TENSION SUTURES HAVE BEEN INTRODUCED.

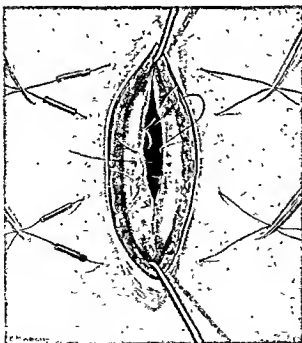


Fig. 622.—METHOD OF RE SUTURE. A SERIES OF MATTRESS SUTURES ARE INSERTED, DRAWING TOGETHER ALL THE LAYERS OF THE ABDOMINAL WALL EXCEPT THE SKIN. THE HOOK RETRACTORS EXERT AN UPWARD PULL UNTIL ALL THE THROUGH-AND-THROUGH SUTURES ARE TIED.

soon as they are passed they are put on the stretch and clipped at each end with hæmostats. These sutures embrace all the layers of the abdominal wall, including the peritoneum. No attempt should be made to define the margins of the peritoneum. It is futile to endeavour to approximate the edges of the peritoneal gap with a continuous suture, as the thread will invariably cut through this cedematous and friable structure, and in addition produce an unnecessary amount of trauma and bleeding. The divided muscular layers with their overlying sheaths should be approximated with interrupted mattress sutures of No. 3 20-day chronic catgut (fig. 622). To facilitate the suturing the skin edges may have to be slightly undermined.

The abdominal pack should be removed before completing the suture of this layer. The tension sutures should be threaded on thin rubber tubing and tied, firmly but not too tightly, and the skin edges drawn together with a few interrupted sutures here and there as may seem necessary. A small piece of corrugated rubber tubing should be used to drain the lower recesses of the wound, as sepsis often follows re suture.

The hook retractors should not be removed nor should the assistant for one moment relax the upward pull on them until the last through-and-through suture has been tied. This upward traction and keeping the through-and-through sutures taut are essential to prevent a stray loop of intestine from becoming ensnared.

The operation is completed by the application of an adhesive abdominal corset, either improvised or as specially designed for such cases. The wound will subsequently need to be dressed at least twice a day as there may be some discharge or localised suppuration. The skin stitches are removed on the seventh day, but the through-and-through or tension sutures should not be withdrawn until the end of a fortnight.

As considerable weakness of the abdominal wall, or even a ventral hernia, may result, some abdominal corset or belt will have to be worn for a long period.

*The method of suture by silver-wire* is very satisfactory for the closure of disrupted wounds, in addition to its usefulness in certain cases of acute abdominal emergencies, as has been emphasised by Mont R. Reid, M. M. Zimninger, and Paul Merrell, in a very interesting article published in the *Annals of Surgery*, Aug. 1933. They write :

"The method as we use it is as follows : Ten- to twelve-inch lengths of virgin silver-wire (the most pliable wire should be used ; it is our custom to buy it in one-ounce lots and rolled on a spool) No. 20 gauge, are threaded on large, curved,

cutting edge needles such as are ordinarily used for inserting 'traction' or 'tension' sutures. The short end is folded back over the eye of the needle and crushed flat with a heavy forceps so the wire will more easily go through the hole made by the needle. A clamp is fastened on the free end as is done with silkworm-gut 'stay' or 'tension' sutures. A series of clamps is placed on the edge of the peritoneum. All the silver-wire sutures are then placed but not tied. For each suture the needle is started about one or one and a half inches

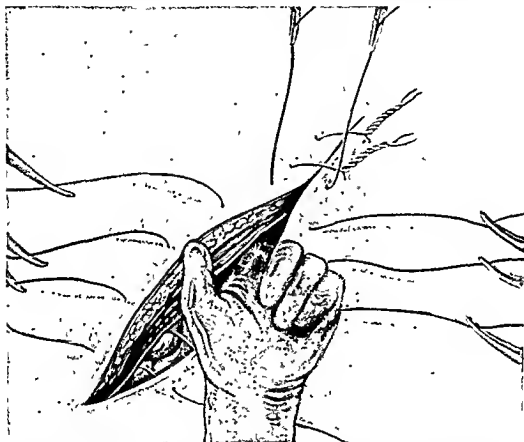


Fig. 623.—METHOD OF SUTURE BY SILVER WIRE METHOD EMPLOYED IN PULLING UP AND TWISTING THE SILVER SUTURES. THE FINGER IN THE ABDOMEN PROTECTS THE INTESTINES AND LETS ONE KNOW WHEN THE SUTURE IS PULLED SUFFICIENTLY TIGHT.

(NEED, ZINNINGER, AND MERRILL.)

(By kind permission of the "Annals of Surgery.")

from the edge of the incision and carried through the entire thickness of the abdominal wall, including the peritoneum. The suture is continued by bringing it out at a corresponding place on the opposite side of the incision. It is important that no kinks be allowed to get in the wire during this step as they are exceedingly difficult to get out smoothly. The needle is unthreaded and a clamp placed on the free end of the wire. A series of such sutures is placed about one and one-quarter to one and one-half inches apart, five to eight being used to close the average incision. After all are placed, the clamps on the peritoneum are removed and the incision closed by pulling up and twisting each wire individually

(fig. 623). Beginning at one end of the incision, the operator pulls up on the clamps at opposite ends of one suture. The first assistant puts a finger inside the abdomen and reports when the wire is pulled sufficiently tight to bring the peritoneal edges in firm contact. The wire is then twisted six to eight times just above one of the openings through which it emerges from the skin—not over the line of incision. Each wire is pulled up in succession and twisted. It is extremely important that the desired tension be obtained before the twisting is started because the twisting is for the purpose of holding only and will not tighten the suture nor remove any slack in it.

"After all the sutures are fixed in this way, one or two silk stitches may, if necessary, be placed in the skin between each two wires to prevent eversion or inversion of the skin edges. The wires are cut rather long, as they are easier to manipulate in the dressings if an inch or more of straight wire is free beyond the

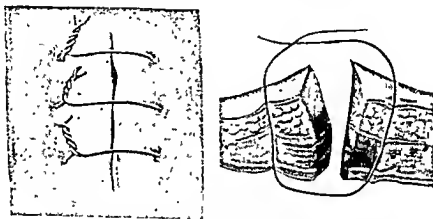


Fig. 624.—METHOD OF SUTURE BY SILVER WIRE. DIAGRAM SHOWING POSITION OF THE WIRE AND ILLUSTRATION OF THE WOUND AFTER CLOSURE.

(REID, ZINNIGER, and MERRILL.)

(By kind permission of the "Annals of Surgery.")

twisted part. No rubber tubing or other material is placed about the wires where they cross the incision or between the wires and the skin (fig. 624). There is usually some cutting of the skin under the wires before they are removed, but this has never constituted a serious complication of wound healing. Various modifications have been tried in an effort to prevent this cutting of the skin, but the method just described has been more satisfactory than any of the modifications. This entire procedure can be carried out in only a fraction of the time necessary for a formal closure of the incision in layers.

"It is also possible by this method to close a wound which is under considerable tension or one in which the peritoneum fails to hold sutures but tears with each attempt to pull it together. In addition, this closure is extremely valuable in all cases in which there is likely to be infection, as it gives a very secure closure which is not affected by infection or even by extensive slough of the fascia. . . .

"Since its adoption there has been no case of rupture of an incision closed by silver-wire during ten years. . . .

"With this type of closure, the following *objections* and *disadvantages* may



be raised: The most marked objection is on the part of the patients, who, almost without exception, complain of pain in the incision. In spite of using the most pliable silver-wire we can get it is more stiff than other suture material, and causes more pain than the average 'tension' or 'stay' sutures. Second, there is usually some infection around the wire. This may be minimal and the incision itself heal without infection, but it is rare to have no discharge from the wire holes, although this is no greater than that which occurs around other sorts of 'tension' or 'stay' sutures in the same kind of cases. Third, there is usually some cutting of the wires into the skin, which causes an obvious cross-hatching of the incision, and in patients with a tendency to keloid formation this may lead to a disfiguring scar. Fourth, the theoretical objections of incomplete closure of the peritoneum predisposing to hernia and obstructive intra-abdominal adhesions have not been borne out in fact. Fifth, we have never seen a block slough of the tissue enclosed by the sutures, although the possibility of occurrence has been a source of worry.

"It is our opinion that the method offers the following *advantages*: First, the closure is very secure. In spite of severe infection, we have had no case of post-operative rupture of the incision and no evisceration. . . . Second, the closure can be carried out very rapidly, so that patients in critical condition can be got off the operating table very quickly after completion of the intra-abdominal manipulations. Third, it can be used to close an abdomen under tension; for example, in the case of an intestinal obstruction, and when other sutures fail to hold. Fourth, an incision of this sort can be easily reopened by untwisting the wires, if a second operation is necessary shortly after the original one. The wires can be pulled aside, the necessary procedure carried out, and the wires pulled up and retwisted. Fifth, in cases of peritonitis or potential infection, such as is present after the perforation of a hollow viscus, the closure with interrupted sutures allows the escape of peritoneal exudate between the sutures without the placing of drains. The absence of any suture material directly in the line of closure of a contaminated incision predisposes to more benign wound healing and reduces the liability to infection. Sixth, the closure is so secure that old and debilitated patients can be got out of bed very early, as soon as five to seven days after an operation through a long rectus incision. Seven, the incidence of post-operative ventral hernia is no greater than after other more formal types of closure in the same types of cases, so far as we have been able to determine. . . .

"It is interesting to note that there have been only eight cases of post-operative evisceration in eleven years, and none of these occurred when silver-wire was used. We attribute this low incidence to the fact that we use silver-wire to close practically all wounds in which evisceration is prone to occur. . . .

"When silver-wire is used in this manner, the sutures are ordinarily removed about sixteen to eighteen days after operation. If they are found to be too tight at any time, they can be loosened by untwisting them, allowing a little slack to be taken up by the wound and retwisting them. . . .

"Ordinarily, part of them are removed about the fifteenth or sixteenth day, and the remaining ones the seventeenth or eighteenth day, though at times all of them are removed at one stage."

*The Tampon Method.* Generally speaking, the tampon method may be recommended in the following conditions:

(a) Where the patient's condition is such that any secondary operative procedure would be too hazardous.

(b) Where the disrupted wound is very foul and suppurating.

(c) Where the disruption has only involved a very small and localised area.

(d) Where on prying into the depths of the wound it is seen that the separation of the muscle layers is minimal, and that the abdominal contents are prevented from further evisceration by being firmly adherent to the deeper tissues.

(e) Where disruption has occurred in a case of frank purulent peritonitis.

When employing this method no anaesthetic is required. If there has been any evisceration, the abdominal contents should be gently replaced and the gaping wound snugly packed with gauze soaked in paraffin and flavine. The wound edges are then approximated as closely as possible with elastoplast strapping or narrow strips of flamed adhesive tape, and a firm dressing applied. Even though discharge may be profuse and offensive and the bandage become sodden, this strapping is not interfered with for two or three days, fresh dressings being applied to the surface of the strapping. After this time the strapping and the plugging are removed, and the process is repeated.

When the wound becomes reasonably clean an abdominal corset is applied to encourage the approximation of the edges and to facilitate the dressing of the wound.

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## CHAPTER III

### FÆCAL FISTULA

It is sometimes exceedingly difficult to distinguish between a faecal fistula and a discharging hæmatoma infected with *bacillus coli*. The discharge from a *bacillus coli* abscess in the wound may simulate that from a faecal fistula in that it may be highly offensive, dark brown in colour, and gas may bubble up from time to time. Eventually, however, the true nature of the discharge (liquid faeces) leaves no room for doubt.

In some cases of general peritonitis the occurrence of a faecal fistula may be a blessing in disguise, the fistula acting as an enterostomy.

#### CAUSES

(1) Erosion of a portion of the gut by a drainage-tube.

(2) Leakage from the line of suture following operations upon the appendix, large or small intestine (e.g. ileo-cælostomy), or disruption of a small necrotic portion of the gut which has been closed by means of interrupted Lembert or purse-string sutures.

(3) Where, owing to a localised patch of gangrene which has escaped detection at operation, a portion of the gut wall is discharged as a slough. This is commonly seen after drainage of an appendix abscess or in cases of appendicitis associated with a severe inflammatory condition of the caecal wall. It is also sometimes seen after operations for the relief of strangulated hernia and other abdominal lesions associated with strangulation or obstruction of the intestines.

(4) When an abscess due to diverticulitis of the colon is opened and drained, a faecal fistula almost invariably results.

(5) Rare cases where advanced cancer of the gut has eroded all the layers of the abdominal wall.

Approximately 80 per cent of all cases of faecal fistula are due to a leak in the caecal wall following operations for gangrenous appendicitis.

A faecal fistula is deliberately produced by the operation of cæcostomy which is often undertaken for the relief of obstruction.

### TREATMENT

(1) Diet. Only small quantities of fluid nourishment are permitted by mouth, and the patient is kept on a semi-starvation diet.

(2) Medicines. The administration of the following powders is recommended: bismuth oxycarbonate, 30 grs. six-hourly, and/or pulv. creta aromat., 1 dr. four-hourly.

(3) Enemata. A daily saline howel wash-out or soap enema is given until the fistula closes. No purgatives are given by the mouth.

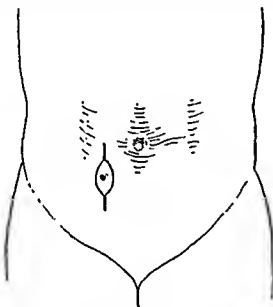


Fig. 625.—FÆCAL FISTULA. AN OVAL INCISION IS MADE TO INCLUDE THE FISTULOUS OPENING, AND THIS MAY BE FURTHER EXTENDED AT EACH END.

(4) Treatment of the wound. As soon as the condition is recognised, a few stitches should be removed here and there to give free vent to the discharge. If a drainage-tube has been inserted at the time of operation it is well to remove it as it may aggravate or even have been the primary cause of the condition. The surrounding skin should be dressed with horse scrub, or protected with thickly applied zinc and castor oil ointment, hyd. subchlor., 30 per cent in lanoline, or liquid rubber solution. Gentle irrigation of the wound with weak hydrogen peroxide, dilute Sanitas, or some other deodorant antiseptic solution should be undertaken at frequent intervals. If the wound is severely infected and there is evidence of pocketing of pus, a small counter-incision should be made for drainage purposes.

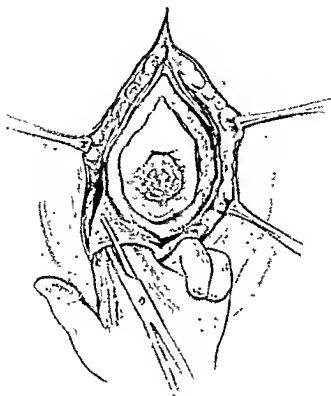


Fig. 626.—FÆCAL FISTULA. A SMALL GAUZE SWAB HAS BEEN PACKED INTO THE FISTULA TO PREVENT LEAKAGE DURING THE OPERATION OF ISOLATION AND EXCISION OF THE FISTULA. THE PERITONEUM IN THE LOWER PART OF THE WOUND IS BEING DIVIDED WITH SCISSORS.

(5) Operation. The majority of fæcal fistulæ will respond very satisfactorily to the treatment outlined above, and will heal firmly and soundly. A few cases, however, will develop a small ventral hernia which may or may not require a subsequent plastic operation.

*A fæcal fistula will not close spontaneously if the mucous membrane of the gut is continuous with the skin.* An operation will be necessary in these cases, and is performed in the following manner. The fistulous opening is tightly packed with gauze to prevent any discharge during the operation, and the surrounding skin is thoroughly swabbed with absolute alcohol. An oval incision is made to include the fistulous opening, and this may be further extended at each end (fig. 625). The incision surrounding the fistula is carried through all the layers of the abdominal wall, until the peritoneum is reached. The peritoneum is

opened at the upper or lower end of the wound to break down the adhesions and to isolate, as far as possible, that portion of the gut from which the fistula arises (fig. 626).

As soon as the gut has been sufficiently mobilised, the parietal peritoneum attached to the under-portion of the isolated segment of the wound is divided with scissors, which permits of the gut being withdrawn through the wound. The fistulous tract with its attached portion of abdominal wall is removed by excising the base of the fistula by an incision through a healthy area of the gut wall (fig. 627). The resulting gap in the intestine is closed transversely by a two- or three-tier suture.

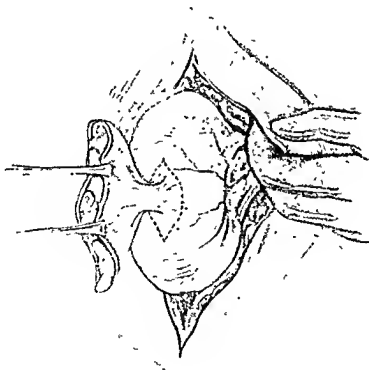


Fig 627.—FACIAL FISTULA. THE FISTULOUS TRACT WITH ITS ATTACHED PORTION OF ABDOMINAL WALL IS REMOVED BY EXCISING THE BASE OF THE FISTULA BY AN INCISION THROUGH A HEALTHY AREA OF THE GUT WALL.

THE RESULTING GAP IN THE INTESTINE IS CLOSED TRANSVERSELY BY A TWO- OR THREE-TIER SUTURE.

## CHAPTER IV

### ACUTE SUPPURATIVE PAROTITIS

#### (Acute Septic Sial-Adenitis)

ACUTE inflammation of the parotid gland is a rare post-operative complication, and is seldom seen nowadays since strict oral hygiene has become an accepted routine procedure and starvation is no longer enforced after abdominal operations.

Acute septic parotitis is due to an ascending infection of Stensen's duct by the staphylococcus and other organisms. Whether the infection travels up to the gland through the interior of Stensen's duct or alongside via the lymphatics is a matter of purely academic interest, but the former is thought to be the more probable route.

The following factors predispose to the infection :

(1) Extreme dryness of the mouth. This is well seen in the starvation enforced during the first 24-48 hours after severe hæmatemesis, in cases of acute dilatation of the stomach where fluid nourishment by mouth must be withheld, or where the patient has been given large doses of atropine.

(2) Frank oral sepsis.

(3) Debilitating conditions, e.g. advanced cases of malignant disease.

(4) Acute suppurative intra-abdominal lesions.

(5) Herpes labialis or facialis associated with severe sepsis.

(6) Severe toxæmia due to acute infectious disease, e.g. typhoid fever.

The infection more often affects one side only, but in about 20 per cent of cases both sides and even the submaxillary glands may become involved. The inflammation is usually first noticed from 3-10 days after operation. It generally starts in one portion of the gland, as



a rule the cervical prolongation, but after a day or two it rapidly spreads until the whole gland is affected. When the inflammation is at its height the condition bears some resemblance to a case of mumps. The parotid is enlarged, the overlying skin is drawn tight, and the normal folds in this region are obliterated. The swelling is hard, elastic, and tender, but fluctuation, even in an advanced case, is difficult or impossible to elicit. There may be a brawny induration, and pitting on pressure in the area around may sometimes be produced.

On compressing the gland thick creamy pus can sometimes be expelled into the mouth through Stensen's duct. The temperature and pulse are raised, and the patient is gravely ill.

The prognosis is always very serious, and the mortality-rate is high, not only on account of the toxæmia produced by the inflammation of the gland but from the primary cause of illness. A certain number of cases resolve, especially if energetic treatment is instituted early, but suppuration more often ensues.

In neglected cases pus may burst through the thick parotid sheath and rupture into the external auditory meatus, the mouth, the pharynx, or even as far afield as the frontal region on the affected side.

#### TREATMENT

(1) Oral hygiene. Frequent mouth-washes with peroxide of hydrogen, listerine, carholie lotion, or a saturated solution of thymol to which is added a little glycerine, are prescribed.

(2) In early cases the gland may be gently massaged with hot sponges and the pus forced through Stensen's duct. This procedure should be carried out at frequent intervals each day.

(3) Hot fomentations or applications of antiphlogistine to the affected side.

(4) Sialogogues, such as chewing gum, citric acid, and acid drops are useful in promoting a flow of saliva and thus diminishing the infection. Frequent sips of orange or lime juice are also beneficial.

(5) Incision. It is difficult to be sure of the right moment at which to operate on these cases, but it may be taken as a general rule that if the inflammation has been present for four days and shows no signs of subsiding an incision through the parotid fascia into the substance of the gland should be undertaken. As has been previously emphasised, it is useless to wait for the sign of fluctuation, as the pus lies deep beneath the parotid fascia. If the swelling increases

rapidly, if the temperature continues high, or if œdema is very marked. operation should not be delayed.

A skin incision, not more than 1 inch long, should be made and should extend through the parotid fascia into the gland in the



Fig. 628.—ACUTE SUPPURATIVE PAROTITIS. THE WHOLE OF THE PAROTID GLAND IS ENLARGED. THE SITE OF THE INCISION FOR OPENING THE GLAND IS INDICATED.

(Reproduced from *Emergency Surgery*, J. Wright and Sons, Ltd., by courtesy of Mr. Hamilton Bailey.)



Fig. 629.—SURFACE MARKINGS OF THE PAROTID GLAND.

A: TRANSVERSE FACIAL ARTERY.

B: STENSON'S DUCT.

C: FACIAL NERVE.

(Reproduced from *Emergency Surgery*, J. Wright and Sons, Ltd., by courtesy of Mr. Hamilton Bailey.)

region of the angle of the jaw (fig. 628). If made above this the duct or the facial nerve is liable to injury (fig. 629). The finger should then be introduced into the gland through this incision, and the pockets of pus be broken down. A small rubber drainage tube is then placed in position. Through this tube frequent irrigations of the wound are performed, hot fomentations being applied in the intervals.

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## CHAPTER V

### POST-OPERATIVE RETENTION OF URINE

POST-OPERATIVE retention of urine is a condition in which, despite normal kidney function, a full bladder and the absence of organic obstruction, the patient is unable to pass water. Retention is said to be present when urination has not occurred after 18-24 hours following operation, or before this time when the patient complains of discomfort and inability to pass urine. Retention may also be diagnosed when the bladder is found to be at least three finger-breadths above the symphysis pubis. Frequency of micturition during the first 24-48 hours after an operation is very suggestive of retention with overflow. In such cases percussion of the suprapubic region will often reveal a distended bladder, and where doubt exists a catheter should be passed to clinch the diagnosis.

Retention occurs in approximately 12-15 per cent of cases operated upon, and 50 per cent of these will require catheterisation. About 15 per cent of all cases that have been catheterised develop cystitis of a mild or severe nature, but it is more prone to occur in those who have been subjected to repeated catheterisation. Such other complications as epididymo-orchitis and ascending infection of the kidneys are sometimes seen.

The incidence of post-operative urinary retention may be considered under the following headings :

(1) *Age.* Retention may occur at any age, but it is very rare before puberty, being commoner in young adults—18-25, and in the aged.

(2) *Sex.* The condition is stated to be slightly commoner in women than it is in men. It should, however, be borne in mind that there are probably more operations performed upon women than upon men, and that the higher incidence in women is therefore relative.

(3) *Type of patient.* Retention is frequent in nervous patients, and is more often seen in private than in ward cases. Claus G. Jordan writes as follows :

"Of the eighty-one cases of retention thirty-seven were private patients and forty-four ward patients. Considering the much larger proportion of ward patients operated upon we have here a definitely higher incidence of retention in private patients. This finding points very significantly toward the type of patient in which we usually are confronted with this problem. These patients can be spotted almost before operation. They are hypersensitive, apprehensive, psychoneurotic individuals with but little self-control. The least pain or discomfort arouses a marked psychic reaction. They are afraid to suffer the pain of urination. When examined the morning after operation these patients will speak to you in a low voice, hesitating even to take a deep breath for fear of pain or rupturing their wound. They have a very low threshold for pain."

(4) *Type of anæsthetic.* The longer and the deeper the anæsthesia the higher will be the incidence of retention. Retention is comparatively common after spinal anæsthetics which may interfere with the bladder reflex, and also after the prolonged administration of ether.

(5) *Proctoclysis and rectal tube.* Retention can often be relieved by discontinuing their use.

(6) *Type of operation.* Over 95 per cent of cases of retention occur after abdominal and pelvic operations, and it will follow in about 5 per cent of cases of operations upon the head, neck, chest, and extremities. As emphasised by Jordan, the opening of the abdomen, the length and depth of anæsthesia required, the type of anæsthetic—ether or spinal, the proximity of the field of operation to the bladder, pelvic colon, or rectum, have a definite bearing upon the incidence of acute retention. The highest incidence is seen after operations upon the stomach (over 30 per cent), and next in order after gynaecological and rectal operations and appendicectomy. It is often seen, too, following operations for strangulated hernia and in elderly male patients suffering from prostatic enlargement.

(7) *The administration of morphia and basal narcotics.* It has been shown that large doses of morphia administered for the relief of post-operative pain often induce spasm of the internal sphincter muscle of the bladder. It has also been thought that the pre-operative

administration of certain drugs of the barbiturate group, e.g. pernocton, avertin, nenbutal, etc., by prolonging the period of hypnosis increase the liability to post-operative retention of urine.

With regard to the *causation* of acute retention we may sum up by stating that there may be several influencing ætiological factors, of which one may predominate.

*Treatment.* Every effort should be made to induce the patient to pass water naturally before resorting to the use of a catheter. The following may be tried in this order :

(1) The patient should sit up in bed and try to pass water in this position.

(2) Hot stupes to the hypogastrium or short intensive applications of radiant heat may be helpful.

(3) Psychic treatment. The suggestion that if the patient cannot pass water naturally the "painful process" of catheterisation will be necessary will often have the desired effect.

(4) Drugs. There is a long list of drugs which have been used in the treatment of retention, but the majority of them, although claimed by some to be specific, have proved of little or no value. Of these :

(a) Pilocarpine,  $\frac{1}{2}$  gr. ; acetyl-choline, 0.5 gm. ; and pituitrin, 1 cc., which have been used with the object of increasing bladder tonus, may be mentioned, but are not recommended.

(b) The following drugs, either singly or in combination, may at times be useful : (i) hexamine, 5-10 grs. in 20 cc. of sterile water, injected intravenously ; hexamine, 15 grs. by mouth, the dose being repeated in 20 minutes if necessary ; or uritone, one ampoule injected intravenously, the dose being repeated  $\frac{1}{2}$  hour later if required ; (ii) the insertion of a belladonna suppository, t.d.s. ; (iii) the following mixture : piperazin, 5-10 grs. ; pot. cit., 10 grs. ; inf. huchu, ad 1 oz. sig : 1 oz. six-hourly ; (iv) pot. acetate,  $\frac{1}{2}$ -1 dr., dissolved in a little milk and given two-hourly for three doses. In addition to being a powerful diuretic, pot. acetate is a parasympathetic stimulant and its exhibition is therefore rational in cases of acute retention.

(5) The lower half of the abdomen should be very firmly bandaged to increase the intra-abdominal and intra-cystic pressure and thus aid the act of micturition.

(6) If these measures are not effective a catheter must be passed. This must be carried out with the strictest aseptic and antiseptic precautions. In elderly male patients the possibility of enlarged prostate should not be overlooked, and it is perhaps wiser and safer in these cases to introduce a catheter (a small de Pezzer for preference) suprapubically under a local anæsthetic, instead of repeatedly using a urethral catheter.

It should be a rule that the total amount of urine passed during each 24 hours after any abdominal operation should be recorded on a chart until the patient is well on the way to recovery. During the first few post-operative days frequent examinations of the urine should also be made to detect any abnormal constituents.

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## CHAPTER VI

### BEDSORE

BEDSORE is, in the majority of cases, due to pressure necrosis. The following may be predisposing factors :

- (1) A bad mattress ; rucking up of the bedclothes ; crumbs and such-like in the bed.
- (2) A moist skin subjected to constant pressure.
- (3) Cachexia and wasting illnesses, necessitating a long confinement to bed.
- (4) Pyæmia ; chronic toxæmia from any cause.
- (5) Spinal cord lesions.

This complication is not often seen after abdominal operations, its incidence being confined chiefly to cases associated with prolonged suppuration and toxæmia, and inoperable cancer of the viscera.

Bedsore develop over certain bony prominences which have a poor blood supply, such as the sacrum, the crest of the ilium, the great trochanter, the scapula, the vertebral column, and the heel, which are subjected to maximum pressure while the patient is lying in bed. In an early case the skin over one of these bony prominences becomes red, inflamed, or blistered. If neglected it will become gangrenous and ulcerate. In a severe case large black sloughs will form which, on separating, will expose the underlying structures, and exude thin watery serum and pus. The base and edges of these wounds are pale pink and callous, and show no signs of the production of healthy granulation tissue. The edges of the skin, too, may be undermined for a considerable distance, and the thin, death-like skin seems incapable of obliterating the space with plastic lymph.

*Treatment.* Bedsore are very resistant to treatment on account of the patient's debilitated condition, his poor recuperative powers,

the nature of the disease from which he is suffering, the difficulties of nursing, and the obstacles to be overcome in the avoidance of pressure upon the affected part. The most important single factor in treatment is an efficient nurse. She can do much to prevent its occurrence, or, if present, to limit its extension and aid in its rapid healing by unremitting attention.

It is essential in the first instance to relieve pressure in the area involved, and to increase its blood supply. It will be necessary, therefore, to change the patient's position from time to time, and to make judicious use of an air cushion. The wound itself should be kept clean, dry, and frequently dressed. The following applications may be useful: Absolute alcohol, tannin alcohol, formalin 1 per cent, Friar's balsam, collodion, mastisol, or Whitehead's varnish. The parts may be kept dry by sprinkling or powdering with Fuller's earth, talcum powder, or bismuth subgallate powder.

If sloughs are present they may be removed with a knife or scissors, or if this is deemed inadvisable their separation may be hastened by frequent applications of hydrogen peroxide or eusol. I have found strapping the affected parts firmly and evenly with elastoplast to be of the greatest advantage. If the ulcerated areas are large and healing is sluggish, skin grafting by Thiersch's method is indicated and will often prove successful.



## CHAPTER VII

### POST-OPERATIVE THIRST

THIS is a troublesome *symptom* rather than a complication following operation. It is most marked after abdominal operations, after the administration of a volatile anæsthetic, where a pre-operative starvation regime has been severely enforced, where vomiting has been frequent, purgation extreme, sweating excessive, hæmorrhage free or uncontrolled during the operation, and where there has been much exposure and chilling of the viscera. It is unquenchable in acute dilatation of the stomach, present in obstructive lesions of the stomach and duodenum, in paralytic ileus and acute peritonitis, and in many other morbid conditions.

The accepted methods for relieving thirst may be briefly outlined as follows :

(1) Frequent small sips of water or fresh fruit juice by the mouth. Tinned grape-fruit and pineapple juice form agreeable variations as they are pleasant to the taste and contain a large amount of assimilable sugar. A constant flow of saliva should be encouraged by making the patient suck acid drops and by frequent mouth-washes. A parched dry mouth is not only unpleasant to the patient but is a positive danger, and is certainly a common predisposing factor to parotitis. Large quantities of fluid given by the mouth before post-anæsthetic sickness has ceased will induce further vomiting and greatly add to the distress of the patient. As soon as the vomiting has ceased the patient should be encouraged to drink large quantities of fluids which may be varied according to his tastes. If vomiting is persistent or intractable, fluids by the mouth should be withheld for the time being and other methods of restoring fluid loss and of quenching the thirst should be adopted.

(2) Intravenous salines with or without glucose. Where there is concomitant alkalosis, hypertonic saline is administered ; in cases of acidosis, liberal amounts of glucose are added to the infusions. When

post operative shock is present, 6 per cent gum acacia in saline will be found efficacious. These are best given by the continuous drip method.

(3) Subcutaneous salines. Although frequently employed as a post operative measure in combating shock, in supplying additional circulating fluid, and for the relief of thirst, and although beneficial in certain cases, these are not to be recommended as an indiscriminate routine since they may be painful and distressing to the patient.

(4) Proctoclysis. It is surprising the large amount of fluid which will be absorbed by the gut if it is administered by the Murphy drip method. When the fluid is introduced at a slow, steady rate several pints can be given without discomfort during the first 24 hours, and this treatment should be continued as long as possible, or until the fluid is no longer retained.

The fluids used for proctoclysis are normal saline, normal saline with 5 per cent glucose, tap water, or distilled water. Normal saline, with or without glucose, is slightly irritant to the gut and may produce a mild degree of proctitis. This irritant action of the saline solution may militate against its retention by throwing the gut into spasm thus causing ejection of the fluid. As an alternative to the drip method, 6 oz. of saline solution may be slowly injected into the rectum every 4-6 hours until the fluid is no longer returned.

## CHAPTER VIII

### POST-OPERATIVE PAIN

THERE are many factors concerned in the production of post-operative pain, and these may be briefly considered under the following headings :

(1) *The type of operation.* Certain major abdominal operations upon the gall-bladder and biliary passages, and operations necessitating much exposure and handling of the intestines such as partial gastrectomy for cancer of the stomach, are necessarily followed by more pain than such an operation as appendicectomy for chronic appendicitis or the removal of an ovarian cyst which is free from surrounding adhesions.

Upper abdominal operations are followed by more post-operative pain than pelvic operations. Pain, again, is more severe after a protracted operation, and as a general rule the longer the time taken over the operation the greater the pain.

The length of the incision and the method of closure may be important factors, as will also be undue traction on the mesentery and trauma of the abdominal muscles and nerves. The injudicious use of abdominal packs, forceful retraction, unnecessary injury to muscles by the application of tissue forceps, and the insertion of sutures in such a manner as to produce undue tension or strangulation of the tissues are also contributory causes of pain. Such errors in technique as imperfect hæmostasis, the introduction of strong antiseptics into the abdominal wound, or mass ligation of tissues will increase the pain in the abdominal wound.

(2) *The position of the patient during and after operation.* Certain operating tables are very unsuitable, no provision being made for the comfort of the patient. The position of the patient on the operating table for abdominal operations should always be very carefully scrutinised by the surgeon. Such faults as hyperextension of the head, the tucking of the hands under the buttocks, the arching of the back, and the full extension of the lower limbs, are all productive of subsequent cramp in the muscles, backache, and transient neuritic pains. The

knees, neck and spine should be very slightly flexed to relieve any strain on or tenseness of the abdominal muscles, while the arms should rest comfortably by the side of the body upon the operating table, and should be kept in this position by means of a binder or special supports. In certain upper abdominal procedures, however, such as operations upon the gall-bladder and biliary passages, total gastrectomy, and splenectomy, it is advantageous to throw the epigastric region forwards by means of a tripartite rubber pillow or by raising the bridge of the operating table to the required height. This enforced arching of the back to facilitate the approach to certain viscera in the upper abdomen may be associated with some degree of post-operative backache. Keeping the patient in one position, which may be a strained one, after his return to bed, and prohibiting movement, will also tend to produce cramp and muscular pain.

The patient should be nursed in a suitable surgical bed which can be adapted to the Fowler or other required position by means of mechanical devices. Unless there are contra-indications the patient should be encouraged to use his arms, move his legs, and alter his position in bed as he may feel inclined.

(3) *Abdominal drains.* Rubber drainage-tubes, and particularly gauze packs, are productive of a great deal of abdominal pain. The management of these tubes and packs, their removal, and their replacement when necessary, calls for much skill and practice.

(4) *Dressings.* Tight bandaging is a frequent cause of post-operative pain, and it should be a routine practice to adjust the bandages shortly after the patient is fully round from the anæsthetic. Dressings caked hard with blood or sodden with irritating discharges are a further cause of pain. If there has been much oozing of blood from the wound, or if there has been much discharge from a drainage-tube, the dressings should be changed at frequent intervals.

(5) *Infection of the wound.* (See page 1215.)

(6) *The type of patient.* Neurotic or obese patients, and those particularly sensitive to pain, suffer more than those who are thin or phlegmatic.

(7) *The anæsthetic factor.* As a rule, though by no means invariably, pain is less pronounced after the administration of a spinal or local than after an inhalation anæsthetic.

A consideration of the above will show that the mitigation of post-operative pain is largely a question of prophylaxis. It should, however, be assumed that during the first 24-36 hours following abdominal operations pain will be such as to call for relief by the administration of certain drugs. The rectal administration of aspirin, 30 grs., and pot. brom., 40 grs. in 6 oz. of saline as soon as the patient has returned to bed, and the subcutaneous injection of morphia,  $\frac{1}{4}$  gr., when the patient is fully round from the anæsthetic are often required to produce rest and relief from pain during the first 12 post-operative hours. After this period a further injection of morphia may be necessary. Short applications of radiant heat to the abdomen are particularly soothing, and are helpful in the relief of abdominal pain and discomfort.

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## CHAPTER IX

### POST-OPERATIVE PHLEBITIS

by

A. DICKSON WRIGHT

*This is a complication of great seriousness following operations, and one fraught with great danger to the patient because of the risk of pulmonary embolism. The condition occurs exclusively in the legs, with the exception of a few cases in the arms following intravenous injections during or after the operation. The hypertonic and the constant intravenous drip are the injections most likely to produce phlebitis, and as phlebitis in the arms is much less serious than in the legs it is wise to give these injections in the arm veins.*

Post-operative phlebitis attacks more frequently :

- (1) Patients over 40.
- (2) The obese.
- (3) The septic case.
- (4) Cases in which there is a history of previous phlebitis.
- (5) Patients suffering from varicose veins.
- (6) Patients who have certain operations :
  - (a) Splenectomy and Cæsarean section.
  - (b) Cholecystectomy.
  - (c) Prostatectomy.
  - (d) Appendicectomy.
  - (e) Reduction or plating of a fractured femur.
  - (f) Hernia operation, especially femoral hernia.

It is important, therefore, to be prepared for the complication following operation in these cases. The liability to phlebitis may make one decide not to operate in certain cases, such, for instance, as gall-stones in a stout woman of 60 with an old white leg and numerous varicose veins which have been the seat of recurrent attacks of phlebitis; such

a case might be better left alone provided she does not suffer too much from the gall-stones.

Femoral thrombosis when it occurs is a catastrophe from which the patient never entirely recovers. Having escaped death from pulmonary embolism the patient after 6 weeks in bed gets up with a heavy painful swollen limb which never really gets well, and after an interval of from 6 months to 12 years the ankle begins to become eczematous and to ulcerate. These ulcers are intractable and tend to recur, and the patient is also greatly troubled by recurrent attacks of phlebitis in the collateral veins and in the other leg.

*Prevention* is a very difficult matter, and one in which one comes into conflict with the nursing profession. Fowler's position is a great predisposing factor in the production of phlebitis because it causes venous stagnation in the legs, and it is possible that this position is used much too often after abdominal operations, and that the patient should at least be allowed to lie down to sleep at night. The knee pillow or "donkey" presses upon the popliteal veins leading to posterior tibial thrombosis, often the starting-point of a femoral thrombosis. The difficulties of abolishing the knee pillow I have found almost insuperable. The ring cushion also causes venous stagnation in the gluteal region, and it should not be used continuously. The necessity for continuous fidgeting on the part of the patient is a thing that nurses cannot understand, as is the necessity for sleeping in a different position at night, and turning on to the side as soon as possible after operation.

Special exercises have been devised to encourage the patient to use his legs after operation. At first, "tensings and twitchings" of glutei, quadriceps, ham-strings, and tibial muscles are advised. A special adjustable foot-board covered with sponge rubber provides the foot with kneading and grasping exercises. After the second day, a pulley at the top of the bed is used, and with one end of the cord in the band and the other round the foot simple exercises can be carried out.

All these prophylactic methods run so contrary to tradition that it is hard to get them adopted. In some German hospitals a text adorns every wall in the surgical wards: "Breathe deep, spit hard, wave the legs."

The administration of thyroid extract and a high calorific diet have been suggested to stimulate metabolism and make the patient definitely hyperthyroid and fidgety. Good results have been claimed for this method by some, while others have decried it. Unless definite hyperthyroidism is produced the method might as well not be used.

In cases with varicose veins a prophylactic method of value is to

bandage the legs firmly with elastoplast before the operation, in order to obliterate the varices ; this bandage is worn until the patient gets up and about again.

#### TREATMENT

A complete femoral thrombosis can only be treated with the patient flat on his back. The bottom of the bed is not raised, and pain is controlled with the various sedatives according to its severity. Local applications of ichthyol, belladonna, etc., cannot possibly affect the thrombosed vein lying so far from the skin, and one should have the strength of mind not to recommend them. After two to three weeks the tension in the leg begins to fall, and the elastoplast bandages should be used to hasten the absorption of the cedema. When the affected leg is reduced to the same size as the other the patient may get up with the bandage still on. Support is maintained by elastoplast for three months, and then an elastic stocking reaching to the thigh is worn for six months—after this, elastic stockings to the knee must be worn for life.

A posterior tibial thrombosis shows itself by pain below the fulness of the calf, and should be treated by an elastoplast bandage from the toes to just above the knee, in the hope that extension of the thrombosis may be prevented.

Internal saphenous thrombosis or phlebitis in pre-existing varicose veins is treated with firm elastoplast bandaging with a sorbo pad above the clot, and the patient is got up as soon as possible to prevent deep trouble starting.



PART III

RECTUM AND ANUS

by  
W. ERNEST MILES

CHAPTER I  
Hæmorrhoids

CHAPTER II  
Anal Fissure

CHAPTER III  
Pruritus Ani

CHAPTER IV  
Prolapse of the Rectum

CHAPTER V  
Fistula in Ano

CHAPTER VI  
Stricture of the Anus and of the Rectum

CHAPTER VII  
Benign Tumours of the Anus and Rectum

CHAPTER VIII  
Malignant Growths of the Anus and Rectum

## RECTUM AND ANUS

### CHAPTER I

#### HÆMORRHOIDS

##### A. EXTERNAL PILES

THE following three distinct conditions are usually described as external piles or hæmorrhoids. They are : (a) a circumscribed blood extravasation at the anal margin ; (b) a redundant fold of peri-anal skin ; and (c) dilated peri-anal veins. Combined with them there is always present a pronounced pecten band and hypertrophy of the external sphincter, together with consequent narrowing of the anal aperture. Internal piles may be associated with any of these three conditions. Constipation always accompanies the first two varieties, and constipation or diarrhoea is invariably met with in the third.

(a) *Circumscribed Blood Extravasation at the Anal Margin.* This condition is usually referred to under the name of *venous* or *thrombotic pile*, and is supposed to be due to thrombosis taking place in a previously dilated or varicose vein at the anal margin. With this opinion I do not concur, because the blood clot is always outside the vein. Hence, the condition is due to a rupture taking place in one of the peri-anal veins, which is followed by a limited extravasation of blood into the surrounding connective tissue. If a case of this kind is seen soon after the swelling has made its appearance, that is before coagulation of the extravasated blood has taken place, the tumour is found to be tense and soft, but if some days have elapsed, the sensation imparted to the examining finger is that of a pea or bean beneath the skin. At this later stage the blood has coagulated, and the surrounding connective tissue is becoming condensed, by round cell exudation, into a true capsule. If an incision be made into the tumour at this stage, the clot can be enucleated, while the capsule is left behind, or the capsule

together with its contained clot can also be removed as a distinct tumour. The longer the duration of the extravasation, the more distinct will be the capsule, and no doubt this circumstance has led to the belief that the capsule is the wall of a dilated vein.

*Ætiology.* The rupture of a peri-anal vein, resulting in blood extravasation, is most commonly caused by violent straining during defæcation, by over-exertion in lifting heavy weights, or by a severe fit of coughing.

*Symptomatology.* During straining at stool, while lifting a heavy weight, or when coughing violently, there is a sensation that something has given way in the anal region, and a painful swelling is found to have suddenly appeared at the margin of the anus. The pain in the anus persists for some time afterwards. The swelling is also tender to the touch, and the patient is unable to sit in comfort. Defæcation is rendered exceedingly painful, and difficulty is experienced in emptying the bowel of its contents owing to the induced spasm of the sphincters and levatores ani. These painful phenomena usually begin to abate after two or three days, and disappear entirely by the end of a week, unless further straining has caused the rupture of a vein at some other part of the anal circumference.

*Pathological Anatomy.* A swelling, varying in size from one-sixteenth of an inch to one inch in diameter, is found situated at the anal margin, its inner border extending into the anal canal, and its outer margin extending beneath the skin covering the external sphincter. In shape the swelling is either circular or oval. When the skin over it is tense the colour is bluish purple, but when the skin is loose there may be no discoloration at all. When seen immediately after its appearance, the swelling is tense and cystic in character, but after the lapse of some days it feels like a pea or bean beneath the skin. In the latter condition it is freely movable beneath the overlying skin and upon the subjacent tissues, and can be easily lifted up from the latter. When the clot is the seat of inflammation and impending suppuration, it becomes adherent to surrounding structures. In many cases of extravasated blood clot, the external sphincter will be found to be hypertrophied and, in the majority of them, the rectum is loaded with hardened fæces.

*Natural Termination.* If these local extravasations are not irritated, and constipation is prevented, the blood will be completely absorbed

in four or five weeks, and leave no trace of ever having existed. When the primary extravasation has been so extensive that the skin covering it is tightly stretched, ulceration occurs on the surface and the blood clot is spontaneously extruded, leaving a cavity which will often slowly contract and heal, but occasionally a blind external subcutaneous fistula will result. When subjected to irritation, or when occurring in debilitated subjects, infection of the blood clot by pyogenic organisms is apt to take place, and suppuration to ensue, resulting in a marginal abscess.

*Treatment.* The majority of these blood extravasations can be completely cured without any operative treatment, but, in whatever way the cure is effected, succeeding extravasations may take place should the cause again become effective. The probable reason for the extravasation should therefore be clearly explained to the patient, in order that he may avoid the risk of its recurrence. All cases, in which the skin over the extravasated blood is not tense or in which inflammation has not already supervened, may be successfully treated in the following manner. An aperient, of sufficient strength to ensure that the bowels will be relieved at least once or twice daily, should be given every night at bedtime. A lotion consisting of

R/

Liq. plumbi subacetatis	3 ss.
Spiritus vini rectificati	3 i.
Glycerini	3 ss.
Aquæ rosæ	3 x.

should be kept constantly applied to the anal region by means of a piece of lint or a pad of cotton wool, squeezed nearly dry and retained in position by a T-bandage. This application should be renewed every two hours for the first three days, and subsequently every four hours until absorption of the blood clot has taken place. Within forty-eight hours after the commencement of this treatment, all pain and discomfort will usually have disappeared, and the swelling will be found to be much reduced in size. The patient need not be confined either to a bed or a couch, though he should, as much as possible, avoid sitting for the first few days. Under this treatment the extravasated blood will be completely absorbed in from three to five weeks.

Another way in which these cases may be treated is by making an incision into the tumour and turning out the coagulum, under the influence of a local anæsthetic. This is most conveniently done by

introducing the left forefinger into the rectum and rendering the seat of the extravasation prominent by compressing it between the finger and the thumb outside, in the same way as when operating for fissure. While the part is held in this position, the incision should be made through the centre of the swelling, commencing at its outer margin along the line radiating from the anus. When the coagulum has been pressed out, the cavity should be sponged with some antiseptic solution and plugged with a small piece of cotton wool. This plug should be allowed to come away of its own accord, and should not be renewed. An external dressing should be kept applied until the wound has healed. The objection to this operation is, that the part is painful for a week or ten days and healing of the wound is not completed under a fortnight.

When operative interference has been decided upon, and when there is no suppuration, the best method is to administer a general anæsthetic, and to excise an elliptical piece of the skin over the most prominent part of the swelling.

*Operative Technique.* The piece of skin to be removed should be about one quarter of an inch wide, and three-fourths of the length of the tumour. The excision is effected by lifting up the skin from the surface of the tumour with a pair of dressing forceps, and removing the required piece with curved scissors. Through the opening thus made the blood clot, in its capsule, can be readily protruded and completely removed with the scissors. Bleeding can be arrested by pressure, and when it has ceased (this may take twenty or thirty minutes) the edges of the wound should be carefully adjusted, without sutures, and kept in position by an external dressing.

Healing will, almost invariably, be completed within forty-eight hours after the operation. The only objection to this method is that a general anæsthetic is required. Occasionally it is not possible to raise the piece of skin required to be removed, in which case it should be mapped out and dissected off. In doing this, there is some risk of puncturing the cyst wall, which will increase the difficulty of enucleation.

In those cases in which inflammatory action has begun, the tumour should be freely laid open by either a T-shaped or crucial incision.

(b) *Redundant Folds of Peri-anal Skin.* These cutaneous out-growths are exaggerated peri-anal rugæ. They vary both in size and shape, being sometimes distinctly pedunculated, but generally sessile. In length these folds measure, as a rule, from one quarter to three-quarters of an inch, though they may be longer. There is a certain

quantity of connective tissue contained within the fold, but usually there are no distinctly dilated veins. The connective tissue is frequently the seat of inflammation, which, when it subsides, leaves more or less induration. In this way the soft, pliant character of the fold is more or less lost, so that it ultimately stands out prominently from the surface. In the intervals between neighbouring folds, follicular secretion collects, causing excoriation and dermatitis, which may spread to the whole of the peri-anal area. The lodgment of faecal matter between the folds also increases the irritation.

*Ætiology.* The most frequent primary cause of this redundancy of peri-anal skin is constipation. The over-stretching of the skin round the anal aperture, during the passage of a large and hard mass of faeces, causes the normal rugæ to be slightly torn, and thus opens up the path for septic infection. As a result of this, the redundant skin becomes inflamed and œdematous. When the inflammation has subsided, the rugæ do not contract to their former size and, in addition, lose some of their natural elasticity. This sequence of events is sometimes repeated with each subsequent constipated action of the bowels, and gradually leads to a marked increase in the size of one or more of the rugæ. Consequently, the crevices between adjacent ones are deeper and permit of an accumulation of follicular secretion therein. The secretion decomposes and causes additional irritation. In all cases of constipation flatus is frequently voided and, with it, often a small quantity of rectal mucus which lodges between and around the folds and irritates them. Faeculent matter also readily lodges between them when they have attained a certain size, and thus becomes a source of irritation. Later on, the more or less constant irritation in the anal region from the above causes makes the patient frequently rub the part in order to allay the itching and discomfort. The rubbing still further congests the region and tends to increase the size of the folds of skin.

*Symptomatology.* If not the seat of inflammation, the hypertrophied rugæ, even when large, cause very little inconvenience beyond the difficulty experienced in cleansing the anal region after defæcation. At times there may be slight itching, especially after the passage of flatus. When inflamed the constant smarting, irritation, and swelling render sitting uncomfortable and walking painful. When the inflammation terminates in suppuration, the symptoms become those of a subcutaneous abscess.

*Pathological Anatomy.* In the earlier stages nothing more than an enlargement of one or more of the normal rugæ will be observed. On separating adjacent folds retained secretion or fecal matter will be found between them. When this is wiped away the surface under it will be found to be red, glistening, and perhaps excoriated. Sometimes separating the rugæ may cause tearing of the skin between them. In the later stages the rugæ will be found to be much larger and, when inflamed, cedematous and glistening. The surrounding skin is thickened, and sometimes inflamed and excoriated in places, especially in the middle line posteriorly. Occasionally the surrounding skin is blanched, much of its natural pigmentation having quite disappeared. This blanching may also extend into the anal canal. In any stage of the existence of these folds, it will be frequently noticed that the external sphincter is hypertrophied; that there is a pronounced pecten band; that both sphincters and the levatores ani are in a state of irritability; and that the rectum is nearly always more or less distended with firm feces.

*Treatment.* Constipation should receive careful attention. When there are not more than two or three enlarged rugæ, they should be removed by scissors, the point of section being about one-eighth of an inch above the bottom of the sulcus. This will allow the sides of the wound to come together without causing contraction of the perianal skin. When the folds are numerous, only two or three of the more prominent should be removed at first, lest contraction of the anal aperture should result. If, after these wounds have healed, there is a tendency to contraction, additional ones may be removed, if thought necessary. In all cases, if the external sphincter be much hypertrophied or thickened, the pecten band should be carefully and completely divided.

Should an operation not be performed, the following method of treatment will be found useful. After thoroughly cleansing the region with warm water (without soap), the skin should be dried and then carefully wiped over with cotton wool which has been dipped in olive oil. By the use of the oil all the adherent secretion, as well as any ointment that may have been previously applied, can be completely removed. As soon as the cleansing has been completed the following ointment should be smeared over the surface.

R/		
Zinci oxidi	3	ii
Linimenti camphoræ	3	iii
Vasellini	3	ii

This ointment should be used as a nocturnal application, and during the day the following powder should be applied :

R/		
Zinci oxidi	3	iii
Pulv. camphoræ	3	ii
Pulv. amyli	ad	3 ii

In some instances the camphor is objectionable to the patient, in which case a corresponding quantity of starch should be added in place of it.

(c) *Dilated Peri-anal Veins.* The superficial veins of the anal margin, forming the communication between the superior and inferior hæmorrhoidal venous plexuses, occasionally become dilated, and involve the whole circumference of the anus. During a straining effort, when the veins are distended with blood, there will be observed a subcutaneous, compressible, and more or less uniform swelling surrounding the anal orifice, the skin being stretched but not œdematous. After the straining has ceased for a little while, the swelling almost completely subsides, leaving the skin round the anus loose and redundant, but not thrown into distinct folds. There is no induration or excoriation of the redundant skin.

*Ætiology.* The dilatation of the veins is due to straining during defæcation, caused by hypertrophy and irritability of the sphincters. Repeated straining thus rendered necessary causes hypertrophy of the veins, while the spasmodic action of the sphincters impedes venous return, with the result that the walls of the veins become over-stretched and remain permanently dilated.

*Symptomatology.* The mere dilatation of the peri-anal veins gives rise to little or no inconvenience, beyond the swelling and a feeling of fulness at the anus. Consequently the patient seldom seeks relief solely for this condition. His chief complaint is the constant difficulty experienced in defæcation. Feeling that his bowels should be relieved, he cannot, even by persistent straining, void the whole of the contents of the rectum, and is sometimes obliged to make several attempts before obtaining complete relief. Often he is driven to aid expulsion by introducing a finger into the rectum. Much soreness and discomfort are generally complained of after the bowels have acted. There is also a dull aching sensation, together with a feeling of fulness in the anus after an unsuccessful effort has been made to obtain an evacuation.



*Pathological Anatomy.* When the patient is not straining but resting quietly on his side, the anal skin is observed to be redundant and the anal orifice is firmly closed.

When a straining effort is made, the patient being on his side, the anus becomes prominent and is forced down to the level of, and in some instances beyond, the plane of the ischial tuberosities; and then the anal skin becomes cedematous, creating a distinct fulness round the whole circumference of the anal margin. The anal orifice is now seen to be firmly contracted, and to form the apex of a cone-like prominence. During the straining effort, a small quantity of flatus and rectal mucus may escape, but, on account of the spasmodic action of the sphincters, solid feces are seldom passed.

When the finger is introduced into the rectum the sphincters grip it tightly and impede its introduction. When the finger is fully inserted, the extent of the hypertrophy and thickening of the sphincters and the presence of the pecten band can be easily appreciated. The rectum will be found to be dilated, and often filled with a large quantity of firm feces as well as flatus, constituting the condition known as ballooning of the rectum. In women, the chronic distension of the rectum caused by the spasmodic action of the sphincters sometimes produces a rectocele, which should be looked for at the time of the examination.

It must be borne in mind that external piles are often complicated by the presence of internal piles, which should in all instances be carefully looked for. Even though internal piles may have attained large size and have existed for a considerable time, they generally afford no external indication of their presence, because protrusion seldom occurs on account of the spasmodic contraction of the sphincters and consequent narrowing of the anal orifice. Hæmorrhage, occurring during the period of difficulty in defecation, is an almost certain sign that internal piles are developing, and should be regarded as an additional indication of their probable presence. The recognition of the presence of internal piles in these cases is very important, because, after division of the external sphincter, the undue narrowing of the anal canal will have been remedied and the obstacle to their prolapse removed. Consequently the patient, though relieved of the symptoms for which he sought relief, will soon experience discomfort from the protrusion of the internal piles, and will have to undergo a second operation, which could have been easily performed as a part of the first.

*Treatment.* The only method of treatment that will permanently cure this condition is an operation. When, from any cause, an operation

cannot be performed, an injection of one ounce of olive oil at bedtime (to be retained all night) will greatly facilitate defæcation. The motions also should be kept soft by gentle aperients and by attention to diet.

*Operative Technique.* Under the influence of a general anæsthetic, the pecten band and the external sphincter should be completely divided in the right posterior quadrant. The effect of the division of the external sphincter is to suspend its action for the time being, and to bring about absorption of the induration between the muscular fibres. Should an internal pile be present, it should be removed by ligature. Finally, the anterior margin of the wound, made by dividing the external sphincter, should be treated in the following way. An incision, involving nothing but the skin, should be commenced three-eighths of an inch in front of the wound at the muco-cutaneous junction. From this point the incision should be carried outwards and backwards in a slightly curved direction, so as to end in the outer extremity of the incision through the external sphincter. The piece of skin thus mapped out, together with the corresponding piece of mucous membrane above it and the dilated veins under it, should be included in a ligature and tightly strangulated. It is not absolutely necessary to remove this margin of the wound, but as it will, if left, become cedematous and fall into the wound, healing will be considerably delayed, and therefore time will be gained by its removal. The wound should be packed, to keep the edges apart, so that healing by granulation may be ensured.

When cicatrisation has taken place, it will be found that formed fæces can be easily passed without undue straining, and that the circum-anal veins no longer become distended. If, after an interval of two or three months, it be found that the skin of the anal margin is inconveniently redundant, portions of it, together with the subjacent veins, should be removed.

*After-treatment.* The details of the after-treatment are the same as those for internal piles (see page 1286).

## B. HÆMORRHOIDS OR INTERNAL PILES

A hæmorrhoid, or internal pile as it is more commonly called, is a vascular tumour consisting chiefly of tortuous and dilated veins accompanying the branches of the superior hæmorrhoidal artery at their termination in the submucosa of the anal canal immediately

above *Hilton's white line*. The dilated veins form clusters beneath the mucosa and are arranged parallel to one another around the circumference of the upper third of the anal canal and the lower inch or so of the rectum.

*Pathological Anatomy.* Each hæmorrhoid consists of a central artery, a mass of thickened tortuous veins and a quantity of fibrous tissue. The pathological change that has taken place is partly hypertrophic and partly fibrotic. Arteries, capillaries, and veins simultaneously participate in the change. The central artery is one of the terminal branches of the superior hæmorrhoidal artery and is usually greatly increased in size, its magnitude occasionally being as great as that of the radial artery at the wrist. The venous elements are dilated, thickened and tortuous branches of the venous tufts from which the rootlets of the superior hæmorrhoidal vein arise and form the greater part of the bulk of the tumour. The dilatation does not always involve the entire circumference of the vein, as in simple varicosity, more often consisting of saccular bulgings at one or more points. The naked-eye appearance of a dissected hæmorrhoid is that of a number of saccules communicating with veins by means of minute apertures. When the blood in a saccule coagulates, the clot becomes organised and is transformed into fibrous tissue. When a hæmorrhoid has existed for a long time, and especially if it has been the seat of repeated attacks of inflammation, the veins are usually obliterated and the bulk of the tumour consists of fibrous tissue.

*The Number of Hæmorrhoids which may develop.* Since a hæmorrhoidal tumour develops in connection with the veins accompanying the branches of the superior hæmorrhoidal artery, it follows that the number of hæmorrhoids that may develop in a given case is determined by the number of branches which the artery gives off. When every possible hæmorrhoid has been developed, for instance, as may occur in an old-standing case which has not been operated upon, there are usually seven present, four on the right side and three on the left. This is due to the anatomical fact that the branch of the superior hæmorrhoidal artery which is distributed to the right side of the rectum ultimately breaks up into four branches; whereas that distributed to the left side of the rectum breaks up into three branches only.

In about 70 per cent of cases there are only three or four hæmorrhoids present, and these are generally found to exist in different stages

of development. Even in cases of old standing which have not been subjected to operation, the full complement of seven hæmorrhoids is seldom seen, because, at some time or another, one or more of them has sloughed as the result of spontaneous strangulation.

*The Mode of Distribution of the Arterial Supply to the Terminal Portion of the Rectum.* The arterial supply of the lower portion of the rectum and of the greater part of the anal canal is derived from the superior hæmorrhoidal artery which is the terminal branch of the inferior mesenteric. The superior hæmorrhoidal, after emerging from the pelvic mesocolon and while situated upon the posterior surface of the rectum, divides into two main branches, one for distribution to the right side of the rectum and the other for distribution to the left side.

The right superior hæmorrhoidal artery, after having passed through the muscular coat, enters the submucous tissue and immediately divides into two primary branches, an anterior and a posterior primary branch. The *anterior primary* branch courses downwards towards the anal verge in the right anterior quadrant of the rectum, midway between the anterior and right points of the circumference, and finally breaks up into a meshwork of small vessels in the submucosa of the anal canal. This artery does not give off any secondary branch during its course along the rectal wall. When the veins accompanying this branch become tortuous, dilated, and thickened they give rise to the *right anterior primary* hæmorrhoid which always remains discrete and never has any connection with any other hæmorrhoid (fig. 630).

The *posterior primary* branch passes downwards in the right posterior quadrant to terminate in an anal canal meshwork. During its course it gives off two secondary branches, an anterior and a posterior. The former proceeds towards the right point of the anal circumference along the line separating the right anterior from the right posterior quadrant. The latter passes downwards in the middle line posteriorly. The veins accompanying the posterior primary branch give rise to the right posterior primary hæmorrhoid. Those accompanying the secondary branches give rise to the right and to the posterior *secondary* hæmorrhoid respectively (fig. 631). The right posterior primary hæmorrhoid, therefore, has two satellites connected with it, viz. the right and the posterior *secondary* hæmorrhoids.

The left superior hæmorrhoidal artery, after perforating the muscular coat, passes downwards towards the anal margin along the line separating the left anterior from the left posterior quadrant, the accompanying veins giving rise to the *left primary* hæmorrhoid (fig. 630). This artery

gives off two branches, an anterior and a posterior. The veins accompanying the former give rise to the *left anterior secondary* hæmorrhoid, and those accompanying the latter form the *left posterior secondary* hæmorrhoid (fig. 631). The left primary hæmorrhoid may, therefore, have two satellites, namely, the left anterior secondary and the left posterior secondary hæmorrhoids. The artery supplying the left anterior secondary hæmorrhoid very rarely gives off a small branch which is distributed to the middle line anteriorly. The veins accompanying it then give rise to a small anterior secondary hæmorrhoid.

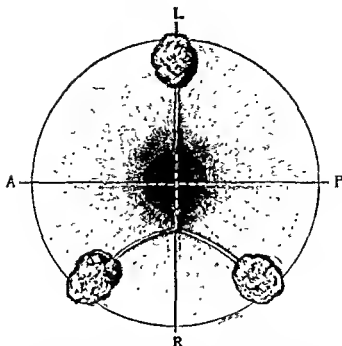
The veins arise in a minute plexus located in the submucosa of that position of the anal canal, which is bounded below by Hilton's white line, and above by the free edges of the valves of Morgagni (pectinate line). This zone is known as the pecten, so called by Stroud, an American anatomist, who imagined that it resembled a comb in appearance.

When pathological changes take place in the hæmorrhoidal vessels, rendering them tortuous, dilated and thickened, those first affected are the larger or main branches. The smaller or secondary branches become involved later. Consequently the first vessels to undergo pathological change are those distributed (a) to the right anterior quadrant; (b) to the right posterior quadrant; and (c) to the left point in the anal circumference. Hence, hæmorrhoids resulting from changes in the above-mentioned vessels may be spoken of as *primary hæmorrhoids*.

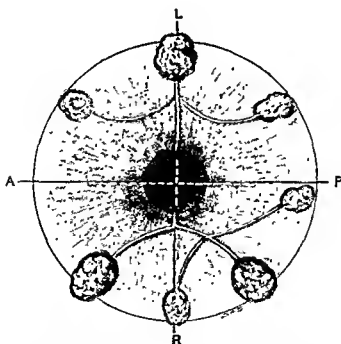
Similarly hæmorrhoids developing in connection with the secondary branches, namely, those distributed (a) to the right point, (b) to the middle line posteriorly, (c) to the left posterior quadrant, and (d) to the left anterior quadrant, may be considered to be *secondary hæmorrhoids*.

*The Position of Internal Hæmorrhoids in Relation to the Circumference of the Anal Canal.* Since the anatomical distribution of the various branches of the superior hæmorrhoidal vessels is constant, each branch supplying a definite area of the circumference of the anal canal, it follows that the hæmorrhoids which develop in connection with these vessels preserve an invariable position in relation to the circumference of the anal canal.

The primary hæmorrhoids are three in number, and are always found to occupy the same relative position, namely (1) in the right anterior quadrant; (2) in the right posterior quadrant; and (3) at the left point in the circumference of the anal orifice. Accordingly, these

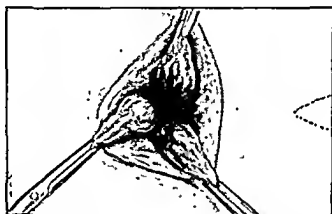


*Fig. 630.*—**DIAGRAMMATIC REPRESENTATION OF THE ARTERIAL BLOOD SUPPLY OF THE THREE PRIMARY INTERNAL PILES, NAMELY THE RIGHT ANTERIOR, THE RIGHT POSTERIOR AND THE LEFT.**  
A anterior, R right, P posterior, and L left, points of the circumference.



*Fig. 631.*—**DIAGRAMMATIC REPRESENTATION OF THE ORIGIN OF THE ARTERIAL BLOOD SUPPLY OF THE SECONDARY INTERNAL PILES, NAMELY, THE RIGHT, THE POSTERIOR, THE LEFT POSTERIOR AND THE LEFT ANTERIOR.**  
A anterior, R right, P posterior, and L left, points of the circumference.

primary hæmorrhoids are recognised as the right anterior, the right posterior, and the left primary internal hæmorrhoids (fig. 630). As mentioned above, these three hæmorrhoids are the first to be developed, a fact which is found to be amply borne out by clinical experience. The presence of these three primary hæmorrhoids is revealed in about 70 per cent of the cases examined, so that it appears that this is the commonest combination (fig. 632). It does not necessarily follow, however, that the three hæmorrhoids are in the same stage of development, so that if one of them has only reached the first stage, it might easily escape detection when an operation is being performed, unless specially looked for.



*Fig. 632.*—ILLUSTRATING THE EXPOSURE OF THE THREE PRIMARY INTERNAL PILES AFTER PERCTENOTOMY. A PAIR OF KOCHER'S FORCEPS IS PLACED AT THE ANAL MARGIN AT THE RIGHT ANTERIOR, THE RIGHT POSTERIOR AND THE LEFT POINTS OF THE ANAL CIRCUMFERENCE. SLIGHT TRACTION CAUSES THE PILES TO PROTRUDE.

The secondary hæmorrhoids develop in connection with the primary, and are practically offshoots from them. The arterial branch which is distributed to the right anterior quadrant does not give off a secondary offshoot so that a secondary hæmorrhoid is never found to be associated with the right anterior primary hæmorrhoid. The arterial branch distributed to the right posterior quadrant gives off two branches and, accordingly, two secondary piles are developed in connection with the right posterior primary hæmorrhoid, namely (1) a hæmorrhoid situated at the right point in the anal circumference, the right secondary hæmorrhoid; and (2) a hæmorrhoid situated in the middle line posteriorly, the posterior secondary hæmorrhoid. Similarly, two secondary hæmorrhoids are developed in connection with the left primary hæmorrhoid, namely: (1) a hæmorrhoid situated in the left posterior quadrant, the left posterior secondary hæmorrhoid; and

(2) a hæmorrhoid situated in the left anterior quadrant, the left anterior secondary hæmorrhoid (fig. 631). In connection with the latter, there rarely may develop a small anterior secondary hæmorrhoid.

The secondary hæmorrhoids that may be present, therefore, are : (1) the right ; (2) the posterior ; (3) the left posterior ; (4) the left anterior ; and (5) very rarely the anterior.

There are therefore seven or, in very rare instances, eight hæmorrhoids which may possibly be developed in any given case of hæmorrhoids whose position in relation to the anal circumference is constant. Commencing at the middle line anteriorly and passing around the circumference in a direction opposite to that of the hands of a clock, these hæmorrhoids are :

- (1) Anterior secondary (very rare).
- (2) Right anterior primary.
- (3) Right secondary.
- (4) Right posterior primary.
- (5) Posterior secondary.
- (6) Left posterior secondary.
- (7) Left primary.
- (8) Left anterior secondary.

The secondary hæmorrhoids are, moreover, closely connected with the primary hæmorrhoids from which they are developed. Thus, in instances in which all of the possible hæmorrhoids are present, the following constant arrangement will invariably be found. The anterior hæmorrhoid, when present, is usually connected with the left anterior above, but is separated from it by a definite sulcus below.

The right anterior hæmorrhoid is always unattached.

The right hæmorrhoid is practically coalesced with the right posterior, while the posterior, though separated from the right posterior by a sulcus below, is blended with it above. The left posterior hæmorrhoid is coalesced with the left, whereas the left anterior is separated by a shallow sulcus from it. By this arrangement it will be seen that, in those instances in which all of the possible hæmorrhoids are present, they are formed into three groups, viz. :

Group 1. The right anterior primary internal hæmorrhoid.

Group 2. The right posterior primary hæmorrhoid with its two satellites, the right and the posterior secondary hæmorrhoids.



Group 3. The left primary hæmorrhoid with its two or rarely three satellites, namely, the left posterior, the left anterior, and the anterior secondary hæmorrhoids.

As a result of this natural grouping, it is never necessary, when performing the ligature operation, to apply more than three ligatures even when seven or eight internal hæmorrhoids are present. The practical advantages of bearing in mind the relative position of hæmorrhoids in regard to the circumference of the anal orifice are twofold, namely :

(1) One knows exactly where to look for the existing hæmorrhoids, and the risk of overlooking one which may be in an early stage of development is minimised.

(2) It enables the observer to determine definitely whether supposed post-operative recurrence is due to re-growth of hæmorrhoids which have previously been removed, or whether there has been subsequent development of hæmorrhoids which did not exist at the time of the previous operation ; for instance, if the record of the previous operation reveals that the right anterior, the right posterior, and the left primary hæmorrhoids (the usual combination met with) had been removed, and if, when the same patient presents himself several years afterwards on account of a recrudescence of symptoms, it is revealed that the posterior and the left anterior secondary hæmorrhoids are present, then it is obvious that the primary hæmorrhoids that were removed at the previous operation have not grown again, and that the now existing hæmorrhoids did not exist at the time of that operation but have developed since.

#### STAGES IN THE DEVELOPMENT OF HÆMORRHOIDS

Hæmorrhoids are progressive in development, and pass through three distinct stages, namely: (a) primary; (b) intermediate; and (c) final. It does not necessarily follow that all of the hæmorrhoids present in a given case are in the same stage of development. In fact, this very rarely happens, it being a common experience to find that, with the exception of very old-standing cases, the three stages are represented.

*The Primary Stage.* When seen at this stage the hæmorrhoid is small and covered by healthy, unaltered mucosa. Its presence cannot be recognised by digital exploration of the anal canal on account of the

small size of the tumour and the compressibility of the dilated vessels. The tumour has not, at this stage, become sufficiently elongated to permit of it being protruded through the anal orifice during an expulsive effort, though it may be long enough to allow its lower extremity to become gripped by the sphincters. A hæmorrhoid in the primary stage of development bleeds readily, often profusely, whenever it becomes engaged in the grip of the sphincters because the mucous membrane covering it still preserves its delicate structure and has not yet become thickened and fibrosed through long-continued friction. The only symptom to which hæmorrhoids at this stage of their history give rise is *hæmorrhage*. This is often profuse, and is repeated with each act of defæcation. The blood passed is usually bright red in colour, a circumstance which no doubt has given rise to the idea that a hæmorrhoid in this stage of its existence is arterial in structure.

*The Intermediate Stage.* On account of being constantly dragged upon by the action of the sphincters during defæcation, hæmorrhoids become progressively elongated. When sufficiently elongated to permit of it being protruded through the anal orifice, it may be considered to have reached the second or intermediate stage of development. Concomitantly with increase in length, the hæmorrhoid increases in bulk, partly from increased dilatation of the component vessels and partly from exudation into the areolar tissue between the vessels. On account of repeated protrusion the mucosa becomes thickened, with the result that bleeding is less frequent and much less profuse than in the preceding stage. A characteristic feature with regard to protrusion in this stage is that the protruded hæmorrhoid becomes spontaneously reduced as soon as the expulsive effort has ceased or almost immediately afterwards. After reduction has been effected, protrusion does not recur until the next act of defæcation. Owing to changes in the mucosa, the hæmorrhoid has lost some of its original bright red colour. The characteristic features of a hæmorrhoid in the second or intermediate stage of development are : (1) bleeding is less frequent and less copious, several actions of the bowels often taking place without even a trivial loss of blood ; and (2) protrusion through the anal orifice occurs with every act of defæcation, but the protruded mass is spontaneously reducible as soon as the expulsive effort ceases and does not recur except during defæcation.

When hæmorrhoids have reached this stage of development their presence can readily be detected by the examining finger, as a thickened longitudinal fold, especially if the finger be rotated upon its axis at the

points of the circumference of the anal orifice where the hæmorrhoids are normally situated. A hæmorrhoid in this stage can also be seen to protrude through the anal orifice when the patient forcibly strains down.

*The Final Stage.* This stage is considered to have been reached when protrusion is pronounced, is continuous, and does not spontaneously become reduced. It becomes necessary to reduce the protrusion manually after each act of defæcation. Even after manual reduction has been effected, recurrence of the protrusion takes place on slight provocation, such as voiding flatus, coughing, sneezing, walking or standing for any length of time. The mucosa, from constant rubbing against clothing, becomes thickened and tough. In colour the hæmorrhoid has assumed a purplish hue. Bleeding seldom occurs owing to the thickened mucosa. When bleeding occurs from hæmorrhoids which have apparently reached the final stage, it is either due to traumatism or to one or more of the hæmorrhoids not having progressed beyond the primary or intermediate stages.

The recognition that there are *three distinct stages* in the development of a hæmorrhoid is of considerable practical importance, because, in any given example, it is exceptional to find that all of the hæmorrhoids have reached the same stage. In fact, it is not uncommon to find that whereas one hæmorrhoid has reached the final stage, a second may only come into view when the patient forcibly strains down; and a third may not be capable of being protruded at all, and cannot be recognised during a digital exploration. Such a hæmorrhoid may still exist in the primary stage of development, is probably the cause of the bleeding complained of, and may be overlooked during the performance of an operation. Consequently, under such circumstances, if the obvious hæmorrhoids only are removed, while one, still in an early phase of development, is left behind, hæmorrhage, one of the chief symptoms for which an operation for hæmorrhoids is undertaken, may recur after an interval of a few weeks or months.

*Ætiology.* Hæmorrhoids occur most frequently during the fourth and fifth decades of life. It has been estimated that about 70 per cent of individuals who have attained the age of forty years suffer from hæmorrhoids to some extent, but only in a small proportion are they sufficiently pronounced to require surgical treatment. Men are affected nearly three times as frequently as women. Sedentary occupations appear to predispose to hæmorrhoidal formation, though on the other hand laborious work involving great straining is a potent factor.

The most important ætiological factor in the production of hæmorrhoids is habitual constipation. The greater prevalence of hæmorrhoids in the male sex is *most probably due to the fact* that men, by reason of their occupations, necessitating an early departure from home, are less able to devote attention to daily defæcation than the opposite sex. It is obvious that a man who is obliged to take a journey to his work, at an early hour in the morning, must often be obliged to leave home before an evacuation has taken place. The result is that the rectum is frequently converted into a temporary reservoir in which the longer the fæces remain the harder the mass becomes from absorption of moisture. Consequently, prolonged straining is often necessary in order to expel the contents of the rectum when an opportunity offers. The straining necessary to expel hardened masses of fæces must have considerable influence in the production of a dilated and varicose condition of the hæmorrhoidal veins. Those who suffer from hæmorrhoids nearly always give a history of constipation or diarrhœa. In England habitual constipation almost invariably precedes their appearance, but in tropical countries diarrhœa is often an exciting cause.

Heredity appears to exert considerable influence in the tendency to hæmorrhoid formation in young persons. When hæmorrhoids develop under the age of thirty years enquiry often elicits the fact that both parents have been operated upon for hæmorrhoids. Pressure upon the inferior mesenteric veins, such as that exerted by the gravid uterus or tumours of the uterus, and the repeated straining during micturition associated with an enlarged prostate or stricture of the urethra, cause engorgement of the hæmorrhoidal veins with the result that hæmorrhoids are formed.

*Effect of the Presence of Hæmorrhoids upon the Tissues of the Anal Canal.* Varicose veins in any situation lead to passive congestion in the tissues from whence they arise. The passive congestion thus engendered ultimately results in fibrous induration. Evidence of such induration as a sequela to passive congestion is well exemplified in the case of the indurated zone in the neighbourhood of a varicose ulcer of the leg. Similar induration ensues in the submucosa of the anal canal as a result of hæmorrhoids.

The branches of the superior hæmorrhoidal veins arise in a minute capillary plexus situated in the submucosa of that portion of the anal canal known as the *pecten*, which is bounded below by *Hilton's white line*, and above by the free edges of the Morgagnian valves constituting the so-called *pectinate line*. When these vessels have undergone

varicose changes, the plexus becomes the seat of passive congestion, with the result that a deposit of fibrous tissue occurs in the submucosa of the pecten. The fibrous deposit takes the form of a circular band, varying in thickness and density, completely surrounding the lower part of the anal canal, and which is situated between the mucous membrane and the external sphincter muscle. In well-marked instances, the band of fibrous tissue can be distinctly felt to encircle the anal canal, and gives the impression to the examining finger such as would be obtained if a rubber umbrella ring had been inserted beneath the skin at the anal margin. I have named this deposit of fibrous tissue which is circularly disposed in the submucosa of the pecten the *pecten band* (see fig. 634). The pecten band does not exist in the healthy anal canal. It is purely pathological in origin. It is due to passive congestion engendered either by the varicose condition of the superior hæmorrhoidal veins or possibly in some measure to impediment to venous return as a result of habitual pressure upon the hæmorrhoidal veins by a mass of feces in the chronically constipated. The band varies both in thickness and in density. It limits the expansibility of the anal orifice just as effectively as would a piece of whipecord if tied loosely round the anal orifice. In nearly all case of hæmorrhoids, two characteristic symptoms due to the presence of the pecten band manifest themselves, apart from those due to the hæmorrhoids themselves. One of these is difficulty in obtaining complete emptying of the rectum during defæcation, and the other is a diminished size in the calibre of the faecal mass evacuated, the motions being generally voided in short pieces about the size of an ordinary index finger and tapering off at one end. Both of these symptoms are due solely to the presence of the pecten band and both of them disappear after the band has been completely cut through (pectenotomy).

The limiting effect of the pecten band upon the degree of expansibility of the external sphincter muscle is responsible for the straining at stool in order to pass a formed motion and also for strangulation of the hæmorrhoids which sometimes occurs when they have been forcibly protruded through the anal orifice. Such strangulation is usually considered to be due to spasmodic contraction of the external sphincter under the stimulus of painful protrusion, but it is evidently not due to muscular constriction because a muscle is incapable of keeping up tonic contraction for an indefinite period without relaxing. The constriction is due to the unyielding pecten band just in the same way as the rigid margin of the entrance in the femoral canal constricts the portion of intestine or omentum which has been forced into it.

## TREATMENT

When hæmorrhoids begin to make their appearance, their subsequent course is progressive. If untreated they progressively pass through the three stages of development. This is, in large measure, due to the traction exerted upon the hæmorrhoidal tumours by the sphincters during the act of defæcation. A great deal, however, can be done to retard progressive development by judicious *palliative* treatment. Constipation should be combated by means of suitable mild aperients so as to render the motions soft and capable of being voided without undue straining. Drastic purgatives should be avoided. Protruded hæmorrhoidal masses should be returned into the rectum with the least possible delay. For this purpose the patient should be instructed to pass his index finger, encased in a rubber finger-stall, into the anal canal immediately after an action of the bowels, so as to ensure that the hæmorrhoids are pushed well up beyond the grip of the sphincters. Even in those instances in which protrusion of the hæmorrhoids has not yet occurred, it is a good plan to instruct the patient to pass his finger, smeared with ointment or other lubricant, into the anal canal both before and after an action of the bowels for the following reasons: first, the lubrication of the surface of the anal canal ensures the transit of the fæcal masses with a minimum of friction, and consequently there is less likelihood of existing hæmorrhoids being forced down into the grip of the sphincters; secondly, the passage of the finger after defæcation ensures that any hæmorrhoid which may have been forced down is disengaged from the sphincteric grip. Palliative treatment of this kind, if conscientiously carried out, is often successful in retarding progress through the stages and, therefore, an operation may never become necessary. In a certain proportion of cases, however, palliative treatment is of little avail, and sooner or later operative interference becomes inevitable.

*Indications for Operation.* It is quite erroneous to assume that every case, in which the symptoms indicative of hæmorrhoids are present, needs operative interference. It often happens that such symptoms completely subside after a few days' rest in bed, together with due attention to diet, and may not be repeated for many months or even years. In such cases it is obviously unnecessary to recommend an operation. When, however, attacks recur at frequent intervals and the symptoms become more pronounced with each successive attack, the question of early operative interference necessarily arises. There

are only two reasons for carrying out an operation for the removal of hæmorrhoids, namely: (1) copious and recurring bleeding, and (2) uncontrollable protrusion.

*Bleeding.* Slight and occasional bleeding is not of much moment, but when a patient loses considerable quantities of blood at every action of the bowels, his general health soon begins to suffer. He becomes obviously anæmic and the anæmia is progressive. Under such circumstances the sooner an operation is undertaken the better it will be. It is a mistake, as is so often done, to regard the existence of anæmia as a contra-indication to operation, because the anæmia is being produced by daily losses of blood and consequently it will progress so long as the bleeding is permitted to recur. It is time enough to adopt measures for combating the anæmia after the operation has been performed and the leak has been stopped.

*Protrusion.* So long as the protrusion is amenable to reduction, and does not recur until the next act of defæcation, it is only a nuisance and can be tolerated. When, however, the protrusion recurs almost immediately after reduction, after slight exertion such as walking or standing, or during slight expulsive efforts, such as voiding flatus or during micturition, the disability becomes a menace to health because it prevents the patient from taking sufficient exercise. Moreover, it may prevent him from following his occupation if of an active kind. When, therefore, a patient is losing blood daily and his existence has become unendurable on account of recurring protrusion, an operation should be recommended without hesitation.

*Selection of an Operation.* Many operations have been devised from time to time for the cure of hæmorrhoids. Those most commonly practised are: (1) ligature operations; (2) operations by excision of the hæmorrhoidal tumours; and (3) operations by means of the clamp and cautery. Each of these methods have their advocates, and it does not appear to matter much which particular method be adopted, provided that the operation is performed efficiently. Some methods, notably Whitehead's of excising the pile-bearing area, entail the loss of a considerable quantity of blood, so that it would be unwise to select such an operation for a patient who had been reduced to a condition of pronounced anæmia on account of daily copious losses of blood.

An operation undertaken for the cure of hæmorrhoids should be uniformly successful. There should be no failures attributable to the

operation. The operation may be regarded as having failed in its purpose if (1) the symptoms, for the relief of which it was undertaken, persist or return within a period of three or four years; or (2) if symptoms which did not exist prior to the operation develop immediately afterwards. Failure is due to two causes: (1) faulty operation technique, and (2) sequelæ due to the operation itself, and therefore out of the control of the surgeon. The first can be remedied by experience, but the second can only be remedied by discarding that particular operation. Should, therefore, a method of operating be followed by even a small percentage (2 or 3 per cent) of recurrences, the method should be considered to be bad and should not be practised.

The operation by ligature (Salmon's operation), or some modification of it, gives the best results, and is recommended in preference to all others.

*Preparation before Operation.* Careful pre-operative preparation is essential. It is a mistake to think that, because the operation for hæmorrhoids is a comparatively simple one, little care need be exercised in this respect. The preparation should extend over two days, during which period the patient should be kept in bed and given a light diet. Smoking, especially cigarette smoking, should not be permitted as it creates irritability of the pharynx and larynx, with the result that after an anæsthetic there is a good deal of mucus secretion, necessitating coughing. The act of coughing is felt in the perineum and causes unnecessary pain. The colon should be thoroughly emptied by means of efficient purgatives and by lavage. Castor oil should never be administered prior to rectal operations because it is often followed by fecal impaction. The best results are obtained by the administration of a pill consisting of

Calomel	gr. i
Extracti colocynthis co.	grs. iiii
Extracti byoscyami	gr. i

at bedtime, followed by a saline draught on the following morning. The following scheme of preparation is found to be very satisfactory:

*First Day.* The patient is kept in bed on a light diet; neither smoking nor alcohol is permitted. At bedtime an aperient is administered.

*Second Day.* Early in the morning a Seidlitz powder or an equivalent saline is given followed by a cup of hot tea. After the bowels have



acted, a plain water enema (1½ pints at a temperature of 70° F.) should be administered. Light diet is allowed. The patient should remain in bed. A plain water colon wash-out is given before the patient retires to sleep.

*Third Day. (Day of operation.)* If the wash-out on the previous evening has been satisfactory there is no need to repeat it. The perineum should be carefully cleaned with soap and water and then a solution of picric acid should be applied to the surface. It is not necessary to shave the peri-anal skin, unless it happens to be abnormally hirsute. A sterile dressing is then applied and kept in position by a suitable bandage.

No food of any kind is to be given during the four hours preceding the operation, nor is smoking to be permitted.

*Choice of Anæsthetic.* This, of course, should be left to the anæsthetist. Nowadays he has several methods to choose from. One of the most satisfactory is intrathecal percaine, because the anæsthesia of the perineal region persists for five or six hours afterwards. Should this be the chosen method, a hypodermic injection of omnopon, ½ gr., and scopolamine, ⅙ gr., is given half an hour before the time arranged for the operation. At the expiration of half an hour ½ cc. of 1/200 solution of percaine is introduced intrathecally.

*Instruments Required.* Scalpel 1, Salmon's scissors 1, hæmorrhoidal forceps (Author's pattern) 3, Koehler's forceps 3, pressure forceps 1, and a reel of No. 16 plaited silk.

*Technique of the Author's Modification of Salmon's Ligation Operation.*  
*First Stage.* The patient is placed in the right lateral and semi-prone position. In order to ensure that an operation for hæmorrhoids shall be successful it is essential that complete exposure of all of the existing hæmorrhoids is obtained. Hæmorrhoids are usually exposed to view by means of forcible stretching (divulsion) of the external sphincter muscle. Stretching the external sphincter is supposed to be necessary because it is said not to relax even when the patient is deeply anæsthetised. It is, however, unreasonable to suppose that the external sphincter differs from the remaining 519 voluntary muscles of the human body in its reaction to anæsthesia. As a matter of fact it is just as susceptible to anæsthesia as any of the others, but it is incapable of becoming relaxed on account of the constricting effect of the pecten

hand. When the pecten band has been divided it is found that the external sphincter relaxes under the influence of anaesthesia just like any other voluntary muscle. It is not possible to stretch the pecten band. Consequently, if forcible dilatation of the anus is resorted to, the band is torn. As a result, blood is extravasated in the tissues, and eventually becomes converted into fibrous tissue. On account of the blood suffusion, an imperfect exposure of the existing hæmorrhoids is obtained. In order, therefore, to obtain satisfactory exposure of all existing hæmorrhoids the pecten band should be divided with the knife. To do this, the index finger of the left hand is passed into the anal canal as far as the second interphalangeal joint and flexed so as to evert the anal margin. A short linear radiating incision is made through the muco-cutaneous junction. The pecten band, which is quite superficial, is exposed and recognised by its pearl-white appearance (see fig. 634). The incision is carried through the band until the muscular fibres of the external sphincter are exposed, but on no account should they be divided. As soon as the division of the pecten band has been completed, the external sphincter becomes completely relaxed.

*Second Stage.* The skin of the anal margin is seized by Kocher's forceps at the points of the circumference corresponding to the position of the primary hæmorrhoids, viz., midway between the anterior and the right points, midway between the right and the posterior points, and at the left point. Slight traction upon these forceps at once brings the primary hæmorrhoids, together with their satellites if any, into view, thus ensuring that none remains undiscovered (see fig. 632).

*Third Stage.* The hæmorrhoidal forceps are now placed upon the exposed hæmorrhoids. The right anterior primary hæmorrhoid has no satellites and extends higher up than the others, so that it needs a pair of forceps to itself. The right posterior primary hæmorrhoid is next dealt with. It should be remembered that two secondary hæmorrhoids are developed with this one, so that when present they should be included in the forceps. Finally, the left primary hæmorrhoid is similarly dealt with together with the secondary hæmorrhoids which develop in connection with it.

It will be observed that even when the full complement of seven hæmorrhoids are present, they can be manipulated into three groups so that only three ligatures need be used.

*Fourth Stage.* A V-shaped flap of the skin of the anus is dissected up at the base of each primary hæmorrhoid, the extremities of the

V extending to but not beyond the muco-cutaneous junction. A ligature is then placed under the flap, carried round the hæmorrhoid and tied above the end of the hæmorrhoidal forceps as tightly as possible (fig. 633). The right anterior hæmorrhoid is dealt with first, then the right posterior, and the left last.

*Fifth Stage.* The index finger is passed into the anal canal in order to free the lumen which has been somewhat constricted from the application of the three ligatures. The lower end of the hæmorrhoid should not be cut away because by doing so blood escapes from the cut surface causing the pedicle of the hæmorrhoid to shrink so that it may slip through the loop of the ligature, thereby giving rise to serious

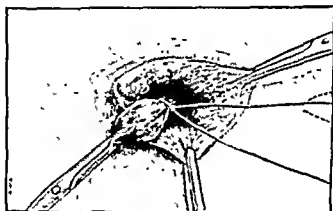


Fig. 633.—ILLUSTRATING THE METHOD OF APPLYING A LIGATURE TO THE RIGHT ANTERIOR PRIMARY INTERNAL PILE. THE KNOT IS PLACED ON THE LUMEN ASPECT.

primary hæmorrhage. Moreover, even if the stump of the hæmorrhoid does not actually slip through the loop of the ligature it shrinks to a certain extent and then strangulation by the ligature is not complete. When, therefore, the slough ultimately separates, a central viable portion of the pedicle is left behind. This, in the course of time, increases in size and forms a small fibrous polypus which, later on, gives rise to further trouble. Finally, the strangulated hæmorrhoids are thoroughly cleansed by washing with an antiseptic solution and then returned into the rectum. A small piece of cotton wool soaked in the antiseptic solution is placed in the pectenotomy wound to prevent the edges from adhering together.

*After-treatment.* Upon the manner in which after-treatment is carried out depends much of the success of the operation. A good

operation may be completely spoiled by neglect in carrying out the details of after-care. An operation for hæmorrhoids is often attended by a considerable amount of after-pain, which is much more pronounced when the sphincters have been forcibly stretched. There is no doubt whatever that exposure of the existing hæmorrhoids by pectenotomy has diminished the severity of the after-pain considerably. After-pain increases in severity during the first six hours, after which period it gradually subsides and finally disappears at the expiration of about twenty hours after the operation. Should the patient, however, become very restless the pain may be kept up indefinitely. It is important, therefore, that steps should be taken to relieve pain as soon as possible. For this purpose a hypodermic injection of  $\frac{1}{4}$  grain of morphia should be given as soon as the patient shows signs of being conscious of pain. A sedative mixture containing opium should then be given at definite intervals with the object of relieving pain and preventing the bowels from acting too soon.

The following prescription will be found to be useful for this purpose :

R/

Liquor ammonii acetatis	m.	xxx
Tinct. catechu	m.	xx
Tinct. opii	m.	x
Tinct. cardamon co.	ʒ.	i
Spirit chloroformi	m.	x
Ex. aqua. cinnamomi	ad ʒ.	i

The first dose of this mixture should be given four hours after the hypodermic injection and repeated at the following intervals : Every 4 hours during the first 24 hours, every 6 hours during the second 24 hours, and then every 8 hours until bedtime on the fourth day (the day of operation being the first), when an aperient is administered.

On the morning of the fifth day, a strong dose of sulphate of magnesia or a Seidlitz powder is given to ensure a satisfactory movement of the bowels. Unless the aperient is strong enough to produce a fluid evacuation, the process of defæcation will be extremely painful, and the patient will be inclined to refrain from allowing the bowels to act in future, with the probable result that an impaction of fæces will ensue. Throughout the remainder of the after-treatment a mild aperient should be given every night to ensure a free action of the bowels taking place every day.

On the ninth or tenth day after the operation, the hæmorrhoid-sloughs separate. These usually become detached quite painlessly without bleeding and are voided with the feces. In the event of a slough remaining adherent it can readily be detached by slight traction. On the twelfth day after the operation, the index finger should be introduced into the anal canal primarily in order to ascertain whether all of the sloughs have separated, and secondarily for the purpose of smoothing out the granulating surfaces resulting from the separation of the sloughs. The digital exploration must be carried out every day until the wounds have healed, an event which takes place on the nineteenth day, on an average. The object of the introduction of the finger into the anal canal is to prevent the granulating surfaces adhering together and so leading to stenosis. The patient may be allowed out of bed on the fifteenth day, and on the nineteenth day he is allowed to proceed to his home.

*Diet During the After-treatment.* During the first four days, whilst the bowels are being confined, the diet should consist chiefly of fluids, e.g. tea, milk and soda, beef tea, jellies, Benger's and toast. No meat or vegetables should be allowed during that period. As soon as the bowels have acted freely on the morning of the fifth day, ordinary diet may be resumed.

*Change of Dressings.* All external dressings must be changed every morning and evening and, in addition, after each action of the bowels. The peri-anal skin should be carefully cleansed before fresh dressings are applied. The pledget of cotton wool, which was inserted into the pectenotomy wound, need not be extracted since it will come away when the bowels act.

*Micturition After Operation.* After an operation for hæmorrhoids, spasm of the sphincter muscle of the urinary bladder and also of the recto-urethralis muscle persists for about twenty hours. During this period, therefore, the patient should not attempt to pass urine; if he does he will in all probability not be able to void it, and the straining during the attempt is apt to prolong the spasm so that catheterisation becomes necessary. During the twenty-hour period, therefore, it is important that the patient should not be allowed much fluid to drink. At the expiration of twenty hours, the spasm will have passed off, and it will be possible to void urine naturally, more especially if the erect posture is adopted for the purpose.

*Recurrence After Operation.* If a hæmorrhoid has been completely removed it cannot possibly recur. Should a patient who has undergone an operation for hæmorrhoids develop symptoms indicating their presence at a future date, one of two things has probably happened: either the hæmorrhoids which existed at the time of the operation were not completely removed, or others have subsequently developed which were non-existent at the time of the operation. It appears that a great deal depends upon the age of the patient when he is operated upon. Thus, if an operation becomes necessary before the patient has attained the age of 30 years he may not by that time have developed all of the hæmorrhoids that are liable to be formed. Since it is only possible to remove hæmorrhoids that exist, others which do not exist may subsequently develop after an interval of from 10 to 15 years. If, however, a patient has attained the age of 40 years before requiring an operation, at that time of life all of the hæmorrhoids that are likely to develop will be present, so that if they are efficiently removed a second operation will never be necessary. It appears, therefore, from the above observations, that an operation for hæmorrhoids in a patient under 30 years of age confers immunity for 10 to 15 years only, whereas it ensures a permanent cure if performed upon an individual over 40 years of age, provided always that the operation has been efficiently performed.

This rule is not peculiar to operations performed by the ligature methods. It holds good for Whitehead's operation, which aims at removing the whole of the hæmorrhoid-bearing area.

*Complications After an Operation for Hæmorrhoids.* *Hæmorrhage* may occur from cutaneous vessels, in which case it soon becomes evident because the blood soaks through the dressings. When the bleeding takes place within the rectum, however, such as may happen when a ligature slips off a pedicle when the lower part of a hæmorrhoid has been removed, the colon may become filled with blood before any outward signs of bleeding manifest themselves. The gradual onset of restlessness with pallor and a tendency to syncope, with perhaps cramps in the lower extremities, and especially if colicky pain in the abdomen ensues, and there is a desire to evacuate the contents of the rectum, these are indications that point definitely to occult bleeding. Since a patient may easily bleed to death from this form of hæmorrhage it is necessary that the bleeding should be arrested as quickly as possible. The most effective way of stopping bleeding into the rectum from the superior hæmorrhoidal vessels is to introduce a vulcanite tube into the rectum

for three or four inches and leave it there for forty-eight hours. The tube permits blood and gas to escape so that when the rectum contracts upon it the bleeding is arrested by compression of the vessels.

*Retention of Urine.* This complication can be prevented by insisting upon the patient emptying his bladder immediately before the operation, and by limiting as much as possible the quantity of fluid taken during the ensuing twenty-four hours.

Reflex irritation may induce a desire to micturate within an hour or so after operation, but should the attempt be made it will usually fail and the vesical irritability will be considerably aggravated. Should a patient not be able to void urine in the recumbent position he should be allowed to stand up and micturate in the usual way. Spasm of the sphincter vesicæ generally subsides after twenty or twenty-four hours so that if no attempt is made to void urine until after that period micturition will take place normally. Should the retention persist  $\frac{1}{2}$  cc. of pituitary extract administered subcutaneously is often efficacious. As a last resort a catheter should be passed under strict aseptic conditions.

*Narrowing of the Lumen of the Anal Canal.* When two or more hæmorrhoids have been removed there is a tendency to constriction of the lumen of the upper part of the anal canal, about  $1\frac{1}{4}$  inches above the anal verge, if the granulating surfaces, left after the separation of the sloughs, are allowed to heal in a horizontal direction. It is important, therefore, that the index finger should be passed daily from the twelfth day until the wounds have quite healed. As a result of the daily digitation the wounds heal from above downwards instead of laterally, and consequently narrowing of the lumen cannot take place. It sometimes happens that when the finger is inserted into the anal canal the patient retracts the rectum to such an extent that the upper extremities of the granulating surfaces cannot be reached. Under such circumstances a bougie should be passed to ensure that dilatation is carried well above the upper margins of the granulating surfaces.

*Stricture of the Anus* sometimes results from excessive removal of anal skin. When healing of the granulating surfaces has been completed, it may be found that expansion of the anal orifice has become so limited that the index finger cannot be passed into the anal canal.

The condition is best treated by completely dividing the external sphincter muscle in the right posterior quadrant, and keeping the edges apart throughout the healing process by careful packing with cotton wool. The resulting scar will be broad, and in most cases is equivalent to increasing the circumference of the anal orifice from  $\frac{1}{4}$  to  $\frac{1}{3}$  inch.



## CHAPTER II

### ANAL FISSURE

A FISSURE is a small, more or less superficial ulcer, situated at the anal margin, usually at the bottom of the cleft between the rugæ which the anal orifice exhibits as the result of the tonic contraction of the external sphincter muscle. The ulcer is, as a rule, pear-shaped or triangular in form and extends upwards in the longitudinal axis of the anal canal for half or two-thirds of an inch. The apex of the ulcer is usually situated at the level of the lower border of the internal sphincter. The broad portion of the ulcer extends down as far as the anal verge. The ulcer varies in depth in different instances; in some, the floor consists of the partially torn-through mucous membrane, whereas, in others, it consists of submucous connective tissue. When the ulcer is of long standing, the base consists of fibrous tissue which is the exposed "pecten band." The margins of the ulcer are well defined and are regular in outline. On account of the contraction of the external sphincter the lateral margins of the ulcer overlap the floor so that its extent cannot be estimated unless the margins are separated. At the lower border of the ulcer there is sometimes a small tag of skin, the so-called *sentinel pile*, which is supposed to indicate that the origia of a fissure is due to a torn-down anal valve.

A fissure may be located at any point in the anal circumference, but in the majority of instances the lesion will be found on the posterior margin of the anus in the middle line or just to one or other side of it.

### ÆTIOLOGY

Chronic constipation is the potential cause of anal fissure. As a result of constipation the rectum is habitually loaded with feces which become hardened from absorption of moisture. So long as the anal orifice expands naturally during the defecatory act hardened feces can be voided without inflicting injury upon the anal margin. As a result, however, of long continued pressure upon the hæmorrhoidal

veins by solid masses of *feces*, passive congestion of the capillary plexus in the submucosa of the pecten leads to the formation of a fibrous ring surrounding the lower part of the anal canal (the pecten band) which narrows the anal orifice and necessitates straining at stool. A vicious circle is thus established, the fibrosis extending until the mucosa of the pecten and the integument of the anal canal below Hilton's white line become adherent to the underlying tissues. The anal orifice being no longer capable of normal expansion during the passage of a mass of formed *feces* is apt to split and give rise to a crack or fissure. The greatest strain is thrown upon the posterior margin of the anal orifice and consequently the resulting fissure is most commonly found to be situated at or near the middle line posteriorly.

Ball of Dublin attributes the formation of a fissure to the tearing down of the lateral attachments of one of the valves of Morgagni. In support of this he points out that the torn-down valve is situated at the lower extremity of the fissure constituting the so-called *sentinel pile*. There is no doubt whatever that a fissure may be produced in this way, but as the presence of a sentinel pile is observed in a very small (7 per cent) number of cases, the tearing down of a Morgagnian valve cannot be regarded as a common cause.

In addition to the constipated habit, the following may be regarded as possible causes of anal fissure, viz., congenital narrowing of the anal orifice, hypertrophied anal papillæ, uterine enlargements and displacements, pelvic inflammations, syphilis, and tubercle.

Anal fissure may be regarded as a disease of adult life, children very rarely suffering from it, except in cases of congenital syphilis. It is found to be much more prevalent in those who lead sedentary lives than among those whose occupation is of an active nature.

#### SYMPTOMATOLOGY

The symptoms indicating the presence of an anal fissure are in nearly all cases well marked and characteristic. They are expressed by pain, both local and reflex, by spasm of the muscles controlling the lower portion of the rectum, and by alteration in the size of the motion passed. To these may be added the occasional loss of blood at stool.

*Pain* is generally well marked and may be considered to be the most definite symptom. It varies considerably in intensity and duration, and may be reflected to distant parts, producing more or less general distress. It is described by patients as a burning, aching,

shooting or throbbing sensation, situated just within the anus; or as an intense itching in the vicinity of the anus. The pain usually ensues either during the passage of a motion or soon afterwards, and may persist for several hours or until the next action of the bowels. On account of the painful phenomena associated with an anal fissure the sufferer is inclined to postpone an action of the bowels as long as possible and so aggravates the constipated habit with the result that the passage of the hardened faecal masses becomes more painful than ever. When the pain has been intermittent and then becomes continuous, the development of an abscess either in the submucous or subcutaneous tissues in the immediate vicinity of the fissure should be searched for.

*Muscular Spasm.* Spasmodic contraction of the external and internal sphincter muscles and also of the levatores ani may be regarded as a constant accompaniment of a fissure. The severity of the spasmodic action of these muscles varies in different cases from merely increased irritability, as evidenced by sudden forcible contraction of the external sphincter induced by the introduction of the finger into the anal canal, to tonic spasm which persists until the next action of the bowels.

*Hæmorrhage.* Bleeding is not a usual accompaniment of a fissure. At the moment when laceration of the anal integument occurs, a small quantity of blood may be lost (not more than a few drops), but it is exceedingly rare, unless hæmorrhoids are also present, for the bleeding to be profuse.

The above symptoms may be regarded as pathognomonic of the disease. They vary in different instances, the one or the other being more pronounced. It should be remembered, however, that sometimes, even when a fissure is well marked, the most prominent symptom is only peri-anal irritation, usually ensuing after defecation and occasionally after voiding flatus. In the majority of cases of uncomplicated fissure, even when pain is not a prominent symptom, expansion of the anal orifice during a straining-down effort is not possible on account of the spasm of sphincters set up by the presence of the fissure.

#### PATHOLOGICAL ANATOMY

When the presence of an anal fissure is suspected a local examination is indispensable. For the purpose of the examination a good light is essential, and the most convenient position to be adopted by the

patient is to lie on his right side with both knees well drawn up. On separating the buttocks, the anus is fully exposed to view so that the condition of the peri-anal skin can be noted. The anal skin is usually redundant and thrown into folds. When these exist, the intervals between the folds should be systematically examined for cracks or excoriations. The condition of the sphincter is next observed. By placing the tip of the index finger upon the external sphincter the presence of spasm can be detected. The degree of spasmodic contraction of the sphincters and of the levatores ani can be readily estimated by requesting the patient to strain down. Should the muscles be in a state of spasm he will not be able to relax the anal orifice. Digital exploration of the rectum should never be omitted, and if attended by severe pain should be completed under the influence of an anæsthetic. The exploration is not made for the purpose of diagnosing the presence of the fissure, as that may have been obvious from the previous inspection, but in order to ascertain whether other diseased conditions co-exist, such for instance as polypi, hæmorrhoids, submucous fistula, abscess, or carcinoma. In all cases of anal fissure a well marked pecten band is present. The band effectually prevents expansion of the anal orifice to normal capacity, with the result that the rectum never evacuates its contents completely.

#### DIFFERENTIAL DIAGNOSIS

The diagnosis of anal fissure is in most cases readily made, the symptoms being so characteristic that it is possible to arrive at a correct conclusion even without recourse to local examination. Nevertheless, however certain we may feel that an anal fissure exists, a thorough local examination, for reasons pointed out above, should be urged in all cases. If then a patient presents himself with a history of having experienced a tearing sensation at the anus with a slight loss of blood during defæcation, especially if followed by intense pain, which supervened either during the act of defæcation or after a short interval, and especially if the pain persisted for a considerable time afterwards, eventually ceasing entirely and suddenly and not recurring until the next action of the bowels, the presumption is strongly in favour of the presence of a fissure either alone or complicated with other rectal disease. There are three conditions from which it is important that an anal fissure should be differentiated, namely (a) syphilitic lesions of the anus; (b) a submucous fistula; and (c) disease of neighbouring pelvic viscera.

(a) *From Syphilitic Fissures.* These are usually multiple and are generally situated on the right or left margin of the anus, seldom in the middle line. They bleed readily when their edges are drawn apart. Although they give rise to pain during an action of the bowels, pain occurs at night irrespective of defæcation. In nearly all cases of syphilitic fissure the horizontal set of inguinal lymphatic glands are enlarged and hard. In addition, other signs of syphilis will generally be present, such as mucous tubercles in the mouth and fauces and the various syphilodermata. A positive Wassermann reaction proves a syphilitic origin.

(b) *From Submucous Fistula.* Fissure may be simulated symptomatologically by a submucous fistula. Both are attended by pain associated with the act of defæcation and by existing sphincterospasm. Pain accompanying a submucous fistula is continuous in character and exhibits no tendency to subside. When such continuous pain has persisted for a few days, a discharge of pus from the anus invariably occurs. A discharge of pus from the anus and the absence of a fissure at the usual site, that is in the middle line posteriorly, points conclusively to a submucous fistula. It should be borne in mind, however, that as the result of septic phlebitis or lymphangitis either submucous or subcutaneous suppuration may ensue in connection with a fissure, so that a submucous or a subcutaneous fistula may exist as complications of a pre-existing fissure.

(c) *From Diseased Conditions of Neighbouring Pelvic Viscera.* In some instances, although symptoms indicating the presence of a fissure may exist, no such lesion can be found even after careful exploration of the anal canal both by the finger and with the anoscope. In such cases careful examination of the pelvic organs should be made. In women a retroflexed or retroverted uterus, or a prolapsed ovary in Douglas's pouch, may cause pain and difficulty in defæcation, thus simulating the symptoms of fissure; while in men, disease of the prostate or the vesiculae seminales, or even of the trigone of the urinary bladder, may be the cause of the symptoms produced.

*Pathological Conditions Complicating a Fissure.* It not infrequently happens that in addition to a fissure other diseased conditions may co-exist. Small fibrous polypi arising from the free edges of the Morgagnian valves are frequently found. They contain well-developed nerve terminals, and when faeces pass over them during the defæcatory act, reflex spasm of the sphincters is produced so that faecal material

is retained in the rectum. The retained faecal material becomes hardened from the absorption of moisture and, when voided, is apt to produce an abrasion of the anal margin. In this way these fibrous polypi are indirectly responsible for the production of a fissure. In size they vary considerably, from a split pea to a kidney bean, and are usually multiple. Recognition of their presence is important because unless they are removed when the fissure is operated upon, they are apt to increase in size and interfere with the healing of the wound. Internal piles are very often present, and account for the copious attacks of bleeding in some cases of fissure.

A blind internal fistula of the subcutaneous, submucous, sub-sphincteric or ischio-rectal type, is very frequently met with as the result of septic phlebitis or lymphagitis arising in the fissure. The type of the fistula depends upon the part of the fissure in which the septic process starts. When originating at the lower margin of the fissure, the subcutaneous veins and lymphatics are involved with the result that the fistulous tract is subcutaneous. Similarly when the septic process starts at the upper margin of the fissure the fistulous track is found beneath the mucosa, and generally takes an oblique course upwards and forwards beneath the hæmorrhoidal vessels. When, however, the septic process starts in the floor of the fissure, three courses are open to it, either between the superficial and deep portions of the external sphincter, or between the external sphincter and the fascial expansion which separates the muscle from the ischio-rectal fat, or between the external and the internal sphincters into the ischio-rectal fossa. It is important, therefore, that these localities should be searched for possible fistulous tracks whenever an operation for fissure is performed.

#### TREATMENT

The treatment of an anal fissure may be divided into (a) palliative, and (b) operative. Palliative treatment generally suffices in recent uncomplicated cases. In all complicated cases, and in simple ones of chronic duration, there is so much spasm of the sphincters, generally with attendant hypertrophy and induration, and pain is at times so severe that operative measures should be undertaken without delay. Palliative treatment, however, should always be given a trial for two or three weeks and then, unless marked relief has been obtained, an operation should be advised. In the event of the fissure being uncomplicated, palliative treatment is likely to be successful if the abrasion is superficial, is of short duration, and spasm of the sphincters is not

marked. Should the fissure, however, be complicated by the presence of other rectal disease, or when the edges of the ulcer are thickened and there is definite spasm of the musculature of the pelvic outlet associated with severe pain whenever an action of the bowels takes place, operative interference is indicated.

#### PALLIATIVE TREATMENT

This consists of measures for preventing constipation, attention to local cleanliness, and the application of local remedies.

*Constipation.* A daily action of the bowels must be ensured by a suitable aperient every night at bedtime. Whenever possible the aperient should be taken at a stated time, because habit enters so largely into the scheme of daily evacuation. The most suitable aperient is one which acts without causing general discomfort and at the same time ensures free evacuation of the contents of the bowel. The motions should be kept soft but not liquid. An injection of two or three ounces of olive oil at bedtime, to be retained, has an excellent local effect and will often suffice to produce a free action of the bowels without the aid of aperients.

*Cleanliness.* This is an absolute necessity, since an accumulation of faecal matter in the fissure or between the anal rugæ causes irritation which excites spasm of the sphincters. The anus should be carefully bathed with warm water at least twice daily and also after each evacuation.

*Local Applications.* When the mucosa of the anal canal becomes dry, and especially when it becomes sclerosed by passive congestion from the pressure of scybulous masses upon the hæmorrhoidal veins, it is apt to crack during the slight dilatation caused by the passage of the examining finger. Exactly the same thing occurs during the passage of a formed motion so that it is advisable to introduce some form of lubricant into the anal canal before and after defæcation. Perçine ointment (1 per cent) is especially useful for this purpose on account of its anodyne properties.

In the majority of cases of uncomplicated fissure relief and often complete cure can be obtained by attention to these details.

## OPERATIVE TREATMENT

Fissures of long standing, and particularly those that are complicated by the co-existence of other diseased conditions, can only be cured by operation. There are two methods, namely, incision and stretching the sphincters. The former is the one that is usually practised, and is known as Boyer's operation. The latter has found favour among French surgeons, and is known as Recamier's method. Both of these procedures have the same object in view, namely, the elimination of the spasmodic action of the external sphincter muscle, in fact, to paralyse it temporarily and thus ensure the physiological rest which is so essential for the process of repair.

*The Operation by Incision (Sphincterotomy or Boyer's Operation).* There is no doubt that spasmodic contraction of the external sphincter muscle prevents cicatrization of a fissure. Since the muscle surrounds the anal orifice it forcibly constricts the orifice when it contracts and, therefore, when it has been completely divided closure of the anal orifice is no longer possible until union of the divided ends has taken place. In the interim the fissure is afforded the physiological rest necessary for repair. That division of the external sphincter is instrumental in permitting the fissure to cicatrize is proved by the fact that repair takes place equally well when the muscle has been divided at a point other than that occupied by the fissure, and also that a single division suffices for the healing of two or more co-existent fissures. Boyer divided the external sphincter through the base of the fissure. It is not necessary to do so because the fissure will heal perfectly well by reason of the sphincter having been thrown out of action. The seat of election for dividing the muscle is at the junction of the middle and posterior thirds, as in that situation no damage is inflicted upon the nerves supplying the muscle, because the anterior two-thirds are supplied by the inferior hæmorrhoidal and the posterior third by the muscular branch of the fourth sacral nerve.

It is not necessary, however, to divide any of the fibres of the external sphincter in order to cure a fissure. All that need be done is to divide the pecten band which is always well marked in cases of fissure. As previously described, the pecten band is a deposit of fibrous tissue in the submucosa of the part of the anal canal between Hilton's white line and the edges of the Morgagnian valves. The band constricts the anal orifice and the efforts of the levatores ani to expand it cause reflex spasm of the external sphincter. Division of the pecten



band allows the levatores to expand the anal orifice during defaecation without producing reflex sphincteric spasm and the fissure obtains the physiological rest necessary for cicatrisation. This operation is called pectenotomy.

*The Operation of Pectenotomy. (Author's Operation.)* The pecten band is within the anal canal and not beneath the skin of the anal margin. Consequently in order to expose it the lower half of the anal canal must be everted. This is done by introducing the index finger of the left hand (the patient being in the right lateral and semi-prone position) as far as the terminal interphalangeal joint and then flexing

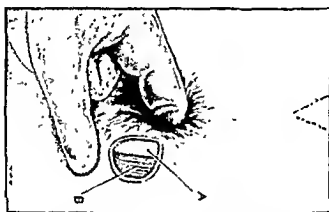


Fig. 634.—SHOWING THE METHOD OF EVERTING THE LOWER PART OF THE ANAL CANAL IN ORDER TO EXPOSE THE PECTEN BAND. THE LINEAR INCISION THROUGH THE ANAL SKIN GIVES WIDELY EXPOSING A THE PECTEN BAND AND B THE INNERMOST FIBRES OF THE EXTERNAL SPHINCTER MUSCLE.

it forcibly in order to evert the anal orifice. At the same time the thumb of the left hand presses the skin of the anus outwards (fig. 634). The effect of this is to push the inner border of the external sphincter outwards. An incision is now made through the mucosa of the lower part of the anal canal in the right posterior quadrant. As soon as the mucosa has been divided the pecten band comes into view and is recognised by its pearl-white appearance. The band is cautiously divided strand by strand until it has been cut through completely. During the operation no muscular fibres are divided. A careful search is then made for polypi, internal piles, or a blind internal fistula that may be present. Finally a small piece of cotton wool soaked in perchloride of mercury solution (1 to 500) is inserted into the pectenotomy wound to prevent the edges from adhering (fig. 635).

The results of the operation are extremely satisfactory, and so far as

my experience goes it has never failed to cure a fissure. The wound takes about nineteen days to heal, and by that time the fissure has disappeared.

*After-treatment.* Pain after an operation for fissure is generally pronounced during the first twenty hours owing to spasmodic contractions of the external sphincter. So soon, therefore, as the patient shows signs of distress a hypodermic injection of morphia or omnopon should be given. The dressings, with the exception of the pledget of cotton wool in the sphincterotomy or pectenotomy wound, are changed at the expiration of twenty-four hours and the anus and peri-anal skin are carefully swabbed with an antiseptic solution. Throughout the after-treatment the external dressings should be changed every morning

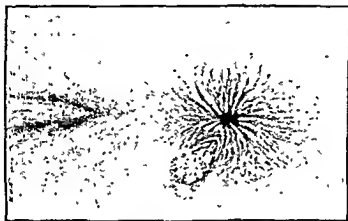


Fig 635.—SHOWING THE METHOD OF KEEPING THE EDGES OF THE PECTENOTOMY WOUND APART BY INTRODUCING A PACKING OF COTTON WOOL. IN THIS INSTANCE THE PECTENOTOMY WOUND IS IN THE RIGHT ANTERIOR QUADRANT OF THE ANUS.

and evening, and also after each action of the bowels. On the fourth day the pledget of cotton wool in the wound should be removed. On the fifth day the bowels are freely relieved and subsequently every day afterwards. Unless a satisfactory action is procured every day faecal impaction may occur. The patient should be kept in bed for ten days or a fortnight. When the operation of sphincterotomy has been performed, the wound takes about four weeks to heal, but a pectenotomy wound is generally completely cicatrised in three weeks. In those instances in which delayed healing occurs, it will be found that the anterior margin of the wound has become œdematous and falls into the wound. If left, healing of the wound will be delayed considerably, so that in order to expedite the healing process the œdematous margin should be treated like an internal pile and ligatured under the influence of either general or local anæsthesia.

*Gabriel's Operation.* This consists in removing a triangular area of skin extending backwards from the posterior margin of the anal orifice. Gabriel claims that the removal of this somewhat extensive area of skin is essential for ensuring external drainage until the wound in the anal canal has healed. The operation is performed in the following manner: Commencing at the anal margin on each side of the fissure an incision is made backwards for about two inches on either side of the middle line. The extremities of these incisions are then connected by means of a cross-cut about  $1\frac{1}{2}$  inches long, a triangular area being thus mapped out. The triangular piece of skin is then dissected up with scissors, including the fissure and the sentinel tag when one exists. The operation is completed by dividing the inner fibres of the external sphincter muscle and gently stretching the sphincters. The patient is fit for discharge at the expiration of three weeks. He is instructed to pass a bougie every night for a few weeks. Such exercises as running, strenuous games, and riding on horseback are not to be indulged in until the scar is some months old. I have not performed the operation because it seems to me to be unnecessary to sacrifice so extensive an area of healthy skin merely for the purpose of drainage.

*Stretching the Sphincters. (Récamier's Operation.)* By this means the spasmodic contraction of the sphincters is temporarily suspended. After an interval of two or three weeks the muscles regain their normal tone and are capable of closing the anal orifice, but in the interim the fissure has had the opportunity of healing under the enforced conditions of rest from muscular contraction.

*Operative Technique.* The patient is placed in the lithotomy or in the right lateral and semi-prone position. The index fingers are introduced into the rectum, placed back to back and forcibly separated in the antero-posterior axis. During this manoeuvre the terminal phalanges should not be fully flexed lest laceration of the mucosa result. Traction is steadily maintained until the resistance of the sphincters has been overcome. Similar traction is then carried out laterally, that is, in a direction at right angles to the first. As soon as the dilatation has been carried out to the required extent the anal orifice gapes and the rectal mucosa protrudes slightly. It almost invariably happens that the fissure is lacerated during the stretching process, but this does no harm. As a result of the stretching there is generally local extravasation of blood beneath the skin posteriorly to the anus, but it does not amount to a hæmorrhoma and usually clears up in about a week.

## CHAPTER III

### PRURITUS ANI

THOUGH the term *pruritus ani* simply means an itching sensation in the region of the anus, general usage has caused it to indicate a definite pathological change in the structure of the skin of the anus and peri-anal region.

This change is inflammatory in character and is really a dermatitis pure and simple. In some individuals, especially in those who exhibit a tendency to gouty and eczematous disorders, the inflammatory process is acute, whereas in those in whom no such tendency exists, it is chronic in type and takes the form of a skin sclerosis.

A simple itching sensation in and around the anus is not uncommonly met with as a symptom of fissure or fistula, but this should not be spoken of as *pruritus ani* unless definite changes in the skin have taken place.

In the acute form the peri-anal skin is reddened, the natural rugæ are enlarged, sometimes definitely oedematous, and often multiple superficial excoriations are discernible. In the chronic form the skin immediately surrounding the anal orifice and also that lining the lower portion of the anal canal is thickened and indurated and has lost much of its natural elasticity. The rugæ are enlarged and thickened and are often thrown into distinct folds. The skin of the anal canal cracks readily when put upon the stretch, as for instance during digital examination, or during the passage of a formed motion. These cracks are extremely painful and also constitute sources of infection by *streptococcus faecalis* and other organisms, thus aggravating the dermatitis.

### ÆTIOLOGY

It is not possible to define a particular ætiological factor in the production of *pruritus ani*. It is probable that several pathological conditions of the anal canal and lower half of the rectum may produce it in individuals who are predisposed to inflammatory skin conditions. The fact that the majority of patients who suffer from *pruritus ani*

display evidence of a gonty, rheumatic, or eczematous diathesis and not uncommonly are affected with eczema in other parts of the body, supports this view. Accordingly in patients thus predisposed, morbid conditions of the rectum are capable of producing the changes in the peri-anal skin which are associated with pruritus ani.

Given the predisposition, then, the following may be considered to be the more common exciting causes, viz.: want of cleanliness, rectal constipation by which is meant incomplete emptying of the bowel, the existence of a well-developed *pecten band*, the presence of small fibromata attached to the edges of the Morgagnian valves (hypertrophied anal papillæ), protruding internal piles, prolapsus mucosæ recti, and blind internal fistula. Of these the most important is imperfect cleansing of the anal region after defæcation. Rectal constipation is also a very common cause. In such instances the rectum is never empty, and the presence of fecal masses not only causes venous congestion from direct pressure, but gives rise to the necessity of voiding flatus frequently. Whenever flatus is voided under these circumstances a small quantity of mucus escapes and makes the peri-anal skin moist and liable to crack. The commonest cause of rectal constipation is the presence of a well-developed *pecten band*, which necessitates straining at stool and causes passive congestion in the circum-anal veins.

### SYMPTOMATOLOGY

The most prominent symptom is an intolerable itching of the anus and peri-anal region. In acute cases the itching is more or less continuous and is accompanied by smarting and burning sensations if excoriations have been produced by scratching or rubbing. In the chronic form the itching or irritation is intermittent and generally supervenes (1) after an action of the bowels, and (2) soon after the patient has become warm in bed. The itching which supervenes as soon as the patient retires to bed is exceedingly distressing, and disturbs his rest to such an extent that his health, in time, begins to suffer.

### TREATMENT

The discovery and the elimination of the exciting cause of pruritus ani are essential if a cure of this most troublesome complaint is to be effected. In about 80 per cent of the cases met with, the changes in the structures of the anal and peri-anal skin are due to the presence of pathological conditions in the anal canal and the lower half of the

rectum. In the remaining 20 per cent the dermatitis is part and parcel of a gouty or rheumatic diathesis, and is therefore associated with similar manifestations in the skin elsewhere. We have seen that the commonest cause of pruritus ani is habitual constipation, and that the commonest cause of the latter is the presence of the pecten band which limits the expansion of the anal orifice during the defæcatory act and prevents complete emptying of the contents of the rectum. As a direct consequence the rectum always contains fæces which, by pressing upon the hæmorrhoidal veins, produce passive congestion of the circum-anal veins and cause sclerosis of the anal and circum-anal skin.

It is of primary importance, therefore, to ascertain whether a pecten band is present and if so to divide it completely (pectenotomy). At the same time any existing pathological conditions such as fibromata of the anal canal, internal piles, prolapsus mucosæ recti, or a blind internal fistula, should be appropriately dealt with.

Operative interference such as indicated above, although essential, will not, however, effect a complete cure unless the rectal constipation, which is invariably present in cases of pruritus ani, is also treated. For this purpose a complete evacuation of the contents of the rectum and sigmoid colon should be obtained at least once during twenty-four hours. A suitable aperient should be given every night at bedtime and at the same time the rectum should be washed out with half a pint of plain water at a temperature of about 80° F. after an attempt has been made to evacuate the contents of the rectum naturally. Even though an evacuation has taken place, it will generally be found that the enema causes a further evacuation, showing that some degree of fæcal retention existed.

Quite as important as the above, however, is the need to impress upon the patient the value of thoroughly cleansing the peri-anal region after defæcation and of drying the skin with a soft towel without undue rubbing.

The peri-anal skin should then be smeared with an ointment such as percainal (1 per cent) to allay the irritation. If the dermatitis is acute an ointment consisting of :

R/

Pulv. zinci oxidi	ʒ ii
Liniment camphoræ	ʒ iii
Anæsthesine	ʒ ii
Lanoline	ʒ i
Vaseline	ʒ i ss.

should be applied to the skin surface. In those instances in which there is œdema of the skin of the anus, a lotion consisting of lactate of lead gives great relief. The lactate of lead can be easily prepared by mixing one drachm of the liquor plumbi subacetatis with seven drachms of fresh milk. A cream-like substance is thus formed which can be applied as an ointment. In those instances in which a decided gouty or rheumatic diathesis is apparent, or when there is evidence of eczema in other parts of the body, suitable remedies and dietary for such conditions should be prescribed in addition to the local treatment. For obstinate cases a saturated solution of nitrate of silver is useful for destroying the superficial layers of the dermis. The solution is applied to the surface with a camel's hair brush and left *in situ* for about ten minutes. A solution of sodium chloride (1 dr. to 1 pint) is then applied to neutralise the nitrate of silver.

From time to time various substances such as alcohol, quinine and urea and anæsthetic solutions in oil have been injected into the subcutaneous tissue of the peri-anal region, but so far have met with little success.

Such procedures as dividing the peripheral nerves or undercutting the anal and peri-anal skin, as in Ball's operation, are so seldom successful that they are scarcely worth doing.

## CHAPTER IV

### PROLAPSE OF THE RECTUM

THIS consists of an eversion and protrusion of part or of the whole of the rectum through the anal orifice, the eversion commencing at the anal margin. There are two varieties of prolapse, in one the protrusion consisting of mucous membrane only, whereas in the other all of the coats of the bowel are involved. The former is known as partial prolapse (*prolapsus mucosæ recti*), and the latter as complete prolapse (*procidencia recti*).

#### PARTIAL PROLAPSE OF THE RECTUM (*Prolapsus Mucosæ Recti*)

A prolapse consisting of the mucous coat of the bowel never attains large dimensions. The extent of the protrusion, as measured from the muco-cutaneous junction to the free border, does not exceed two inches in length. It may involve only one margin of the anus (unilateral partial prolapse), or may affect both sides (bilateral partial prolapse). When unilateral, the more common condition, the protrusion may involve part or the whole of the anal margin. When bilaterally disposed the appearance is that of two lateral flaps separated anteriorly and posteriorly by a distinct hiatus, which is very characteristic, and therefore of value in differential diagnosis.

The protruded mucosa is, as a rule, healthy in appearance, unless excoriated or ulcerated as the result of contact with the clothing. The hæmorrhoidal veins are not thickened or dilated, though the mucous membrane is more or less indurated on account of long-standing passive congestion, and in consequence may present a somewhat purplish colour in contrast to that of normal mucous membrane.

*Ætiology.* Normally, slight protrusion of the mucosa of the anal canal takes place during the act of defæcation, but this becomes spontaneously reduced so soon as the expulsive effort has ceased. The readiness with which, in some instances, the mucosa can be protruded is attributable to the redundancy of the mucous coat of the rectum.



length of protrusion, as measured from the free margin to the mucocutaneous junction, rarely exceeds two inches (fig. 637).

*Differential Diagnosis.* There are two conditions with which partial prolapse of the rectum (*prolapsus mucosae recti*) may be confused, namely, prolapsed internal piles and complete prolapse (*procidentia recti*).



Fig 637.—PROLAPSUS MUCOSAE RECTI. THE PROLAPSED MUCOSA CONSISTS OF TWO LATERAL FOLDS SEPARATED BOTH ANTERIORLY AND POSTERIORLY BY A DISTINCT HILUS.

(From a drawing made from a Photograph.)

*From Internal Piles.* The protruded mass in prolapsed internal piles is divided by vertical sulci or hiatuses into distinct segments of which the right anterior, the right posterior, and the left are the primary divisions. In cases of prolapsed mucosa the continuity of the protruded tissue is interrupted anteriorly and posteriorly and nowhere else. If an internal pile be grasped between the finger and the thumb a mass of veins will be felt to exist between the layers of mucous membrane; whereas if the protruded tissue in a case of prolapsed mucosa be felt in the same way, it will be ascertained not only that the

veins are neither dilated nor thickened, but that the two layers of mucosa glide easily over each other unless cohesion between them has occurred from chronic inflammation.

*From Procidentia Recti.* Of much greater importance is the diagnosis from complete prolapse because upon the correctness of the interpretation of the physical signs of each depends the safety and the success of the remedial measures to be adopted. In prolapsed mucous membrane the protrusion seldom exceeds two inches in length, and is generally longer on one side than on the other, whereas in cases of procidentia the length of the protruded mass is uniform on both sides and measures from three to six inches or even longer. The aperture leading into the rectum is oval or circular in prolapsed mucosa, and is generally centrally placed, whereas in procidentia it is often slit-like in appearance and is situated posteriorly on account of the traction backwards exerted by the meso-rectum. In procidentia there are no hiatuses to be seen, creating separate segments, but the mucosa covering the protrusion is thrown into concentric furrows which are quite characteristic (fig. 638). Finally, when the index finger is introduced into the bowel and the intervening tissues are grasped between it and the thumb placed externally, the presence of the muscular coats of the rectum in cases of procidentia can be recognised by the solidarity of the tissues between the layers of mucosa.

*Treatment.* Partial prolapse of the rectum, consisting as it does of a double layer of mucous membrane with nothing but cellular tissue and normal blood-vessels between them, can be completely and permanently cured by surgical means. When, however, the patient refuses an operation or his general health precludes operative interference, considerable relief may be afforded by palliative measures.

*Palliative Treatment.* This should consist of (1) reduction of the protruded mass; (2) the prevention of its recurrence; and (3) adopting measures to improve the tone of the sphincter muscles.

*Reduction of the Protruded Bowel.* The patient should be instructed how to reduce the protrusion as soon as possible after it has taken place. For this purpose he should lie on his right side, and, having anointed the protrusion thoroughly with a suitable lubricant, grasp it between the fingers of both hands, one hand anteriorly and the other posteriorly, and apply continuous pressure in the direction of the lumen of the

bowel. When the protrusion has disappeared into the rectum, the index finger of the nterior hand should be made to follow it up into the bowel so as to ensure that the protruded tissue has been pushed upwards beyond the level of the internal sphincter muscle, that is, for at least one and a half inches. After the reduction has been completed, the prone position should be maintained for nu hour.

*Prevention of Recurrence of the Protrusion.* The avoidance of all straining efforts, lifting heavy weights, standing for long periods, and exercise of a fatiguing nature should be enjoined. Constipation, with its attendant straining at stool, should not be permitted to occur. An action of the bowels should be obtained immediately before retiring to bed to ensure that the protrusion does not occur during the night-time. The diet should be regulated so that the minimum of waste material is formed. Rest in the recumbent position during an hour after an action of the bowels is advisable, to diminish the tendency to protrusion. In order to prevent the faecal contents of the rectum becoming hard, the injection of an ounce of olive oil or paraffin into the rectum at bedtime, to be retained, is exceedingly useful. In many cases it will be found that an injection of an ounce of olive oil into the rectum an hour or so before the morning action of the bowels takes place prevents undue straining by ensuring that the faecal masses are well lubricated.

*Improving the Tone of the Sphincter Muscles.* The inevitable result of continuous dilatation of the anal orifice is loss of power in the sphincter muscles. Not only is it produced by over-stretching but actual atrophy of the muscular fibres ensues as the result of continual inactivity. It is obvious, therefore, that the sooner the protrusion is reduced and its recurrence effectually prevented, the sooner will the loss of sphincteric power be regained. The patient should be instructed to contract the anal orifice forcibly ten or a dozen times at frequent intervals during the day-time. The exercise of the sphincter and levator muscles in this way is very beneficial.

*Operative Treatment.* By far the most satisfactory way of treating partial prolapse is by operation, which should be undertaken in all cases in which the general condition of the patient is satisfactory.

The bowels having been thoroughly emptied by aperients and colonic lavage, extending over three or four days if necessary, the patient is anæsthetised and placed in the right lateral and semi-prone position on the operating table. By gently dilating the anal canal the

mucosa is made to protrude as far as possible. In those instances in which the prolapse is confined to one lateral margin, the condition of the mucosa of the opposite side should be carefully ascertained, so as to avoid failure to detect a commencing prolapse on the opposite side.

*Operative Technique.* The redundant mucosa may either be excised or may be treated by ligature in sections, as in the operation for internal hæmorrhoids. Of these the ligature method is the more satisfactory. When the section method is employed the prolapsed fold of mucosa is divided into segments by longitudinal incisions. Each segment is ligatured separately. The chief objection to this method is that a considerable quantity of blood may be lost when the incisions are made, and, in the event of a dilated hæmorrhoidal vein being partially cut through, a severe loss of blood may take place during a few hours after the operation. The safest plan of applying ligatures is that devised by the late Mr. D. H. Goodsall. A piece of ligature material (No. 16 plaited silk) about four feet long is threaded through three straight three-inch Hagedorn needles which are placed as follows: one in the centre and the others on either side of the first, midway between it and the end of the ligature. The central needle is introduced at a point one-eighth of an inch distant from the muco-cutaneous junction in the middle of the prolapsed fold of mucosa and made to emerge on the inner aspect half an inch above the level of the point of entry. The remaining needles are introduced in a similar manner midway between the centre and the extremities of the fold respectively. The ligature is then cut across at the site of each needle, thus creating four separate loops. Each loop is then tied as tightly as possible without interlocking. In the event of a fold of mucosa of equal dimensions existing on the opposite side it should be dealt with in a similar manner. When the operation has been completed, the prolapsed mucosa is returned into the rectum and maintained in position by a pad and bandage.

*After-treatment.* An action of the bowels should not be permitted until the fifth day after the operation when an olive oil enema should be given. Until a satisfactory result has been obtained the oil enema should be repeated. Afterwards two ounces of oil should be introduced into the rectum every morning and evening to ensure that scyphala are not formed. Aperients should not be given until the ligatures have been cast off, usually on the ninth or tenth day after the operation. On and after the twelfth day the finger should be introduced into the anal canal to ensure that the granulating surfaces, left after the separation

of the sloughs, cicatrise from above downwards instead of laterally. Should cicatrisation take place laterally some degree of stenosis will occur.

#### COMPLETE PROLAPSE (*Procidentia Recti*)

Complete prolapse of the rectum consists of an eversion and extrusion of the entire wall of the rectum throughout its circumference. The eversion begins at the anal margin, so that the mucosa at the base of the protrusion is directly continuous with the skin at the anal margin. The extrusion may either be incomplete, when only a portion of the rectum is extruded, or complete when the entire length of the rectum has been everted.

*Pathological Anatomy.* In those instances in which the whole of the rectum has been extruded, the tumour consists of a double tube, one placed within the other. The outer tube consists of the rectum, the mucosa being continuous with the skin of the anal margin at the base and with the mucosa of the pelvic colon at the apex of the protrusion. The inner tube consists of an equal length or more of the pelvic colon which has also been extruded. Between the two tubes is a prolongation of the pelvic peritoneum which forms a pouch anteriorly and at the sides, but not posteriorly. In those instances in which the whole length of the rectum has been extruded, the apex of the protrusion is situated at the recto-sigmoidal junction. Eversion of the bowel does not extend beyond that point on account of the unyielding nature of the so-called recto-sigmoidal sphincter. In cases of pronounced visceroptosis, coils of small intestine descend into the peritoneal pouch, with the result that the protrusion is increased in bulk but not in length. Similarly, extrusion of a greater length of pelvic colon than that corresponding to the length of the rectum increases the size of the protruded mass. Anatomically the rectum measures about five inches in length, so that when it has been completely extruded, after allowing for some degree of stretching, the protrusion seldom exceeds six inches in length, though in bulk it varies considerably.

*Ætiology.* The causes of complete prolapse are divisible into (a) predisposing, and (b) exciting.

*Predisposing Causes.* Age appears to have some influence, because the protrusion is most commonly met with during the first decade of

life or after the fourth. The usual history obtainable from an adult is that he remembers that, when a child, he was treated for prolapse and that there has been no recurrence of it until he had reached middle life. In such instances it is probable that the musculature of the pelvic floor had regained tone during the period of vigorous life.

Among adults, males appear to be quite as much predisposed to prolapse as females. Among children under ten years of age boys are more often affected than girls. This is probably due to the fact that boys are disposed to suffer from difficulty in micturition caused by phimosis or the presence of a vesical calculus.

Work of a laborious nature, necessitating repeated straining, such as the lifting of heavy weights, is undoubtedly an occasional cause. Prolapse of the rectum does not occur in individuals of robust health during adult life, but is occasionally met with in those whose general health is impaired from any cause associated with wasting and loss of muscular tone. Improper feeding and bad hygienic surroundings may be contributory causes among children.

(b) *Exciting Causes.* These are divisible into (1) those that operate during child life, and (2) those that are incidental to adult life.

(1) *Causes in Children.* In both sexes protracted diarrhoea is one of the most potent causes. The tenesmus thus engendered not only relaxes the sphincters but so strains the muscular coats of the rectum and the musculature of the pelvic floor that eversion and protrusion of the terminal portion of the rectum take place as a natural consequence. Similarly, repeated straining efforts to evacuate the bowel in the habitually constipated produce the same result. The custom, so often insisted upon by ignorant domestic nurses, of allowing a child to sit upon a commode during long periods in order to obtain an action of the bowels cannot be too strongly condemned. In boys, phimosis and the presence of a vesical calculus produce straining efforts which are apt to cause rectal prolapse.

(2) *Causes in Adults.* Chronic constipation in old and feeble subjects, in whom the rectum is habitually distended with faecal accumulation, is a common cause. The sphincters in such cases are generally lacking in contractile power and the levatores ani fail to afford adequate support to the pelvic floor. As a consequence, eversion and protrusion of the rectum are apt to occur, especially when prolonged straining is necessary to expel scybalous masses. Persistent diarrhoea, especially when associated with tenesmus, unless treated, may give

rise to prolapse of the rectum. The existence of a bulky tumour in the rectum, such as a villous papilloma, has been known to have been the exciting cause.

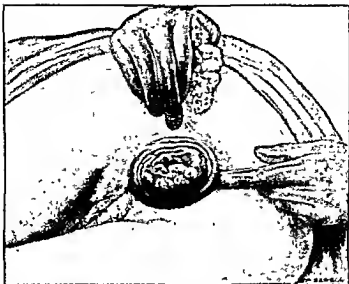
Stricture of the urethra and enlargement of the prostate, by reason of forcible expulsive efforts during micturition, may, in the course of time, lead to eversion and protrusion of the bowel. In either sex paralysis of the sphincter muscles, as the result of disease or injury of the spinal cord, and the loss of sphincteric control due to division of the sphincters and the levatores ani during operation for a para-rectal fistula are potent causes.

*Symptomatology.* The most prominent symptom of complete prolapse of the rectum is a definite eversion and protrusion of the bowel commencing at the muco-cutaneous junction and involving the entire circumference of the anus. The protrusion takes place during defæcation, micturition, or any straining effort. In recent cases the protrusion is slight and is readily replaced by manual pressure, or is spontaneously reducible when the recumbent posture has been assumed. In the course of time the protrusion gradually increases in bulk, more and more of the bowel becoming everted, until eventually the whole of the rectum has been extruded. Spontaneous reduction is now no longer possible, even when the patient remains recumbent for an indefinite time. Active exercise must be abandoned on account of the discomfort caused by the protrusion, with the result that the patient's health deteriorates.

Whenever the bowel is protruded a dull aching, throbbing pain ensues, which entirely disappears immediately after reduction has been effected. As a result of frequent protrusion the mucosa loses its normal appearance and becomes indurated and the seat of superficial ulceration. The ulceration is usually found at the apex of the protrusion. Slight bleeding takes place from the ulcerated surface.

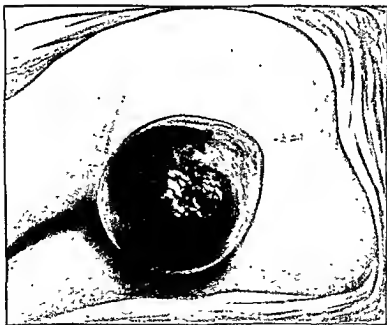
On account of the stretching of the sphincters, the contractile power of the anal orifice is greatly diminished, so that involuntary evacuations are apt to take place. In some instances, especially in women, frequency of micturition as well as incontinence of fæces is complained of.

*Pathological Anatomy.* In well-marked examples, after the protruded mass has been reduced, the anus is patulous, readily admitting two or three fingers. The natural anal rugæ have become effaced from the over-stretching of the anal orifice. Sphincteric control is



*Fig. 638.*—COMPLETE PROLAPSE OF THE RECTUM, THE APEX OF THE TUMOUR CONSISTING OF THE RECTO SIGMOIDAL JUNCTION. THE PERITONEAL POUCH DID NOT CONTAIN SMALL INTESTINE. THE INDEX FINGER IS PLACED AT THE MUCCUTANEOUS JUNCTION AT THE BASE OF THE TUMOUR, INDICATING THE ABSENCE OF THE SULCUS WHICH ALWAYS EXISTS IN PROTRUDING INTUSSUSCEPTION

*(Drawing made from a Photograph.)*



*Fig. 639.*—COMPLETE PROLAPSE (PROCIDENTIA) OF THE RECTUM. THE EXCESSIVE SIZE OF THE TUMOUR IS DUE TO SEVERAL COILS OF SMALL INTESTINE HAVING BEEN PROTRUDED INTO THE PERITONEAL POUCH. THE PROLAPSE WAS COMPLETELY CURED BY THE OPERATION OF RECTO SIGMOIDECTOMY.

*(Drawing made from a Photograph.)*



practically absent and reflex contraction of the external sphincter muscle is not stimulated by digital exploration. When the rectum has been extruded to the fullest extent of which it is capable, the resulting prolapse forms a cylindrical, oval, or globular tumour. Its size depends upon the duration of the prolapse, the age of the patient, and the severity of the straining effort which produced the protrusion. The mucosa covering the tumour is usually bright red in colour, though, if there is impediment to venous return, a purplish hue is imparted to it. The base of the tumour is situated at the muco-cutaneous junction, the diameter here being often considerably less than that of the widest part of the tumour. The mucosa is as a rule thrown into transverse and parallel folds (fig. 638) unless the peritoneal pouch between the outer and inner tubes contains coils of small intestine which so distend the tumour that the folds of mucosa become effaced (fig. 639). Induration and ulceration of the mucosa is apt to occur from friction against the patient's clothing in cases of long standing. The orifice leading into the interior of the bowel is generally situated at the apex of the tumour or immediately to one side of it. When the tumour is of moderate dimensions, the orifice is usually in the centre of the tumour and somewhat oval in shape, but in those cases in which the protrusion measures several inches in length, the orifice becomes elongated and slit-like.

When the peritoneal pouch contains coils of small intestine the anterior part of the tumour is distinctly resonant to percussion, and when manual pressure is made upon it for the purpose of reducing the protrusion a gurgling sensation is imparted to the hand as the intestine slips back.

*Differential Diagnosis.* Complete prolapse of the rectum must be distinguished from (a) partial prolapse (prolapsus mucosæ recti); (b) intussusception of the rectum protruding through the anus; (c) intussusception of the colon protruding through the anus; and (d) a protruding neoplasm.

The distinguishing features of partial prolapse have already been described. When an intussusception of the rectum protrudes through the anus there is a space between the wall of the rectum and the protrusion into which a finger can be introduced and passed completely round the protrusion. The space is not more than an inch or so deep. In examples of protruding intussusception of the colon, the space between the protrusion and the rectal wall may be several inches in depth according to the site of the commencement of the intussusception.

In both of the above conditions there is an entrance into the bowel at the apex of the protrusion.

A protruding neoplasm is usually attached to the rectum by a definite pedicle which is much smaller in calibre than the growth, and, except in instances in which the growth extends completely round the circumference of the bowel, there is no passage-way into the bowel at its apex.

*Treatment.* The treatment of complete prolapse of the rectum is either palliative or operative. It must be borne in mind that in some instances the prolapse may be due to some definite exciting cause, which, if removed, may be effective in preventing recurrence. Thus the possible existence of phimosis or a vesical calculus should always be sought for in children and appropriately treated. In adults uterine or other pelvic tumours may be causative factors and should receive surgical attention. Similarly, the removal of a rectal polypus may be the means of preventing recurrence of the prolapse if it is the exciting cause.

*Palliative* treatment consists of reducing the protrusion as soon as possible and in preventing recurrence by suitable means. In some cases, in which the onset of the protrusion has been due to a sudden and violent expulsive effort, instant reduction may effect a permanent cure. When, however, the protrusion has been caused by general loss of tone in the musculature of the pelvic floor, recurrence is likely to occur during the slightest straining effort. Under such conditions, especially in children, the protrusion occurs during defæcation or micturition. In children under three years of age an action of the bowels should take place either in the recumbent position or while the child is suspended vertically by the nurse placing her hands in the armpits. The latter posture makes a straining effort impossible. Children over three years of age and adults should adopt the recumbent position whenever an action of the bowels takes place.

It is of the greatest importance that a prolapse of the rectum should be reduced as soon as possible after its occurrence, firstly, because the longer the protrusion is allowed to remain unreduced the more congested the tissues become, thus rendering reduction difficult; and, secondly, because prolonged dilatation of the anal orifice produces weakening of the muscular power of the sphincters.

The best way of reducing a prolapse of the rectum is to place the patient in the right lateral and semi-prone position and then to grasp the tumour firmly between the fingers and thumb of the left hand.

Whilst firm and prolonged pressure is maintained in this way the thumb of the right hand is placed upon the apex of the protrusion and at the same time made to exert pressure in the direction of the longitudinal axis of the bowel. Reduction of the prolapse in this way can generally be accomplished without resort to næsthesia unless there is marked spasm of the sphincters. After the prolapse has been reduced great care should be taken to avoid recurrence. Steps should be taken to avoid either constipation or diarrhœa, both of which give rise to straining at stool. The patient should be given a light nutritious diet, and his general health should be improved by the administration of suitable tonics. Cod-liver oil is especially useful in these cases as it ensures that the motions are soft. Should constipation occur, the injection of an ounce of olive oil into the rectum at bedtime is particularly effective for securing a daily action of the bowels.

Various forms of artificial supports have been from time to time devised, but none of them seem to be satisfactory.

*Operative Treatment.* Operative interference should be advised in cases of long standing, or when palliative treatment has failed or is unlikely to be productive of good results, and also when the bowel continues to prolapse in spite of all efforts to prevent it. It should be borne in mind that, when prolapse of the rectum has taken place, the tendency is for it to increase in size as time goes on, so that if the usual palliative measures are unavailing for the prevention of a recurrence, the sooner operative interference is undertaken the better it will be for ultimate success. In an early stage the protrusion is of small dimensions, and the sphincters are not sufficiently stretched to abolish their contractile power, so that an operation under such circumstances is much more likely to be successful.

Almost from time immemorial the minds of surgeons have been exercised in an endeavour to devise a method of permanent cure. Many methods have been from time to time suggested, but none of them have been universally successful. These methods may be divided into four categories, viz. : (1) narrowing the anal orifice ; (2) contracting the lumen of the rectum ; (3) fixation of the rectum to the sacrum ; and (4) suspension of the rectum from the abdomen, e.g. rectopexy and sigmoidopexy. Of these the method which has been the most successful is fixation of the rectum to the anterior surface of the sacrum (Mummery's operation). This procedure can be relied upon to effect a lasting cure in examples of partial procidentia, but when the whole of the rectum has been extruded, and especially in those in which coils

of small intestine occupy the peritoneal pouch, recurrence of the protrusion generally takes place after a short interval (one to two years).

There is no doubt that the best and safest way of preventing recurrence of the prolapse is to perform left inguinal colostomy, but the majority of patients do not like the idea of having to void their motions in an unnatural manner and prefer to endure the discomforts of the disability. It often happens, therefore, that patients suffering from prolapse of the rectum, when told that an operation is necessary, are never seen again because they imagine that colostomy is intended.

*Narrowing the Anal Orifice.* As the result of the extrusion the anal orifice becomes markedly dilated and offers no resistance to recurrence after reduction. Attempts, therefore, have been made from time to time to create stenosis of the orifice by various means. A method which has found considerable favour among French surgeons is that recommended by Thiersch, who encircles the anus with silver wire introduced subcutaneously. The wire is removed at the expiration of from two to three months, the fibrous tissue which has been deposited around the wire being sufficient to maintain the stenosis after its removal. Several successful cases have been reported.

Some degree of stenosis of the anal orifice can be obtained by removing co-existing internal piles. When internal piles co-exist the probability of narrowing the anal orifice sufficiently to prevent recurrence of the prolapse, at any rate for a considerable time, by ligaturing them is extremely good. At the base of each pile a triangular piece of anal skin is dissected up and included in the ligature. When the sloughs separate, granulating surfaces remain which cicatrise and stenose the orifice.

A similar result may be obtained, in the absence of internal piles, by applying pure nitric acid to the mucosa throughout the circumference for a space of half an inch from the muco-cutaneous junction. The acid should be applied in the following manner. The bowel having been extruded to the full extent, the mucosa above the muco-cutaneous junction is carefully dried. The acid is then applied by means of a glass rod to the mucosa in the form of a band, commencing at the muco-cutaneous junction and extending upwards for half an inch. Immediately after the acid has been applied, the whole of the surface must be carefully dried and sponged with a solution of bicarbonate of soda in order to neutralise the acid. This is very important because if the acid is permitted to remain, the resulting sloughs may be more

extensive and deeper than is desired, and severe hæmorrhage may occur when they separate.

The protrusion is then completely reduced and prevented from recurring by applying a pad and bandage firmly after introducing an ounce of olive oil into the rectum. Under no circumstances should the acid be applied to the apex of the protrusion. If this is done, the resulting cicatrization after the separation of the sloughs will produce stenosis at a level several inches above the anal margin and will produce an intussusception of the rectum. This method of applying nitric acid is much more beneficial in children than in adults.

*Contracting the Lumen of the Rectum.* This is usually effected by linear cauterisation by means of the Paquelin cautery. The patient having been anæsthetised the bowel is made to protrude to its fullest extent, and then four or more longitudinal stripes are made with the cautery, equidistant from one another and extending from the mucocutaneous junction to a point about half an inch from the apex of the protrusion, care being taken not to burn the tissues as deeply in the region of the apex as at the base of the protruded bowel. It is important, when applying the cautery, that large and obviously dilated veins should be avoided. When the cauterisation has been completed the protrusion is carefully reduced and an ounce of olive oil introduced into the rectum.

Another method of narrowing the lumen of the rectum is by removing ellipses of mucosa anteriorly, posteriorly and at the sides, and then suturing the edges together. The objection to it is that very free bleeding takes place from the hæmorrhoidal plexus.

*Fixation of the Rectum to the Sacrum.* The object of this procedure is to fix the portion of the rectum below the peritoneal reflexion to the lower half of the sacrum. A longitudinal incision is made from the middle of the inter-natal cleft down to the posterior margin of the anus. The posterior surface of the rectum is exposed by separating the coccygeus muscles. The prolapse is completely reduced and then the muscular coat of the rectum is sutured to the sacro-sciatic ligaments. The edges of the coccygei are finally sutured together and the wound closed, a small drainage-tube having been inserted in the lower extremity of the wound. This method of fixing the rectum was introduced by Marchant and is known by his name. Lockhart-Mummery dispenses with sutures and achieves fixation to the sacrum by stripping the rectum, encased in its fascia propria, from the anterior surface of the

sacrum posteriorly and from the upper surfaces of the levatores ani laterally and packing the resulting cavity with gauze. He performs the operation in the following manner: A transverse incision about two inches long is made midway between the tip of the coccyx and the posterior margin of the anus. The origin of the external sphincter muscle and the ano-coccygeal ligament is completely divided and the posterior margins of the coccygeus muscles separated until the retro-rectal cellular tissue is exposed. By means of blunt dissection the rectum, encased in its fascia propria, is stripped up from the anterior surface of the sacrum to a point well above the middle of the sacrum. The rectum is also separated from its lateral attachments above the levatores ani. The resulting cavity is then lightly packed with strips of gauze. Finally, the redundant mucosa of the anterior surface of the lower inch of the rectum is dissected up and ligatured as in the ligature operation for partial prolapse. The gauze packing is removed at the end of a week. The cavity is irrigated twice daily with an antiseptic solution and repacked. The repacking is continued for a month so as to delay the closing of the cavity. The wound heals from the bottom by the process of granulation, and eventually the posterior and lateral surfaces of the rectum become firmly fixed to surrounding structures. This procedure gives excellent results in those cases of complete prolapse in which loops of small intestine do not occupy the peritoneal pouch.

*Suspension of the Rectum from the Abdomen.* By this procedure the terminal portion of the pelvic colon is fixed to the left postero-lateral wall of the false pelvis. The operation is known as sigmoidopexy or pelvic colopexy, and was first described by Lenormant. The patient is placed in a high Trendelenburg position and the abdomen opened by a left paramedian incision extending from the symphysis pubis to an inch above the level of the umbilicus. Traction is made upon the pelvic colon until the prolapse has been completely reduced. An incision is then made through the peritoneum on the outer aspect of the pelvic colon, about three inches long, parallel and externally to the left ureter. The pelvic fascia over the ilio-psoas muscle is exposed and the external longitudinal band of the terminal pelvic colon is sutured to the fascia by a series of sutures introduced at intervals of a quarter of an inch from above downwards. The sutures should not be tied tightly lest they should cut out before firm adhesion has taken place. The outer edge of the peritoneum is then sutured to the colon by means of a continuous catgut suture. Finally the abdomen is closed in the usual way.

Of all the methods which have been advocated for the surgical treatment of complete prolapse of the rectum, none affords such uniformly good results as amputation of the protruded bowel. By this means not only is the whole of the rectum excised, but the extruded portion of the pelvic colon is removed as well. The operation is called *recto-sigmoidectomy* (author's operation).

*Technique of the Operation.* The patient having been anaesthetised—the best anaesthetic for the purpose being intrathecal percaine (1 cc. of 1-200 solution) combined with gas-and-oxygen inhalation—is placed in the combined lithotomy and Trendelenburg position. The rectum having been protruded to its full extent, the apex of the protrusion is grasped by a pair of Lane's tissue forceps to prevent retraction. Commencing anteriorly half an inch from Hilton's white line, a longitudinal incision two and a half inches long is made through the mucosa, and then, by blunt dissection, the mucosa is separated from the underlying muscular coat as far laterally as possible on either side. At the lower angle of the incision through the mucosa a pair of Kocher's forceps is placed transversely on the mucosa, on either side, so as to control bleeding from the haemorrhoidal vessels.

At the level of the upper extremity of the incision the mucosa is divided transversely on either side as far as the lateral borders of the protruded bowel, thus exposing the muscular coat of the rectum. The posterior aspect of the protrusion is then dealt with in a similar manner so that the muscular coat of the rectum is denuded of mucosa throughout its circumference. The muscular coat is now incised longitudinally on its anterior aspect by an incision corresponding in length to the initial incision through the mucosa (fig. 640). The object of commencing this incision half an inch from Hilton's white line is to prevent damage to the external sphincter muscle, and especially to the point of fusion between the levator ani and the external muscular coat of the rectum, upon which depends subsequent control over the contents of the bowel.

The peritoneal pouch, which always exists in cases of complete prolapse, is thus opened, exposing the pelvic colon to view, which, though free anteriorly and at the sides, is attached posteriorly by the vascular pedicle of its mesentery. Should this pouch contain omentum or coils of small intestine, they are returned into the abdominal cavity and kept there by means of a suitable swab.

The muscular coat of the rectum is now divided transversely, throughout its circumference, at the level of the upper angle of the

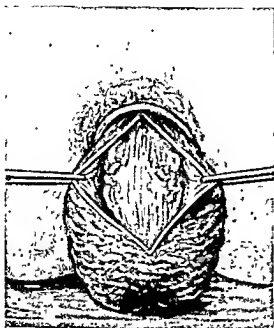


Fig. 640.—EXPOSING THE PELVIC COLON, FORMING THE INNER TUBE OF THE PROCTOSTOMA, BY INCISING THE MUSCULAR COAT OF THE RECTUM LONGITUDINALLY.

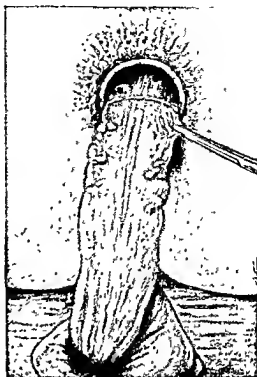


Fig. 641.—THE RECTUM HAS BEEN COMPLETELY DIVIDED TRANSVERSELY HALF AN INCH FROM THE MUCCO-CUTANEOUS JUNCTION AND PEELED DOWNWARDS EXPOSING THE PELVIC COLON. THE INTERRUPTED LINE MARKS THE LEVEL OF THE AMPUTATION OF THE BOWEL.

longitudinal incision, when it will be found that the rectum can be peeled downwards (fig. 641). All bleeding points are secured by forceps and ligatured. The peritoneal pouch is shut off from the general peritoneal cavity by suturing the cut edge to the anterior and lateral surfaces of the pelvic colon after withdrawing the swab which may have been

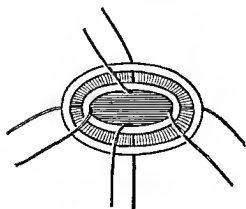


Fig. 642.—SHOWING THE CUT END OF THE PELVIC COLON BEING UNITED TO THE STUMP OF THE RECTUM BY FOUR POINTS OF SUTURE THROUGH THE MUSCULAR COATS.

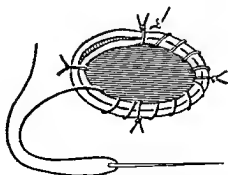


Fig. 643.—SHOWING THE MUCCOSA UNITED BY A CONTINUOUS SUTURE.



introduced to maintain coils of small intestine within the pelvis. When this has been done, the vascular pedicle of the pelvic mesocolon is ligatured half an inch below the point where the pelvic colon is to be cut across. The pelvic colon is then completely divided at a point at least half an inch above the level of the ligature upon the pelvic mesocolon, thus liberating the rectum and the protruded portion of the pelvic colon. The cut margin of the pelvic colon is then sutured to the stump of the rectum by means of interrupted sutures of catgut, first muscular coat to muscular coat and then mucosa to mucosa (figs. 642, 643). Finally, the index finger is carefully inserted into the lumen of the pelvic colon in order to ascertain that it has not been unduly stenosed by the sutures, and then a drainage-tube is inserted to facilitate the avoidance of flatus.

*Post-operative Treatment.* The bowels should be kept confined for four or five days and then opened by an olive oil enema. Strong aperients should be avoided until the suture line is quite sound. At the expiration of ten days, and daily afterwards, the index finger is passed through the junction to obviate constriction of the lumen during the healing process.

*Mortality After the Operation.* It might be assumed that, because the peritoneal pouch is freely opened during the operation, there is considerable risk of septic peritonitis supervening as the result of *B. coli* infection. Curiously enough such a complication never seems to arise despite the fact that the mucosa of the rectum is exposed and cannot be sterilised. Out of thirty-four operations performed by myself there has only been one death. The fatal result in that case was due to strangulation of a loop of small intestine by a band of adhesion.

*End Results.* These, so far as my personal experience goes, have been remarkably good. There has only been one instance of recurrence, which occurred five years after the primary operation. Amputation of the prolapsed bowel was again resorted to. The portion of the pelvic colon which was excised contained several diverticula, each of which contained a small mass of faeces. The narrowing of the lumen caused by the diverticula accounted for the recurrence of the prolapse.

## CHAPTER V

### FISTULA IN ANO

THE term fistula denotes an abnormal communication between the skin surface and the interior of a hollow viscus, or between the skin surface and the interior of a duct, or between two adjacent hollow viscera. The proctologist is concerned only with the variety known as *ano-rectal fistulæ*.

#### ANO-RECTAL FISTULÆ

The rectum is frequently the seat of fistulous communications, which are referred to as fistulæ in ano—a bad term because it implies implication of the anus only—or as ano-rectal fistulæ, a much better term, expressing implication of not only the anus but the anal canal and the rectum as well. The surface apertures of ano-rectal fistulæ are located in the perineum, but it must not be assumed that every surface aperture of a fistulous track encountered in the perineum is necessarily that of an ano-rectal fistula. For instance, in the anterior, lateral, and posterior regions of the perineum surface apertures may exist (fig. 644) which lead into fistulous tracks that have no connection with the rectum whatever. Thus, a surface aperture situated in the anterior part of the perineum, in or near the middle line, is probably that of a urethral fistula, the result of a peri-urethral abscess due to the presence of stricture of the urethra. A surface aperture in the lateral part of the perineum, especially when in proximity to the transverse anal line at a distance of not less than two inches from the middle line, is probably the point of evacuation of a pelvi-rectal abscess. An abscess of this kind is the result of suppuration originating in the parametrium, the base of the bladder, the prostate, or the vesiculæ seminales, and occupies primarily the superior pelvi-rectal space of Richet. The pelvi-rectal space is shut off from the para-rectal space by the fascia propria of the rectum (fig. 645), by which the pus is prevented from being discharged into the rectum. Though originally situated above the levatores ani, the pus extends through the latter and invades the ischio-rectal fossa, ultimately finding an exit through an opening upon the skin surface.

Again, a surface aperture situated in the posterior part of the perineum in the middle line, between the tip of the coccyx and the anal margin, may be the point of issue of a suppurating dermoid. Such dermoids usually develop at the site of a post-anal dimple and often contain hair. When a surface aperture is situated laterally to and about an inch distant from the tip of the coccyx, especially when a second opening is symmetrically placed on the opposite side, the apertures are probably the vents of a pre-sacral abscess. These abscesses are the result of suppuration of the pre-sacral lymphatic glands. The pus is situated between the sacrum and the fascia propria of the rectum. The latter effectually prevents the pus from being discharged into the

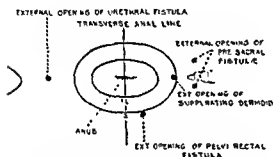


Fig. 644.—SHOWING THE POSITION OF SURFACE OPENINGS OF FISTULÆ HAVING NO CONNECTION WITH THE RECTUM.

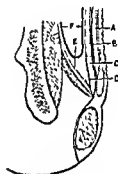


Fig. 645.—SHOWING THE PELVI-RECTAL AND PARA-RECTAL SPACES.

A—External muscular coat.  
B—Internal muscular coat  
C—Para-rectal space  
D—Fascia propria.  
E—Pelvic fascia.  
F—Peritoneum.

rectum, so that it gravitates downwards and invades the posterior part of the ischio-rectal fossa by passing through the interval between the coccygeus and the levator ani to find an exit on one or on both sides of the ano-coccygeal ligament. An aperture situated at or about the middle of the inter-natal furrow may be the surface opening of a canal lined by epithelium which communicates with the interior of a cyst containing hair, the so-called pilonidal cyst.

Lastly, a surface aperture situated close to the middle line, at the level of the upper extremity of the inter-natal cleft, indicates the existence of a sacro-coccygeal sinus. A sinus in this situation is generally supposed to lead down to carious bone, but, so far as my experience goes, it never does, nor has it any connection with the rectum. It is the result of a hæmatoma caused by a fall upon the

buttocks. The aperture leading into the sinus is almost invariably situated on the left side within half an inch of the upper extremity of the inter-natal cleft, a circumstance that is probably accounted for by the fact that right-handed people fall more heavily upon the left side.

With the exception of the above, and also of those leading down to carious bone in the neighbourhood of the tuberosity of the ischium, the sacrum, or the coccyx, all other surface apertures of fistulous tracks found in the perineum are those of ano-rectal fistulæ.

*Ætiology of Ano-Rectal Fistulæ.* Fistulous communications between the skin surface of the perineum and the interior of the anal canal or rectum are occasionally established by penetrating wounds from without, or from extravasation of faecal material into the peri-rectal tissues as the result of perforation of the rectal wall by a carcinomatous growth or by an ulcer in the vicinity of a fibrous stricture. Such ano-rectal fistulæ pursue an atypical course which is entirely dependent upon trauma or pre-existing disease. With these exceptions every ano-rectal fistula is preceded by an abscess which is the outcome of infection conveyed to the tissues, in which the abscess forms, chiefly by means of the lymphatics.

In the majority of cases the infective process starts in a lesion situated either in the anal canal or in the lower half of the rectal ampulla. The lesions usually responsible are: a thrombosed internal pile, extravasation of blood from a ruptured vein in the submucosa, a fissure at the anal margin, a torn-down anal valve, laceration of the pedicle of a polypus, an ulcerated surface in a sinus of Morgagni and puncture or laceration of the mucosa, either by small fragments of bone which have been swallowed, or by the needle of a syringe employed in the treatment of internal hæmorrhoids by the injection method. There is no doubt also that occasionally peri-rectal suppuration is due to micro-organisms gaining access to the tissues by means of the blood stream.

Many years ago very opposite opinions upon this point were held by two eminent contemporaries, Sir Benjamin Brodie and Mr. Syme, both of whom evidently entertained a leaning towards proctology. Brodie confidently asserted that fistula-in-ano was due to the existence of an ulcer in the mucous membrane, and that the suppuration in the neighbouring tissues was subsequent to and dependent upon the ulceration. Mr. Syme, on the other hand, asserted with equal confidence that the inflammation and suppuration took place in the tissues external to the bowel and that the abscess subsequently opened into

the rectum. No doubt Mr. Syme was mindful of the prevalence of ischio-rectal abscesses among carpenters and stone-masons from sitting upon damp and cold seats, and among cavalymen from riding upon wet saddles.

Nowadays, by the aid of blood cultures, we know that micro-organisms circulate in the blood stream under certain conditions and, finding a suitable nidus in low resisting fatty tissue, may give rise to suppuration. Just in the same way as a perinephric abscess may result from staphylococcal infection originating in a suppurating furuncle of the scalp, so may an ischio-rectal abscess be consequent upon dental pyorrhœa or other distant septic focus.

The sequence of events, therefore, leading up to the establishment of an ano-rectal fistula is, in the majority of instances, as follows: (a) Appearance of an initial lesion; (b) septic lymphangitis or phlebitis, as the result of infection by micro-organisms; (c) suppuration eventuating in the formation of a localised abscess; (d) evacuation of the pus either into the bowel or through the skin, or in both situations, thus giving rise to (1) an incomplete, or (2) a complete fistula.

*Classification of Ano-Rectal Fistulæ.* It is usual to divide ano-rectal fistulæ into three varieties: (a) the complete fistula; (b) the blind external fistula; (c) the blind internal fistula.

My objection to the above universally adopted classification of ano-rectal fistulæ is that it conveys the impression that they are varieties of a single type differing only in degree, and therefore equally amenable to treatment by the simple surgical procedure of laying open the fistulous track completely from the external opening to the internal opening and allowing the resulting wound to heal progressively from the bottom. There is no doubt that many fistulæ can be completely cured by this simple procedure, and yet it frequently happens that, when carried out in some instances, disastrous results ensue, on account of loss of control over the anal orifice. The explanation of these divergent results is that there are several types of fistulæ differing in the anatomical relationship of the main track to the various muscles controlling the anal outlet. Every fistula of inflammatory origin is preceded by an abscess, and the main track of the fistula is the contracted but unobliterated original abscess cavity. The anatomical site of the original abscess, therefore, determines the type of the fistula.

The abscesses which precede fistula formation are the result of a septic focus in the terminal portion of the rectum, which extends by means of the lymphatics. The lymphatic system of the rectum consists

of two plexuses, the submucous and the intermuscular, the efferents from which communicate with (1) the lymph sinus of the rectum, a space existing between the external muscular coat and the peri-rectal fatty tissue, and (2) the subcutaneous peri-anal lymphatics. The efferents from the anal canal also pass through the fatty tissue of the ischio-rectal fossa on their way to Alcock's canal. As the result of sepsis within the rectum an abscess may form in any of the above situations, and since the anatomical site of the abscess determines the type of the resulting fistula, the following classification of ano-rectal fistulæ seems to be anatomically advisable :

- |                          |                                      |
|--------------------------|--------------------------------------|
| (A) The subcutaneous.    | (1) blind external.                  |
|                          | (2) blind internal.                  |
|                          | (3) complete.                        |
| (B) The submucous.       | (1) blind external.                  |
|                          | (2) blind internal.                  |
|                          | (3) complete.                        |
|                          | (4) bilateral.                       |
| (C) The intermuscular.   | (1) blind internal.                  |
| (D) The para-rectal.     | (1) blind internal.                  |
|                          | (2) complete.                        |
|                          | (3) bilateral.                       |
| (E) The sub-sphincteric. | (1) blind internal.                  |
|                          | (2) complete.                        |
|                          | (3) bilateral (anterior horseshoe).  |
| (F) The ischio-rectal.   | (1) blind external.                  |
|                          | (2) blind internal.                  |
|                          | (3) complete.                        |
|                          | (4) bilateral (posterior horseshoe). |

#### FEATURES OF INTEREST PRESENTED BY A COMPLETE ANO-RECTAL FISTULA

A complete ano-rectal fistula presents four features of interest, namely : (1) The external opening ; (2) the internal opening ; (3) the main track ; and (4) the offshoots or extensions from the main track.

(1) *The external opening.* A great deal of information can be obtained in regard to the probable type and nature of a fistula by

observing the size, position, and number of the fistulous apertures to be seen in the perineal region. As a general rule, if the opening is small and contracted, and especially if it should be situated within an inch of the anal verge, the fistula is probably subcutaneous in type. Should the opening be large, irregular in shape with undermined edges, and particularly if the surrounding skin is of a reddish-purple hue, the fistula is most probably tuberculous in origin. When the opening is surmounted by a tuft of granulation tissue such as is often observed at the orifice of a sinus leading down to carious bone, a deeply-seated fistula, such as the ischio-rectal or the para-rectal, is indicated. Should the opening be situated close to the anal verge or within half an inch of it, and a probe passes deeply in a direction parallel to the long axis of the bowel, the fistula is invariably of the submucous type.

Lastly, the presence of several external openings is an indication of the existence of offshoots from the main track of a deeply-situated fistula, such as the ischio-rectal or the para-rectal.

Although a great many ano-rectal fistulae have only one external opening, multiple openings are the rule rather than the exception in those of the ischio-rectal type. In these there is only one internal opening, situated in the middle line posteriorly, opposite the interval between the sphincters, and is often of large size. When the internal opening is large, sometimes large enough to admit the tip of the index finger, faecal matter is apt to enter it when an action of the bowels takes place, thus provoking acute inflammation along the main track of the fistula. As the result, then, of septic lymphangitis, secondary abscesses are formed in the vicinity of the main track. These abscesses eventually extend to the skin surface and open there, forming secondary openings. The original external opening is known as the primary opening, and indicates the termination of the main track of the fistula. Openings in connection with the same fistula which appear subsequently are secondary openings and represent the surface terminations of offshoots from the main track. It must not be forgotten, however, that external openings may not always be connected with the same fistula. It sometimes happens that two or more separate fistulae exist in the same patient, each having a separate external opening. Instances occasionally occur in which two, three, or four separate complete fistulae co-exist in the same patient.

(2) *The internal opening* is always located in the interior of the bowel, and is due to perforation of the mucous coat. As a rule, internal openings are of small size and are generally circular in outline, but

occasionally they are much larger and of irregular shape, as, for example, when they are the result of lacerations of the mucosa, such as might be produced by partial tearing through of the pedicle of a polypus, the tearing down of a valve of Morgagni, or an abrasion caused during the passage of a foreign body, such as a fish-bone. When an internal opening is exceptionally large, the fistula is usually tuberculous in origin.

The position of an internal opening varies according to the type of the fistula with which it is associated. Thus, the internal opening of a para-rectal fistula is generally situated above the level of the levatores ani, at a distance from two to three inches above the anal margin. That in connection with a fistula of the submucous type may be situated anywhere along the course of the main track, but is generally found immediately above Hilton's white line. The internal opening of a sub-sphincteric fistula is always situated in the anal canal in one of three positions at the level of the valves of Morgagni, namely, either in the right anterior quadrant or in the left anterior quadrant, or in the middle line posteriorly. The internal opening of an ischio-rectal fistula is always situated in the middle line posteriorly opposite the interval between the external and internal sphincters. The internal opening of the intermuscular type may be situated anywhere in the circumference of the anal canal, but always at the level of the interval between the sphincters.

In the majority of cases there is only one internal opening to each complete or blind internal fistula, but occasionally there are two. When a second internal opening, in connection with the same fistula, exists it is generally situated at a higher level along the course of the submucous track extending vertically or obliquely upwards from the lower opening. When more than one internal opening is discovered at the same level, each opening belongs to a separate fistula. If there is an internal opening at the level of the interval between the internal and external sphincters, and a second opening is found higher up without an intervening submucous track between them, both are internal openings of a para-rectal fistula.

The position of the external opening of a fistula, as regards the distance from the anal margin and its relation to the transverse anal line, is sometimes an indication to the position of the internal opening. This valuable and practical observation was made by Goodsall, of St. Mark's Hospital, and became known as Goodsall's rule. According to his observations, if the primary external opening is situated from an inch to an inch and a half distant from the anal margin, and either



anteriorly to or on the transverse line, the internal opening will be found opposite the interval between the sphincters in the same radial line as the external opening, and the main track will be straight. Secondly, when the external opening is a similar distance from the anal margin but situated posteriorly to the line, the internal opening will be found in the middle line posteriorly opposite the interval between the sphincters, and the main track will take a curved course. Lastly, if the external opening is at a greater distance than an inch and a half from the anal margin, either anteriorly or posteriorly to the line, then the internal opening is always to be found in the middle line posteriorly opposite the interval between the sphincters, and the main track takes a curved course backwards (see fig. 646, D, E). The rule does not apply to the majority of fistulae but only to certain types, which accounts for its not having received the recognition which it deserves. According to my own observations it is only applicable to the sub-sphincteric and the ischio-rectal types of fistulae, representing rather less than 30 per cent of the total number; but for these it is an accurate and valuable guide when an operation is being performed.

(3) *The main track* extends from the internal opening to the primary external opening, and may take a straight, curved, or tortuous course. Its anatomical position in regard to the coats of the terminal portion of the rectum and of the tissues in its immediate vicinity determines the type of the fistula. The exact position of the main track of the fistula in regard to the muscular apparatus controlling the outlet of the rectum is of the utmost importance from the point of view of surgical treatment. Failure to recognise that all fistulae are not of the same type is responsible for the disastrous consequences that sometimes result from operative treatment.

(4) *Offshoots or extensions from the main track.* Whenever there is a free discharge of pus, either from the internal opening or from the primary external opening, the formation of an offshoot from the main track is indicated. Repeated attacks of suppuration lead to the formation of multiple offshoots from the main track so that, in the course of time, a labyrinth of inter-communicating channels is formed. Some types of fistula are more prone to ramification than others. All fistulae are at first unilateral, but secondary offshoots may extend across the middle line so that the fistula ultimately becomes bilaterally disposed. The offshoots from the main track are not necessarily confined to the same anatomical locality; thus, the secondary tracks

in connection with an ischio-rectal fistula may be entirely located in the subcutaneous tissue, a complex fistula resulting to which the designation "fistula of the ischio-rectal type with subcutaneous extensions" should be applied. Again, an offshoot from an ischio-rectal fistula may be located in the submucous tissue, when an "ischio-rectal fistula with submucous extension" results. It is important, therefore, when operating upon different types of fistulæ, to bear in mind the possibility of offshoots existing in lymphatic areas other than that in which the main track is situated.

An offshoot from a unilaterally situated fistula when extending across the middle line to the opposite side, by reason of the fact that the lymphatics on both sides are symmetrically disposed, follows an exactly similar course to that taken by the main track of the original fistula, and is therefore a replica of it. The extension simulates the disposition of the original fistula, terminates in an external opening in a corresponding position to that occupied by the primary opening, and gives rise to offshoots following a similar course to those taken by the offshoots from the original main track. The type of fistula which most commonly extends to the opposite side is the ischio-rectal, though the submucous type may occasionally do so.

#### GENERAL PRINCIPLES OF TREATMENT

Whenever a fistulous communication with the interior of the anal canal or rectum has been established, spontaneous cure seldom results. The treatment, therefore, of an ano-rectal fistula is mainly operative, though in certain circumstances palliative measures may be adopted.

*Palliative Treatment.* This, though it may sometimes succeed in arresting progressive extension, rarely effects a permanent cure. It should, however, be always adopted in cases in which a patient's constitutional condition contra-indicates operative interference. Local treatment, such as the use of warm sitz-baths and the application of fomentations to the perineum, is advisable. Daily evacuation of the bowels should be ensured by the use of mild aperients, but strong purgatives should be avoided, as violent peristalsis might force liquid fæces into the internal opening and set up active suppuration in the main track.

*Operative Treatment.* The surgical treatment of a fistulous track is comparable to the conversion of a subterranean tunnel into an open

trench by digging away the earth covering it from end to end, and then filling in the trench with new material until it is level with the surrounding surface. By adopting this principle the tissues overlying the main track of a fistula, from the primary external opening to the internal opening, are divided so that an open wound is created which is made to heal progressively from the bottom, and so efface the fistula. Offshoots extending into the surrounding tissues are dealt with in a similar manner.

The principle underlying the surgical treatment of a fistula is to lay open the main track from end to end, together with all the offshoots extending from it. It is possible to efface all fistulæ in this way, but whether a satisfactory result is obtained or not depends upon the damage that may have been inflicted upon the muscular apparatus controlling the anal outlet.

Although the majority of ano-rectal fistulæ communicate with the interior of the bowel by means of an internal opening, in only a small percentage of them is the muscular coat penetrated by the main track, but in these the laying open of the main track from end to end results in serious impairment of the muscular control of the anus. If all ano-rectal fistulæ were of the same type, the simple procedure of laying open the main track from end to end, together with all offshoots from it, would produce equally good results. Unfortunately, however, in some instances permanent loss of muscular control ensues. The explanation of this is that the anatomical relationship of the main track to the musculature of the anal outlet varies in different types of fistulæ.

The position of the internal opening can usually be taken as an indication of the point where the main track passes through the bowel wall. The usual position of an internal opening is at the level of the interval between the internal and the external sphincter, which is approximately at Hilton's white line, but in some instances it may be found at a much higher level—for example, quite two or three inches above the anal margin. A high-lying internal opening does not, however, always indicate that the main track of the fistula penetrates the muscular wall of the bowel at a corresponding level.

A great deal of controversy has centred round this point. Some authorities state that in their experience no harm results from laying open the main track of a fistula which has a high-lying internal opening, whereas others, of considerable experience, aver with equal conviction that in similar circumstances permanent loss of control inevitably results. When two such divergent opinions are expressed by those

who have had considerable experience in the operative treatment of ano-rectal fistulæ it is obvious that there must be some explanation to account for the discrepancy. The truth is that neither group of observers has realised the fact that ano-rectal fistulæ differ widely in type, so that whereas both are right in the opinion expressed, both are also wrong.

Let us take, for example, the case of a submucous fistula having a high-lying internal opening such as is represented in figure 646. The main track of such a fistula is, throughout its course, superficial to the muscular coats of the bowel. Laying open the main track of such a fistula into the cavity of the bowel cannot possibly inflict any injury to the musculature and therefore no impairment of control can possibly result. Again, in the case of an ischio-rectal fistula with a submucous extension having an internal opening at the extremity of the submucous portion of the track, as is shown in figure 647, laying open the track from the external opening to the internal opening into the cavity of the rectum only inflicts injury to the external sphincter, from which it is common knowledge that impairment of control does not result.

It is obvious, therefore, that those who hold the opinion that the laying open of a fistulous track, having a high-lying internal opening, may be carried out without inflicting impairment of control must have been fortunate enough to encounter fistulæ of the above types. On the other hand, those who have experienced the disastrous consequences of permanent loss of control have probably been unfortunate in encountering fistulæ of the type shown in figure 648 during the early stages of their experience. It is apparent from a glance at the illustration that laying open the main track of that type of fistula (the para-rectal) into the cavity of the bowel would result in complete division of both sphincters, both muscular coats of the rectum as far as the site of the internal opening and, what is more important still, the point of fusion of the levator ani muscle with the external muscular coat of the rectum. The fibres of the levatores ani radiate from the periphery of the pelvis to the rectum, much in the same manner as do those of the ciliary muscle towards the pupil. Contraction of the ciliary muscle dilates the pupil, and contraction of the levatores ani

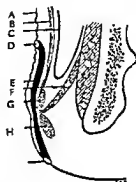


Fig. 646.—COMPLETE SUBMUCOUS FISTULA.

- A—Mucosa.
- B—Muscular coat.
- C—Pelvic peritoneum.
- D—High-lying internal opening of fistula.
- E—Main track of fistula.
- F—Levator ani.
- G—Internal sphincter.
- H—External sphincter.
- I—External opening.

expands the anal canal. When the margin of the pupil is divided, a V-shaped gap is produced by the unopposed contraction of the radiating fibres of the ciliary muscle. Exactly the same thing takes place when the point of fusion between the levator and the external muscular coat of the rectum has been divided. A V-shaped gap is thereby produced owing to retraction of the edges of the wound and hopeless incontinence is the result.

Division of the internal sphincter is generally regarded as the cause of loss of control after fistula operations, but I have not found it to occur even after complete division of the muscle when laying open the main track of an intermuscular fistula. Some observers regard division of the external sphincter as the causative factor of impairment of

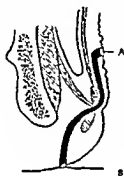


Fig. 647.

Fig. 647.—RECTO-RECTAL FISTULA WITH SCHMECKE'S EXTENSION.

A—Internal opening.

B—External opening.

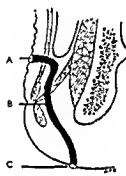


Fig. 648.

Fig. 648.—COMPLETE PARA-RECTAL FISTULA.

A—Internal opening

B—Main track.

C—External opening.

control after operations for fistula, but these have evidently overlooked the fact that the external sphincter is generally completely divided during an operation for fissure without ill effect. The position of the internal opening of a fistula is of very little consequence in determining the nature of the operation to be performed, but the anatomical situation of the main track in its relation to the musculature of the anal outlet is of the greatest importance.

An important principle involved in the operative treatment of a fistula is that the square area of the surface wound should be made at least twice as great as the square area of the rest of the wound. The most satisfactory way of increasing the surface area of a linear wound is to make incisions through the skin and subcutaneous tissue at right angles to the line of the main incision (fig. 649). I call these secondary incisions "relieving cuts." They should be made at points where there appears to be greatest tension along the margins of the main incision.

No definite rules can be laid down in regard to their exact position or as to the number of incisions required, much depending upon individual experience as to the manner in which the wounds heal, but, provided that the surface area is increased to the requisite extent, the fewer incisions required the better the ultimate result will be.

Increase of the surface area of a wound may also be effected by removing healthy skin around the wound made by laying open the main track of the fistula and the offshoots from it. The objection to this procedure is that the wound takes longer to heal, and the deficiency of skin resulting from extensive removal is replaced by dense scar tissue which may interfere with the expansion of the anus during defaecation.

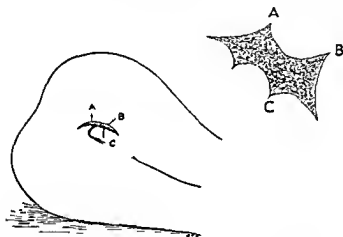


Fig. 649.—SHOWING RELIEVING CUTS, A, B AND C, ALONG MARGINS OF LINEAR WOUND.

Top right hand fig shows increase of surface area resulting from incisions at A, B and C.

When operating upon fistulae lying at a great depth, such as those of the para-rectal type, the area of the whole wound may be so extensive that it is not possible to increase the area of the surface wound sufficiently, on account of lack of room. Under these circumstances the surface wound will be healed long before the remainder of the wound has been completely filled in. Nevertheless, the depth of the original wound will have been diminished considerably, in most instances by more than half of its original extent. In order to permit the remainder of the wound to be completely filled in, the surface wound should be reopened to the requisite extent.

When dealing with this type of fistula, the possibility of a second operation becoming necessary, in order to ensure complete effacement of the fistulous track, should never be lost sight of, and the patient should be warned of this beforehand.

GENERAL PRINCIPLES IN THE TECHNIQUE OF AN OPERATION UPON A  
TYPICAL COMPLETE FISTULA

For an operation for fistula, the most suitable position of the patient is the lateral and semi-prone, on the side on which the primary external opening exists, except when the fistula is situated in the anterior part of the perineum, where the operation field is most conveniently exposed by the lithotomy posture.

First of all, the exact position of the internal opening is determined by digital exploration of the anal canal, and then the course taken by the main track and the extent of the offshoots from it are carefully mapped out by grasping the peri-rectal tissues between the index finger inside the rectum and the thumb upon the skin surface of the ischio-rectal fossa. The point of a probe-director is then introduced into the main track through the primary external opening.

It sometimes happens that the orifice is so contracted that difficulty is experienced in inserting even the smallest probe into it. In these circumstances the orifice should be enlarged by a small incision at right angles to the line of the main track.

As soon as the probe-director has fairly entered the main track, it should be gently passed along it in a direction towards the internal opening. No force should be used, lest a false passage be made.

When the main track takes a straight course no difficulty is experienced in introducing the probe as far as the internal opening, where the point can readily be made to protrude through it. In those cases, however, in which the main track takes a curved course (see fig. 668), some difficulty may be encountered in reaching the internal opening on account of the inability of the probe to adapt itself to the curves of the track.

Accordingly, in order to avoid making a false passage, the point of the probe should be forced through the skin at the spot where its further progress has been arrested. The tissues overlying the probe are then divided, thus exposing a portion of the track. The probe is then re-introduced into the track and passed along it until its point protrudes through the internal opening. Should the passage of the probe be again impeded by a curve in the track the manœuvre above described must be repeated.

As soon as the probe-director has entered the anal canal its point should be hooked down by the index finger and brought out through the anus. The overlying tissues, including the external sphincter muscle in all cases in which the track passes beneath that muscle, are then

divided completely so as to liberate the probe. The floor of the main track has thus been exposed to view in its entirety and can be readily recognised by the granulation tissue lining it.

A careful search is then made along the floor and sides of the exposed track for apertures leading into offshoots. When such an offshoot is found the probe should be passed along it to its extremity and forced through the skin at that point, should it terminate in a cul-de-sac. When all existing offshoots have been laid open, relieving cuts, in order to increase the surface area of the wound to the required extent, should be made at points where they are considered to be necessary.

Finally, all overhanging or undermined edges of skin should be removed and the entire wound carefully packed with strips of gauze and cotton wool.

#### GENERAL PRINCIPLES OF AFTER-TREATMENT

The after-treatment of a fistula wound is exceedingly important. A perfectly successful operation may fail to effect a cure if the details of after-treatment are not attended to. The primary object to be obtained is that the wound resulting from the operation should heal progressively from its deepest part throughout its entire extent.

When the wound is superficial there is no difficulty, and the healing process proceeds without interruption until the granulation tissue becomes level with the surrounding skin and the surface is completely covered by epithelium. In those cases, however, in which the main track lies at a considerable depth and pursues a curved course, the surfaces of the more superficial parts of the wound are apt to come into contact with one another and adhere, unless kept apart. In this way the deepest part of the wound may escape obliteration, and the main track of the fistula is re-established by the process known as "bridging." In order to prevent bridging, the surfaces of the deeper portions of the wound should be kept apart by interposing a thin layer of cotton wool or a strip of gauze between them. On no account should the wound be tightly packed. The only occasion when a fistula should be tightly packed is just after the operation has been completed—never afterwards. Tight packing not only impedes the healing process but prevents the formation of granulation tissue, with the result that the wound ultimately heals with a depressed scar, which, especially when it traverses the anal orifice, is one of the commonest causes of impaired control after operations for fistula.



A fistula wound which is healing healthily is practically free from suppuration. Whenever pus wells up in the wound it is an indication that one of three conditions is present: (1) bridging has occurred at some part of the wound; (2) an offshoot has escaped detection during the operation; or (3) an offshoot, which did not exist at the time of the operation, has developed subsequently as a consequence of infected tissue having broken down. Whenever, therefore, an excess of pus is found to exist, the wound should be carefully explored with a probe in order that the exciting cause of the suppuration may be ascertained and dealt with by a secondary operation without delay.

#### THE SUBCUTANEOUS TYPE OF ANO-RECTAL FISTULA

The subcutaneous fistula is entirely confined to the subcutaneous tissue in the immediate vicinity of the anus, both the main track and any existing offshoots from it being usually superficial to the deep fascia and to the external sphincter muscle. The abscess preceding its formation is generally due to suppuration occurring in a localised hæmatoma occasioned by the rupture of a dilated circum-anal vein (sometimes called a thrombotic pile), a suppurating circum-anal sebaceous follicle or to septic thrombosis or lymphangitis originating in a fissure. The resulting fistula is never extensive, is invariably unilateral, and presents three varieties:

(a) *The blind external subcutaneous fistula* is nearly always due to a suppurating circum-anal hæmatoma, though occasionally it may be of tuberculous origin. The external opening is usually small and often difficult to find when situated in a sulcus between adjacent anal rugæ. The main track, often not more than half an inch in length, is directed towards the anal margin and is entirely superficial to the external sphincter muscle. This variety may exist at any part of the anal circumference.

(b) *The blind internal subcutaneous fistula* (fig. 650) is invariably associated with a fissure, and is due to septic lymphangitis. The internal opening is situated at the lower edge of the fissure and can usually be displayed when the anal margin is everted. The main track radiates in a straight line from the fissure and is generally situated close to the middle line and nearly always on the right side of it. There are no offshoots from the main track.

(c) *The complete subcutaneous fistula* (fig. 651, C, D) is a later stage of the preceding variety. The external opening is usually situated

about one inch from the anal margin. The internal opening is situated at the lower margin of the fissure and is generally larger than in the blind internal variety. On this account faecal matter is apt to pass into the track when the bowels act, and set up active suppuration. Repetition of the inflammatory process may lead to the formation of an offshoot which is invariably single and extends beyond the primary opening in the same radial line as that taken by the main track. A secondary opening may then appear at the extremity of the offshoot.



Fig. 650.



Fig. 651.

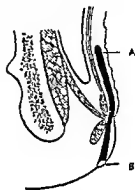


Fig. 652.

Fig. 650.—BLIND INTERNAL SUBCUTANEOUS FISTULA AND COMPLETE SUBMUCOUS FISTULA HAVING AN INTERNAL OPENING IN A FISSURE.

- A—High-lying internal opening of submucous fistula.
- B—Internal opening common to submucous and subcutaneous fistula.
- C—Cul-de-sac of subcutaneous fistula.

Fig. 651.—COMPLETE SUBCUTANEOUS FISTULA WITH SUBMUCOUS EXTENSION RESULTING FROM A FISSURE.

- A—Cul-de-sac of submucous fistula.
- B—Internal opening of the same.
- C—Internal opening of subcutaneous fistula.
- D—External opening of the same.

Fig. 652.—BLIND EXTERNAL SUBMUCOUS FISTULA.

- A—Cul-de-sac of submucous track.
- B—External opening.

Whenever active suppuration occurs in the main track of a subcutaneous fistula arising from a fissure, a submucous fistula may be associated with it (fig. 651, A, B). This possibility should never be lost sight of, because if the existence of the submucous fistula is overlooked, complete effacement of the subcutaneous fistula will not cure the patient of his symptoms.

*Differential Diagnosis.* Since the main track of the subcutaneous fistula is entirely confined to the subcutaneous tissue, the induration caused by its presence is superficial and can be readily picked up between the index finger and thumb and lifted, as it were, away from the underlying structures. In those instances in which the fistula has

existed for a considerable length of time, perhaps for several years, there is a marked thickening of the wall of the fistula, the result of thwarted efforts at repair, and therefore the course taken by the main track can be clearly defined by surface palpation. When the finger is introduced into the rectum and the tissues of the ischio-rectal fossa are grasped between it and the thumb placed externally, induration in the ischio-rectal fossa is found to be absent, thus serving to distinguish the subcutaneous type of fistula from the ischio-rectal, which often possesses subcutaneous extensions in connection with its main track. When deep-seated induration is detected, an ischio-rectal fistula with subcutaneous extension exists. As an external opening of a subcutaneous fistula is often situated close to the anal verge, it may be confused with that of a submucous fistula. The point is readily settled by introducing a probe into the external opening. Should the probe pass deeply into the main track without resistance, there can be no doubt that the opening leads into the track of a submucous fistula.

*Clinical Course.* When once established, the subcutaneous fistula continues to discharge pus from the external opening. When caused by a fissure, pain after defæcation, persisting for several hours afterwards, is a prominent symptom. If the internal opening is large, fecal matter is apt to enter the main track and set up active inflammation occasioning attacks of pain in the fistula itself. In these circumstances secondary abscesses are formed, with the attendant symptoms of pain, local tenderness, and pyrexia. Occasionally, when the main track is short, the whole of it may become epithelialised and spontaneously cured.

*Operative Technique.* The subcutaneous fistula is the simplest form of fistula, consisting, in most cases, of an external opening and an internal opening connected by a straight, superficially situated track. The operation necessary to effect a cure is therefore of the simplest kind. A probe-pointed director is introduced into the external opening, made to traverse the full length of the main track and emerge through the internal opening, and is then liberated by dividing the tissues over it. The result is a linear incision of shallow depth. With the exception of the corrugator cutis ani, no muscular structures are divided and, consequently, the wound heals without the slightest risk of impairment of control. As the edges of a linear incision are apt to turn in during the healing process, the resulting scar may become

depressed and groove-like. In order to prevent this and to expedite healing, a short relieving cut should be made on either side of the primary incision. When a fissure co-exists the pecten band should be divided in the right posterior quadrant of the anal orifice.

The pecten band is a fibrous ring due to the deposit of fibrous tissue in the submucosa of that portion of the anal canal which Stroud described as the pecten. This area is bounded above by the free borders of the valves of Morgagni (spoken of as the pectinate line), and below by Hilton's white line. Whenever an operation is being performed for a subcutaneous fistula caused by a fissure, a careful search should always be made for a submucous track extending upwards from the upper edge of the fissure. When such a submucous track exists, its presence can readily be detected by exploring the upper part of the fissure with a probe. The submucous track should then be dealt with according to the method advised for submucous fistula.

*After-treatment.* Since the wound resulting from laying open the main track of a subcutaneous fistula and the offshoots from it is shallow, the packing introduced into it at the time of the operation usually comes away when the dressing is changed twenty-four hours afterwards. In those cases in which the pecten band has been divided, the small pledget of cotton wool, which was inserted between the edges of the pectenotomy wound, should not be disturbed until the bowels are opened on the fourth day after the operation. The surface of the wound should be irrigated twice daily with an antiseptic solution. At the expiration of a week or ten days the surface of the wound is usually covered throughout by healthy granulations, and healing should be complete at the end of three weeks. In those cases in which a submucous track has been treated by the ligature method (see fig. 654), the ligatures, together with the slough, become detached on the eighth or ninth day and are voided in the stools. On the twelfth day, and every day afterwards until the wounds have healed, the finger should be passed into the anal canal to ensure healing taking place progressively from the bottom of the wound.

#### THE SUBMUCOUS TYPE OF ANO-RECTAL FISTULA

The submucous fistula is confined to the submucosa of the anal canal and lower portion of the rectum, and is usually limited to one side of the bowel, though occasionally it may extend to both sides. It originates as an abscess in the submucosa of the rectal ampulla, and is

due either to septic phlebitis or lymphangitis or to septic infection of blood clot which has been extravasated beneath the mucosa as the result of the puncture of a hæmorrhoidal vein by a foreign body, or to rupture of a vein during a violent straining effort. Occasionally the abscess is of tuberculous origin. Owing to the loose texture of the submucous tissue, the abscess extends laterally as well as downwards towards the anus, where it discharges its contents through an opening either at the anal margin itself or through the skin within half an inch of that point.

This type of fistula presents four varieties: (a) blind external submucous; (b) blind internal submucous; (c) complete submucous; (d) bilateral submucous.

(a) *The blind external submucous fistula* (fig. 652) is generally the result of a submucous hæmatoma. The extravasated blood soon becomes infected and suppurates freely. The resulting abscess gravitates towards the anus, and in doing so takes an oblique course downwards and backwards until it reaches the anal margin where it finds an exit through the skin. The external opening is usually to be found either in the right or in the left posterior quadrant, and is generally situated within half an inch of the anal verge. The main track extends obliquely upwards and forwards and terminates in a cul-de-sac (fig. 652, A). As a rule, the track is situated close to the muscular coat of the bowel beneath the hæmorrhoidal vessels.

(b) *The blind internal submucous fistula* (fig. 651, A) is the result of a submucous abscess which has been caused by septic phlebitis or lymphangitis. The septic process usually starts in a lesion situated in the anal canal such as a fissure, a torn-down anal valve, laceration of the mucosa of the anal canal during the passage of a foreign body, e.g. a fish-bone, ulceration of the interior of a valve of Morgagni, or a puncture by a syringe needle used for the injection treatment of internal hæmorrhoids. The internal opening is situated in the anal canal at the site of the initial lesion and affords a ready vent for the pus in the abscess cavity, so that the abscess seldom attains large dimensions. Consequently the main track is seldom more than an inch long and generally takes a course parallel to the longitudinal axis of the rectum.

(c) *The complete submucous fistula* (fig. 646) is the least common of the varieties of submucous fistula. It is invariably the result of an abrasion of the surface of the rectal mucosa above the level of the

levator ani. The abrasion may be due to puncture or laceration by a foreign body (fragment of bone), or to the partial tearing through of the pedicle of a fibrous or an adenomatous polypus.

An extensive abscess is formed as the result of acute septic infections, and although the pus escapes freely through the internal opening situated at the site of the original lesion, it also tracks down towards the anus and discharges through an opening situated within half an inch of the anal margin.

(d) *The bilateral submucous fistula* (fig. 653) is always the result of a submucous abscess situated on the posterior wall of the rectum above the level of the levatores ani. Owing to the spasmodic action

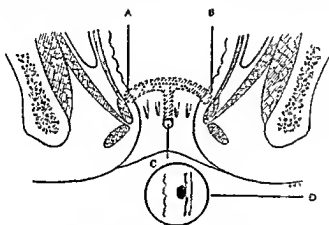


Fig. 653.—BILATERAL SUBMUCOUS FISTULA.

A and B—Cul de sacs of transverse portion of fistulous track

C—Internal opening at level of interval between the sphincters

Inset, D.—Showing the position of the track in regard to the muscular coat of the rectum.

of the levatores, induced by the presence of the abscess, the anal canal is firmly compressed and the purulent collection, unable to track downwards on that account, extends transversely beneath the mucosa on both sides above the level of the levatores. Eventually, the pus tracks down towards the anus exactly in the middle line posteriorly and discharges itself through an opening situated at the level of Hilton's white line (fig. 653, C). As soon as the pus has been evacuated the abscess cavity contracts, but the lateral portions of it remain as bilateral tubular extensions from the upper extremity of the median main track (fig. 653, A, B). These lateral extensions usually terminate in a cul-de-sac and are generally situated beneath the hæmorrhoidal veins (fig. 653, D). The T-shaped disposition of the submucous induration caused by the lateral offshoots from the main track of the fistula is characteristic of the bilateral submucous fistula.

*Differential Diagnosis.* It is of the utmost importance, from the point of view of surgical treatment, that a fistula of the submucous type be distinguished from one whose main track is situated externally to the muscular coat of the rectum, as in the case of the para-rectal fistula. In the complete variety of either type there is an external opening situated in the peri-anal region, associated with a high-lying internal opening through which the point of a probe-director can be made to protrude (see figs. 646 and 648).

Laying open the track of a fistula of the submucous type is not followed by the slightest degree of impairment of the power of control, but if a similar procedure be carried out in a fistula of the para-rectal type permanent incontinence will ensue. In order to distinguish between the two types, a probe-director should be introduced into the main track through the external opening and pushed onwards until its point protrudes through the internal opening. If the edges of the groove in the director can be felt by the examining finger, the track is situated in the submucosa, but if they cannot be felt, then the track is outside the wall of the rectum (see fig. 659, I).

*Clinical Course.* The submucous fistula differs from all other types by not exhibiting any tendency to extend by means of the development of secondary offshoots from the main track. As soon as the contents of the original abscess have been evacuated, the abscess cavity gradually contracts into a linear track. As the opening through which the pus has been evacuated is situated at the most dependent part of the track, there is effective drainage. When there is an external opening the escape of pus is generally continuous, but in cases in which the opening is situated in the anal canal, the discharge is intermittent, taking place either during an action of the bowels or when flatus is being voided.

*Operative Technique.* The principle of laying open the main track of a fistula throughout its full extent can be safely applied to fistulae of the submucous type, provided that the incision is made towards the lumen of the bowel, and *not in the opposite direction*. The tissues thus divided consist only of mucous membrane and the blood-vessels contained in the submucous tissue when the track lies beneath them, so that there is no danger of damaging the muscular control of the anal outlet. Cutting across the hæmorrhoidal arteries and veins causes free bleeding which may be exceedingly difficult to control, especially when the track of the fistula extends upwards for two or three inches above the anal verge. When the track lies between the branches of the

hæmorrhoidal vessels, or is superficial to them, there is no danger, but, since the track passes beneath the vessels obliquely in the majority of instances, it is impossible to lay it open without also dividing them. Consequently, the method of laying open the track with the knife is unsuitable for this type of fistula and therefore the ligature method should always be employed.

When an external opening is present, the portion of the track between it and Hilton's white line can be safely incised, as bleeding points in that situation can readily be controlled, but the remainder of the track should be treated in the following manner: A probe-director is introduced into the lower extremity of the track and passed along until the upper end of the track has been reached. If an internal opening exists at that point, the point of the probe is pushed through

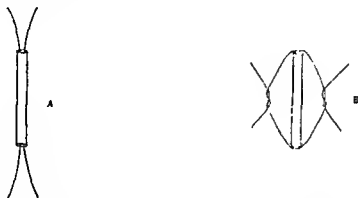


Fig. 654.—THE LIGATURE METHOD OF TREATING A SUBMUCOUS FISTULA.

A—A double ligature introduced into the main track.

B—The area between the dotted lines indicates the granulating surface left after the ligatures have separated.

it, but, if not, the probe must be forced through the mucosa. Since submucous tracks usually take an oblique course upwards, the point where the probe emerges through the mucous membrane is seldom more than two inches above the level of the anal margin, so that the end of the probe can readily be hooked down by the finger and made to protrude through the anus without lacerating the mucosa. Two stout silk ligatures (No. 16 plaited) are then attached to the end of the probe and drawn through the track (fig. 654, A). Without interlocking the ligatures, one of them is tied tightly and then the other is similarly tied a little to one side of it so as to include the portion of the mucosa forming the superficial wall of the track (fig. 654, B). The mucosa included between the ligatures necroses, and when the slough separates, the track is found to have been laid open from end to end just as effectively and with more safety to the patient than when the method of incision has been employed.



Those who follow blindly the doctrine that a fistulous track having a high-lying internal opening should never be laid open into the bowel divide the tissues surrounding the track in the opposite direction, that is to say, outwards and away from the lumen of the bowel. The effect is exactly the same as when the track of a para-rectal fistula is laid open into the lumen of the rectum. I have seen several instances of permanent incontinence result from this plan of operating upon a submucous fistula. There is no doubt, therefore, that those who practise it fail to recognise the important fact that fistulae possessing a high-lying internal opening may differ in regard to the anatomical relationship of the main track to the musculature of the anal outlet, upon the preservation of which the power of control so much depends.

*After-treatment.* The bowels should be confined for four days and then opened by a brisk purgative. Twenty-four hours before the purgative is administered, four ounces of olive oil should be introduced through the anus and retained. An ounce of liquid paraffin is given each succeeding night, combined with a mild purgative, to ensure a daily movement of the bowels. The application of a ligature to the mucous membrane of the rectum does not cause pain, so that sedatives are seldom necessary. The ligatures, together with the slough, usually separate on the eighth or ninth day and are voided in the stools. On the twelfth day the index finger should be passed into the rectum to ascertain, primarily, whether the ligatures have separated, and, secondly, to find out whether any part of the track remains unopened. On each succeeding day, until healing is complete, the finger should be passed into the rectum to ensure that the margins of the exposed track do not become approximated and re-establish the fistula. Epithelialisation of the granulating surface is usually completed in nineteen days in men and in sixteen days in women.

#### THE INTERMUSCULAR TYPE OF ANO-RECTAL FISTULA

The intermuscular ano-rectal fistula is one in which the main track is situated between the internal sphincter and the longitudinal muscular coat of the rectum (fig. 655). It is the rarest type and is seldom more than an inch in length. It is due to septic lymphangitis following suppuration in a sinus of Morgagni, and is generally associated with a fistula of the sub-sphincteric type, but sometimes is found to exist by itself. The main track takes a straight course parallel to the longitudinal axis of the bowel, does not give rise to offshoots, and terminates

in a cul-de-sac. There is an excellent example in the museum of St. George's Hospital, the dissected specimen displaying the track beneath the fibres of the internal sphincter. There is only one variety of this type, namely, the blind internal, the internal opening being situated at the entrance to a sinus of Morgagni about a quarter of an inch above Hilton's white line.

*Differential Diagnosis.* The intermuscular fistula must be distinguished from one of the submucous type, and especially from the submucous extension from either the sub-sphincteric or the ischio-rectal types. The main track of the intermuscular fistula is always perfectly straight and seldom exceeds an inch in length. When a probe-director is introduced into the track, the probe cannot be distinctly felt. The track of the submucous fistula, or of a submucous extension from an ischio-rectal or from a sub-sphincteric fistula, is seldom straight, usually takes an oblique course forwards and upwards, and generally terminates in a cul-de-sac which can be distinctly felt as a rounded enlargement of a linear induration. When a probe-director has been introduced along such a submucous track, the margins of its groove can be easily recognised by the examining finger.

*Clinical Course.* When established, the track never becomes spontaneously obliterated, owing to the constant action of the internal sphincter muscle. Pus is formed continuously and escapes whenever the bowels act. In those instances in which an intermuscular track co-exists with either a sub-sphincteric or an ischio-rectal fistula and has escaped detection during an operation, the wound fails to heal completely, a slight discharge of pus persisting indefinitely unless its presence is discovered.

*Operative Technique.* Since the main track of the fistula seldom exceeds an inch in length, there need be no hesitation in laying it open from end to end into the lumen of the bowel because, even if there is free bleeding as the result of cutting through the hæmorrhoidal vessels, the bleeding points are within easy reach and can readily be controlled. A probe-director should therefore be inserted into the track through the internal opening, and after traversing the whole length of the track, its point should be forced through the wall of the bowel into its lumen. The tissues covering the probe, which of course include the internal sphincter, are then divided completely. I have had occasion

to divide the internal sphincter in this way on several occasions and have never observed the slightest impairment of control to result in consequence.

*After-treatment.* This is practically the same as that described for a submucous fistula, and should be conducted upon those lines whenever an intermuscular fistula exists by itself. In those cases in which either an ischio-rectal or a sub-sphincteric fistula co-exists the treatment applicable to those conditions should also be carried out.

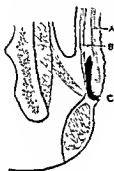


Fig. 653.

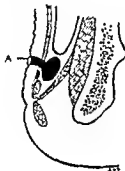


Fig. 656.

Fig. 653.—A BLIND INTERNAL INTERMUSCULAR FISTULA. NOTE THAT THE MAIN TRAIL LIES EXTERNALLY TO THE INTERNAL SPHINCTER MUSCLE.

A—External muscular coat,  
B—Internal mucous coat,  
C—Internal opening of fistula.

Fig. 656.—FIRST STAGE OF A PARARECTAL FISTULA.

A—The internal opening above the level of the internal sphincter muscle.

#### THE PARA-RECTAL TYPE OF ANO-RECTAL FISTULA

The para-rectal fistula is the result of an abscess which has formed in the para-rectal space, bounded internally by the external coat of the rectum and externally by the fascia propria. The compartment so formed contains the lymphatic sinus of the rectum and the peri-rectal fatty tissue. The efferent lymph channels emanating from the intramural lymphatic system of the rectum empty themselves directly into the lymphatic sinus, so that septic lymphangitis originating in the rectum may terminate in para-rectal suppuration. An abscess in the para-rectal space is shut off by the dense fascia propria from the stratum of loose cellular tissue existing between the pelvic peritoneum and the levatores ani, constituting the superior pelvi-rectal space of Richet (see fig. 645). The para-rectal abscess is situated in the space between the fascia propria and the rectum, and is always due to a

lesion in the rectum from which septic phlebitis or lymphangitis extends.

In some instances the septic process is due to puncture of the wall of the rectum above the level of the levatores by a foreign body, such as a fish-bone. Whether it is the result of septic thrombosis of the veins passing through the wall of the rectum, of septic lymphangitis, or perforation of the rectal wall by a foreign body, the abscess cavity communicates with the interior of the bowel by means of a high-lying internal opening (fig. 656, A). An abscess thus situated, being unable to invade the superior pelvi-rectal space on account of the attachment of the fascia propria to the recto-vesical fascia, may extend completely round the rectum. The pus may then invade the subjacent ischio-rectal fossa on the same side by passing through the interval between the levator and the coccygeus, or between the fasciculi of the levator itself (fig. 657, B). Finally, it escapes through an opening on the skin surface and in some instances by an internal opening situated in the middle line posteriorly, at the level of the interval between the internal and the external sphincters (fig. 658, B). In rare instances the collection of pus above the levator finds its way downwards in the middle line posteriorly between the coccygei and invades both ischio-rectal fossae simultaneously, eventually discharging through symmetrically placed external openings (fig. 659, C, D).

There are three varieties of the para-rectal fistula, namely : (a) blind internal para-rectal ; (b) complete para-rectal ; (c) bilateral para-rectal.

(a) *The blind internal para-rectal fistula* (fig. 656) is in reality the primary stage in the development of the complete variety, which is the form in which the para-rectal fistula is most commonly met with. Its onset is characterised by acute constitutional disturbance associated with pain deeply seated in the pelvis. The pain may be very acute and may radiate to the lumbar and sacral regions and, in some instances, extend down the back of the thighs along the course of the great sciatic nerve. Unless symptoms definitely referable to the rectum develop, the presence of a para-rectal fistula in its early stage may easily be overlooked. So soon as the pus has invaded the ischio-rectal fossa, a tender swelling, associated with redness of the skin in the neighbourhood of the anus, points to the supervention of an ischio-rectal abscess. A copious discharge of pus from the rectum is significant of the existence of a deep-seated blind internal fistula, so that an early opportunity

should be taken of examining the rectum with the proctoscope. When a blind internal para-rectal fistula exists, a copious discharge of pus will be seen to be taking place from a single point in the rectal wall, above the level of the levator ani. When the finger is introduced into the rectum a definite tense elastic swelling is felt encroaching upon the lumen of the bowel and generally extending upwards beyond the reach of the finger. Should the lower margin of the swelling be situated above the level of the levator ani, it is evidence that the pus has not yet made its way into the ischio-rectal fossa (fig. 656).

(b) *The complete para-rectal fistula* is a later stage of the preceding variety. With the formation of the external opening a copious discharge

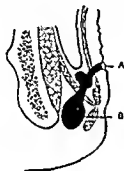


Fig. 657.

Fig. 657.—A PARA RECTAL FISTULA EXTENDING INTO THE ISCHIO-RECTAL FOSSA.

A—Internal opening above the level of the internal sphincter muscle.

B—The extension into the ischio-rectal fossa.

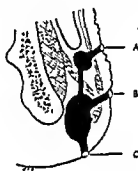


Fig. 658.

Fig. 658.—A COMPLETE PARA RECTAL FISTULA.

A—Primary internal opening. B—Secondary internal opening. C—Primary external opening

of pus takes place and all previous symptoms of an acute ischio-rectal abscess rapidly disappear. If the patient is now seen for the first time, the external appearances are those of an ischio-rectal fistula, the external opening being situated at least an inch and a half distant from the anal verge and a trifle in front of the transverse anal line or slightly posteriorly to it. The main track, however, passes deeply in a direction parallel to the longitudinal axis of the rectum, so that a probe introduced into the external opening easily enters the track to a depth of three or four inches. If the finger is then introduced into the rectum, the point of the probe will be felt to protrude into the cavity of the bowel through an internal opening situated above the level of the levator ani. Throughout its course the main track is situated outside the wall of the rectum, and as it passes through the levator ani or between the muscle and the coccygeus it is also external to the point of fusion of

those muscles with the longitudinal muscular coat of the rectum (fig. 658).

(c) *The bilateral para-rectal fistula* is the most extensive and also the most complex of the ano-rectal fistulæ. The original abscess, having completely encircled the rectum, extends downwards through the interval between the coccygeus muscles, in the middle line posteriorly, and gives rise to septic lymphangitis in both ischio-rectal fossæ, with the result that an ischio-rectal abscess develops on both sides. Since the disposition of the lymphatics is symmetrical on both sides, when the collection of pus in the ischio-rectal fossæ finds an exit on the skin surface, the resulting primary external openings are usually symmetrically placed (fig. 659, C, D).

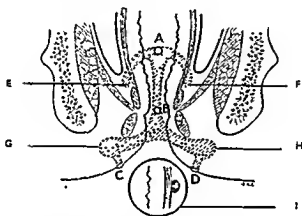


Fig 659.—A BILATERAL PARA RECTAL FISTULA

A—Primary internal opening.

B—Secondary internal opening.

C and D—Primary external openings.

E and F—Blind extremities of the track above the level of the levatores ani.

G and H—Extensions of main track into the ischio-rectal fossæ.

I—Inset.—Showing the main track external to the muscular coat of the rectum.

Should offshoots subsequently develop, the secondary external openings in connection with them are not necessarily symmetrical, as the tendency is for the greater number to develop on the right side. As the result of the invasion of the ischio-rectal fossæ, and also as a consequence of septic lymphangitis, an internal opening usually develops in the middle line posteriorly between the internal and the external sphincters (fig. 659). This is a secondary internal opening, and must not be mistaken for the primary internal opening which is situated much higher up and always above the level of the levator ani (fig. 659, A). In addition to the bilateral extensions from the main track above the levatores and those situated in the ischio-rectal fossæ, there may also exist submucous extensions from the internal openings, unilaterally

or bilaterally disposed, so that the bilateral variety of the para-rectal type of ano-rectal fistula may combine several of the types in one complex fistula.

*Differential Diagnosis.* In the early stage, when still confined in the space above the pelvic diaphragm, the blind internal para-rectal fistula may be mistaken for a high-lying submucous abscess. Both produce a globular swelling encroaching upon the lumen of the bowel. Since the swelling produced by the para-rectal fistula is outside the wall of the bowel, the mucosa is freely movable over it; whereas it is smooth and immobile in the case of the submucous abscess. When the para-rectal fistula has extended into the ischio-rectal fossa, the features of an ischio-rectal abscess are simulated, but the fact that the swelling in the ischio-rectal fossa extends upwards beyond the level of the levator points to its true nature.

When there is an external opening into which a probe passes deeply the para-rectal fistula may be mistaken for: (1) a submucous fistula; (2) a pelvi-rectal fistula; and (3) a pre-sacral (retro-rectal) fistula.

The position of the external opening serves as a useful guide. The external opening of a submucous fistula is usually situated within half an inch of the anal verge. That of a pelvi-rectal fistula is found to be at least an inch and a half from the anal verge and slightly posterior to the transverse anal line. That of the retro-rectal (pre-sacral) is generally located at the level of the tip of the coccyx and about one inch distant from it, whereas the external opening of the para-rectal fistula is generally found at a greater distance than an inch and a half from the anal verge and slightly in front of the transverse anal line. Moreover, neither the pelvi-rectal nor the retro-rectal fistula possesses an internal opening, which is always present in a para-rectal fistula.

*Clinical Course.* The development of an abscess in the para-rectal space produces the symptoms of pelvic cellulitis. Until symptoms definitely referable to the rectum develop, an abscess in the para-rectal space may easily be overlooked and, therefore, digital exploration of the rectum should never be omitted. Sooner or later the ischio-rectal fossa becomes invaded and then the implication of the rectum is apparent. Eventually, unless the ischio-rectal abscess is freely incised, there develops a primary external opening through which the pus is evacuated. Offshoots from the main track in the ischio-rectal fossa may subsequently develop, with the result that numerous secondary external openings appear. As a rule, when off-

shoots from the main track are present, an internal opening, situated in the middle line posteriorly between the external and the internal sphincters also exists (see figs. 658, B and 659, B).

*Operative Technique.* The principle of laying open the main track of an ano-rectal fistula into the bowel throughout its extent from the



Fig. 660.—A COMPLETE UNILATERAL PARA RECTAL ANO-RECTAL FISTULA. THE EXTERNAL OPENING WAS SITUATED ON THE TRANSVERSE ANAL LINE  $1\frac{1}{2}$  INCHES FROM THE ANAL MARGIN. THE MAIN TRACK EXTENDED PARALLEL TO THE RECTUM FOR A DISTANCE OF  $3\frac{1}{2}$  INCHES. THE INTERNAL OPENING WAS  $2\frac{1}{2}$  INCHES ABOVE THE ANAL MARGIN. THE SKIAGRAM WAS TAKEN AFTER THE TRACK HAD BEEN INJECTED BY R.I.P.P.

external opening to the internal opening must *never* be applied to a para-rectal fistula. A glance at figures 648 and 658 show that by doing so not only would both the external and the internal sphincters be divided, but the point of fusion of the levator ani with the longitudinal coat of the rectum, together with the whole thickness of the bowel wall as far as the high-lying internal opening, would be divided as well, resulting in



permanent and irremediable incontinence. The tissues on the outer aspect of the main track alone should be divided as far as but not beyond the point where the main track passes through the wall of the rectum (fig. 662). The incision through the levator ani is made in the direction of its fibres and not transversely. The surface wound should be extensive, as shown in figure 662 by the line B-C, so that the area of the surface wound will be greater than the combined area of the remainder of the wound. Since the details of technique vary in some particulars when operating upon para-rectal fistulæ in the four stages represented by figures 656, 657, 658 and 659, it will be as well if they are described separately.

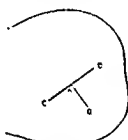


Fig. 661.

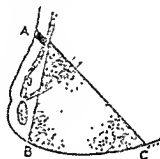


Fig. 662.

Fig 661.—SHOWING THE INCISIONS RECOMMENDED WHEN OPERATING UPON A PARA RECTAL FISTULA.  
 AB—Incision extending outwards from outer margin of the external sphincter muscle.  
 CD—Incision made at right angle from inner extremity of the first incision.

Fig 662.—SHOWING THE WOUND RESULTING FROM THE INCISIONS INDICATED IN FIG. 661. NOTE THAT THE WOUND EXTENDS AS FAR AS THE INTERNAL MUSCULAR COAT OF THE RECTUM BUT NOT THROUGH IT.  
 A—The internal opening and submucous portion of the main track.  
 BC—The extent of the surface wound.

(1) *In the early stage of the blind internal para-rectal fistula.* When the finger is introduced into the rectum, a globular swelling is felt above the level of the levator ani (fig. 656). With the finger acting as a guide within the rectum, the point of a long straight bistoury is introduced through the skin at the point A in figure 661. The blade of the knife is passed onwards, outside the wall of the rectum, parallel to the finger, until the cavity of the abscess has been penetrated, as indicated by the escape of pus along the knife. When introducing the knife the greatest care must be exercised in order to avoid penetrating the wall of the rectum. So soon as the abscess cavity has been penetrated, the knife should be withdrawn in such a way that the tissues are divided away from the rectum, as indicated by the line A-B in figure 661. This incision is then extended by making another incision C-D at right angles to its inner extremity. The resulting wound is pyramidal in shape and, provided that the surface incisions have been

made sufficiently long, the square area of the surface wound will be greater than the combined area of the remainder of the wound. The finger should then be introduced into the abscess cavity in order to ascertain whether a foreign body, such as a fish-bone, is present. I have found pins, needles, and fish-bones in the cavities of this type of fistula on several occasions.

(2) *When a blind internal para-rectal fistula has extended to the ischio-rectal fossa (fig. 657).* Owing to the fact that the abscess has extended into the ischio-rectal fossa, the pus is nearer the surface and the operation is much less difficult, because there is no danger of the rectum being penetrated during the introduction of the bistoury. The abscess cavity in the ischio-rectal fossa is laid open freely by incisions similar to those employed in the operation for the preceding stage. The aperture in the levator ani leading into the para-rectal space is sought for and enlarged in the direction of its muscular fibres. The cavity above the levator is then explored with the finger, in order to ascertain its dimensions and also to disclose the presence of a foreign body, should one exist. Should it be found that the abscess cavity extends upon the upper surface of the levator for a considerable distance, on either side of the aperture through the muscle, a short transverse cut through the fibres of the levator should be made on both sides of the radiating incision, but these must be as limited as possible commensurate with providing adequate drainage.

(3) *When a blind internal para-rectal fistula has become complete (fig. 658).* A probe-director is introduced into the primary external opening and gently passed along the track in the ischio-rectal fossa as far as it will travel without force. The point of the probe is then pushed through the skin if a secondary external opening does not exist. The tissues overlying the probe are then divided to expose the track in the ischio-rectal fossa. A careful search is then made for offshoots from the track, and if any are found they should be laid open freely. When this has been done the aperture through the levator ani is sought for and dealt with as described above. When a secondary internal opening is found to exist in the middle line posteriorly between the sphincters, the portion of the track extending beneath the external sphincter to the internal opening should not be laid open until the deep part of the main track has been obliterated by the healing process. If the portion of the track extending beneath the external sphincter is laid open at the time of the main operation, feces are apt to enter the deep part

of the wound during an action of the bowels, and may greatly impede the process of repair. Nevertheless, the wound will not heal completely until this portion of the track has been laid open, so that in these cases an operation in two stages is necessary.

(4) *When the para-rectal fistula is bilateral.* The tracks in both ischio-rectal fossæ are laid open completely, commencing on the side on which the primary external opening appeared. The track leading into the portion of the fistula situated above the levatores is found to be situated in the middle line posteriorly, between the coccygeus muscles. The muscles are divided for a short distance on either side, in order to drain the lateral extensions of the cavity above them (fig. 659, E, F). The surface wounds are extended by relieving cuts (see fig. 649) wherever required. The portion of the track passing beneath the external sphincter to a secondary internal opening, which is situated between the sphincters in the middle line posteriorly, should not be laid open until the deep parts of the wound on both sides have been filled in.

In all operations undertaken for a para-rectal fistula the high-lying internal (primary) opening should not be interfered with. It will close completely as soon as the deep portion of the wound has been obliterated by the healing process.

*After-treatment.* However carefully and accurately an operation for a para-rectal fistula may have been carried out, unless the after-treatment is conducted with scrupulous care and attention to detail, the operation may fail to effect a cure. This is due to the difficulty in securing adequate drainage for the portion of the main track which lies above the level of the levator ani, without inflicting serious damage to the muscle.

Should the incision through the levator close before the cavity above it has been obliterated, a sinus will remain which will continue to discharge pus indefinitely and prevent complete closure of the surface wound. Especial care, therefore, should be devoted to the part of the track above the levator during the early stages of the after-treatment. The gauze packing introduced into the cavity of the wound after the operation had been completed should be left *in situ* for at least four days and then removed. The advantage of leaving the packing in the wound for that length of time is that, after its removal, the sides of the cavity do not fall together as readily as they otherwise would. After the original packing has been removed the wound must on no account be repacked.

One of the chief reasons why fistulous tracks fail to close after an operation is because they are repacked persistently. It stands to reason that, if a cavity is packed continuously, it is a physical impossibility for healing to take place. The packing prevents the growth of granulation tissue and delays the process of repair. If there is a tendency for the surfaces of the wound to fall together, they should be kept apart by interposing a thin layer of wool or gauze between them, to prevent them adhering together before the deep part of the wound has closed in.

Careful irrigation of the entire wound cavity, at least twice daily and also after each action of the bowels, is all that is necessary to ensure steady healing taking place from the bottom of the wound. For this purpose there is nothing more efficacious than a strong solution (1 in 500) of perchloride of mercury. When, after the expiration of ten days, the surface of the wound is covered by healthy granulation tissue, the perchloride is dispensed with and a solution of iodine or dettol (2 dr. to a pint) is substituted for it.

In cases of long standing, the growth of granulation tissue is usually slow. This is due to excessive induration of the tissues surrounding the track, and it becomes necessary to destroy the indurated tissue in order to expedite the growth of the granulations. For this purpose there is nothing better than nitrate of silver. When the track is straight it can be easily applied to the whole surface by means of the solid stick, but if the track is tortuous, or if there are offshoots extending from it, a saturated solution (960 grs. to the ounce) should be injected into the track by means of a suitable syringe.

As the result of the application of nitrate of silver the indurated wall of the track sloughs and, when the slough exfoliates, at the expiration of six days, a healthy granulating surface is left which proceeds to obliterate the cavity with normal rapidity. The closing of the wound, resulting from the operation upon a para-rectal fistula, may take from two to six months according to its depth and its duration. Should the surface wound close before the deep part has completely filled in, a secondary operation is necessary to reopen the surface wound to the required extent.

#### THE SUB-SPHINCTERIC TYPE OF ANO-RECTAL FISTULA

The sub-sphincteric fistula, as its name implies, is situated beneath the external sphincter muscle (fig. 663). It is prevented from extending into the fatty tissue of the ischio-rectal fossa by the fascial expansion,

by means of which the longitudinal muscular coat of the rectum is inserted into the skin at the outer border of the external sphincter muscle.

It will be remembered that the longitudinal muscular fibres of the rectum, after blending with the levator ani, terminate in a fascial expansion which divides into two strata, an outer and an inner. The outer stratum passes on the deep aspect of the external sphincter to be inserted into the skin at the outer border of that muscle; this stratum forms a compartment for the external sphincter and intervenes between

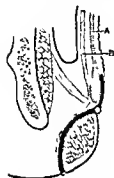


Fig. 663.—COMPLETE SUB-SPHINCTERIC FISTULA WITH SCHEMATIC EXTENSION. NOTE THAT THE MAIN TRACT IS SITUATED BETWEEN THE MUSCLE AND THE EXPANSION FROM THE EXTERNAL COAT

A—Circular muscular coat.  
B—Longitudinal muscular coat.

it and the ischio-rectal fat. The inner layer passes through the external sphincter, dividing it into a superficial and a deep portion, and is also inserted into the skin at the outer border of the muscle. The efferent lymphatics from the anal canal, on their way to join the subcutaneous lymphatic plexus at the outer border of the external sphincter muscle, pass beneath the muscle, between it and the stratum of the fascial expansion which separates it from the ischio-rectal fat. Some of the lymphatics also pass between the superficial and the deep portions of the external sphincter muscle. Venous trunks from the anal canal also follow a similar route in order to join the circum-anal veins.

As the result of septic phlebitis, or lymphangitis, an abscess may form either between the external sphincter and the fascia or between the superficial and the deep portions of that muscle. Such abscesses

find an exit at the outer border of the muscle, the primary external opening of the resulting fistula being situated at a point between an inch and an inch and a half distant from the anal verge (fig. 663). The initial lesion is usually an ulcer in one of the sinuses of Morgagni, laceration at the base of the pedicle of a small fibrous polypus, or a fissure in the middle line, posteriorly, of the anal margin. Occasionally the septic focus is the result of puncture or laceration of the mucosa of the anal canal during the passage through the anus of a fish-bone or fragment of bone which has been swallowed.

The sub-sphincteric fistula presents three varieties, namely: (a) the blind internal sub-sphincteric fistula; (b) the complete sub-sphincteric fistula; and (c) the bilateral sub-sphincteric fistula.

(a) *The blind internal sub-sphincteric fistula* is the early stage of the complete variety, and consists of an abscess cavity situated beneath

the external sphincter muscle and communicating with the interior of the anal canal through an internal opening which is the entrance to one of the Morgagnian sinuses. In those instances in which the source of infection is situated at the lower part of the anal canal—for instance, in a fissure—the initial abscess is often situated between the superficial and the deep portions of the external sphincter muscle. In either situation the abscess eventually points at the outer border of the external sphincter, forming an exquisitely painful swelling associated with a discharge of pus from the anal canal.

(b) *The complete sub-sphincteric fistula* is a later stage of the preceding variety, the pus having found an exit through the skin at the outer border of the external sphincter. The external opening is usually situated about one and a quarter inches from the anal verge. Extension of the fistula, as the result of septic lymphangitis, takes place in the subcutaneous tissue, the offshoots terminating in secondary openings. When the primary external opening is situated anteriorly to the transverse anal line the offshoots extend forwards and towards the middle line, but when the primary external opening is situated posteriorly to the transverse anal line the offshoots extend backwards and outwards.

The position of the internal opening and the direction taken by the main track towards it vary according to the position occupied by the primary external opening in reference to the transverse anal line. When the primary external opening is situated anteriorly to the transverse anal line or on that line, the internal opening is situated directly opposite to it in the same radial line, between the sphincters, and the main track adopts a straight course. When, however, the primary external opening is situated posteriorly to the transverse anal line, the internal opening will be found in the middle line of the anal canal posteriorly, at the level of the interval between the sphincters, the main track taking a curved course backwards and inwards (fig. 664, opening behind B). It is important to bear these points in mind when performing an operation for sub-sphincteric fistula.

(c) *The bilateral sub-sphincteric fistula* is due to subcutaneous offshoots from the termination of the main track of a sub-sphincteric fistula, extending on both sides of the middle line in those instances in which the primary external opening is situated in front of the anus at or near to the middle line (fig. 665, A, B, H). The bilateral disposition of the subcutaneous offshoots from a median sub-sphincteric fistula has given rise to the term “anterior horseshoe fistula.” Several secondary external openings may be present, but the primary external opening is

always situated at the outer margin of the external sphincter, and the direction taken by the main track is straight beneath the muscle. The internal opening will be found opposite the interval between the sphincters, in the same radial line as the external opening.

*Differential Diagnosis.* Sub-sphincteric fistulæ can be distinguished from purely subcutaneous fistulæ by the fact that their main tracks, by passing beneath the external sphincter, cannot be palpated in the

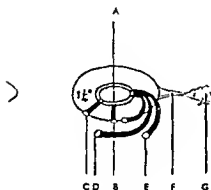


Fig. 664.

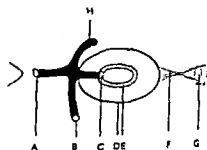


Fig. 665.

Fig. 664.—SHOWING THE RELATION OF THE EXTERNAL OPENING TO THE INTERNAL OPENING IN SUB-SPHINCTERIC AND ISCHIO-RECTAL FISTULÆ.

- AB—The transverse anal line.
- C—External opening of a sub-sphincteric fistula anterior to the transverse anal line.
- D—External opening of an ischio-rectal fistula anterior to the transverse anal line.
- E—External opening of an ischio-rectal fistula posterior to the transverse anal line.
- F—Ano-coccygeal ligament.
- G—Coccyx.

Fig. 665.—A BILATERAL SUB-SPHINCTERIC FISTULA. (ANTERIOR HORSESHOE.)

- A—Primary external opening of a sub-sphincteric fistula in the middle line anteriorly.
- B—Secondary external opening at the extremity of a sub-cutaneous extension from the main track.
- C—Internal opening of the same fistula.
- D—Outer margin of the external sphincter muscle.
- E—Interval between the internal and the external sphincters.
- F—Ano-coccygeal ligament.
- G—Coccyx.

subcutaneous tissue. A perineal urethral fistula, however, exhibiting subcutaneous extensions in close proximity to the anus may resemble a sub-sphincteric fistula, in connection with which subcutaneous extensions may exist in front of the anus. Both fistulæ have their origin in a painful inflammatory swelling in front of the anus—near to the middle line—which increases in size and ultimately discharges through one or more external openings. The direction taken by the main track from the primary external opening is of great importance for the purpose of distinguishing between them. The main track of the urethral fistula extends forwards towards the scrotum, whereas that

of the sub-sphincteric fistula extends towards the anus and passes beneath the external sphincter. The urethral fistula is invariably associated with a stricture of the urethra, the presence of which is easily ascertained by passing a sound. The absence of induration in the ischio-rectal fossa, as determined by palpating the tissue of the fossa between the finger in the rectum and the thumb externally, serves to differentiate the sub-sphincteric from the ischio-rectal or the pararectal types.

*Operative Technique.* In those cases in which the external opening is situated at the outer border of the external sphincter, either anteriorly to the transverse anal line or on that line, the main track is straight and takes a radial course to the internal opening which is situated between the sphincters. All that is necessary, therefore, is to introduce a probe-director into the primary external opening and pass it along the track until it protrudes through the internal opening and then to liberate the probe by dividing the tissues over it. The resulting incision is then extended for a short distance beyond the external opening, so as to provide for adequate drainage. The surface wound is also further increased in area by short relieving cuts made at right angles to the line of the main incision, on either side, through the skin and subcutaneous tissues. The presence of offshoots in the subcutaneous tissue should be sought for by careful probing and, if found, these should be laid open freely.

In those cases in which the primary external opening is situated posteriorly to the transverse anal line and the main track curves backwards and inwards towards the middle line posteriorly, the method advocated for laying open the main track of a complete ischio-rectal fistula should be adopted. When operating upon a fistula of the sub-sphincteric type, careful search should always be made for either a submucous extension or a blind internal intermuscular fistula originating from the site of the internal opening, as these occasionally exist and, if overlooked, prevent complete healing.

*After-treatment.* The wound resulting from laying open the subcutaneous offshoots in connection with a sub-sphincteric fistula needs very little attention, as it is superficial and should be treated on the lines suggested for a subcutaneous fistula. The portion of the wound, however, which extends through the external sphincter into the anal canal must be treated with particular care. The packing in this portion of the wound should be removed at the expiration of four days.



Twice daily afterwards, and after each action of the bowels, the wound should be cleansed, but on no account should it be repacked. Repacking of the wound through the external sphincter ends in healing taking place with a depressed scar, which is one of the commonest causes of impairment of control over the contents of the rectum after operations for fistula. Should there be a tendency for the edges of the wound to adhere to one another, a thin layer of wool is all that is required to keep them apart. When treated in this way, healing takes place progressively from the deepest part of the wound and the resulting scar is not depressed. The duration of the healing process is generally from three to four weeks.

#### THE ISCHIO-RECTAL TYPE OF ANO-RECTAL FISTULA

The fatty tissue of the ischio-rectal fossa, being of low resisting power and exposed to pressure and contusions, is frequently the seat of an abscess which terminates in a fistula. The main track of the resulting ischio-rectal fistula is therefore confined to the ischio-rectal fossa. The abscess preceding the fistula is generally the result of septic lymphangitis extending from a septic focus situated in the posterior part of the anal canal, but occasionally it may be due to local infection by micro-organisms circulating in the blood stream. The initial lesion is always situated in the anal canal posteriorly, between the sphincters.

Whenever an ischio-rectal fistula is found to exist, there is invariably marked separation between the sphincters. Normally, the internal sphincter is so closely superimposed upon the external that it is difficult to determine exactly where one begins and the other ends, but when they are separated from one another a distinct sulcus, seldom exceeding one-third of an inch in extent, can be made out to exist between the muscles on introducing the finger into the anal canal. The separation is more pronounced in the middle line posteriorly than elsewhere.

The separation between the muscles is produced by the levator ani contracting forcibly in an endeavour to overcome the limitation, imposed by the presence of the pecten band, to the normal expansion of the external sphincter during defæcation. Consequently the internal sphincter is pulled away from the underlying external sphincter, and a sulcus between the muscles is formed.

The mucosa in the middle line posteriorly, where the separation is widest, is apt to become abraded during the passage of a constipated stool, and then a septic focus is established. The resulting abscess

discharges on the skin surface by an external opening, situated generally about two inches distant from the anal verge and slightly behind the transverse anal line. The main track of the fistula thus formed curves backwards along the course of the infected lymphatics towards the middle line and terminates in the middle line of the anal canal, opposite the interval between the sphincters, by means of an internal opening which is located at the site of the initial lesion.

For these reasons the main track of an ischio-rectal fistula always takes a curved course backwards from the primary external opening (fig. 664, D, E), and the internal opening is always situated in the middle line posteriorly between the sphincters and nowhere else. Owing to the symmetrical arrangement of the lymphatics, when the infection extends to the opposite side, the extension follows an exactly similar course to that taken by the original main track, with the result that the so-called posterior horseshoe fistula is formed (see fig. 669).

There are four varieties of the ischio-rectal fistula, namely: (a) the blind external ischio-rectal; (b) the blind internal ischio-rectal; (c) the complete ischio-rectal; and (d) the bilateral ischio-rectal (posterior horseshoe).

(a) *The blind external ischio-rectal fistula* is probably the only fistula which is not due to infection from within the rectum. Sitting upon a cold seat or riding upon a wet saddle may so reduce the vitality of the ischio-rectal fat that it readily falls a victim to any micro-organisms that may be circulating in the blood and becomes the seat of an abscess, much in the same way as a perinephric abscess develops as the result of blood infection from a septic focus elsewhere. The external opening of this variety of ischio-rectal fistula is usually situated about two inches distant from the anal verge, either in front of or behind the transverse anal line. The main track curves backwards towards the interval between the sphincters in the middle line, but there is no communication with the rectum (fig. 666).

(b) *The blind internal ischio-rectal fistula* is always the result of infection from within the rectum. The initial lesion is either an abrasion of the mucosa, a fissure, a tear at the base of the pedicle of a fibrous polypus, or an ulcer in one of the large sinuses of Morgagni situated on either side of the middle line of the anal canal. The internal opening, which is often large, is situated in the middle line posteriorly between the sphincters (fig. 667), and discharges pus freely. As there is no external opening, and as contraction of the external sphincter, occurring reflexly on account of the pain occasioned by the

inflammatory process, prevents free escape of pus through the internal opening, this variety of ischio-rectal fistula is usually associated with the signs and symptoms of an acute ischio-rectal abscess.

(c) *The complete ischio-rectal fistula* is a later stage of the preceding variety (fig. 668). Extensions from the main track lead to the formation of multiple secondary external openings.

(d) *The bilateral ischio-rectal fistula* is the result of a septic lymphangitis extending to the opposite side from the original focus. This is more likely to occur when the internal opening of a blind

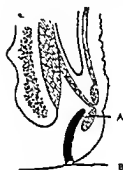


Fig. 666.

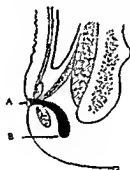


Fig. 667.



Fig. 668.

Fig. 666.—A BLIND EXTERNAL ISCHIO-RECTAL FISTULA.

A—The blind extremity pointing towards the interval between the sphincters.  
B—The external opening.

Fig. 667.—A BLIND INTERNAL ISCHIO-RECTAL FISTULA.

A—The internal opening between the sphincters.  
B—The blind extremity approaching the skin surface.

Fig. 668.—A COMPLETE ISCHIO-RECTAL FISTULA.

A—The internal opening between the sphincters.  
B—The external opening on the skin surface.

internal or of a complete ischio-rectal fistula is exceptionally large, such as that produced by laceration of the pedicle of a fibrous polypus attached to a valve of Morgagni. Under such circumstances faecal matter readily enters the main track of the fistula and sets up active suppuration. In consequence of this, numerous offshoots are formed which terminate in secondary external openings. In those instances of bilateral ischio-rectal fistulae which have multiple secondary external openings on both sides, it is not uncommon to find that a deeply situated offshoot passes forwards beneath the inferior hæmorrhoidal vessels as they cross the ischio-rectal fossa, and terminates in an external opening in front of the anus close to the middle line. Should

a similar offshoot exist on the opposite side, the anus becomes surrounded by the fistulous track. A blind internal fistula may exist on one side and a complete fistula on the opposite side, or both of them may be complete and symmetrically disposed in regard to the course taken by the main tracks (fig. 669, A, B). Even when several secondary external openings exist on both sides, there is never more than one internal opening present, which is situated in the middle line posteriorly between the sphincters (fig. 669, D).

*Differential Diagnosis.* The characteristic features of a complete ischio-rectal fistula are: (1) the primary external opening is usually situated posteriorly to the transverse anal line and at a greater distance than an inch and a half from the anal verge; (2) the internal opening is always in the middle line posteriorly, at the level of the interval between the internal and external sphincters; and (3) the induration is confined to the ischio-rectal fossa and does not extend above the level of the levator ani.

The position of the primary external opening serves to distinguish the ischio-rectal fistula from the submucous, the sub-sphincteric, and the retro-rectal, because each differs in regard to its distance from the anal verge. The primary external openings of the para-rectal and the pelvi-rectal fistulæ, however, occupy a similar position to that of the ischio-rectal, so that the diagnosis rests upon the direction taken by the main track and the position of the internal opening. The main track of an ischio-rectal fistula pursues a course parallel to the skin surface, curving backwards towards the middle line, and terminates at an internal opening situated in the middle line between the sphincters. The main tracks of the para-rectal and the pelvi-rectal fistulæ are parallel to the longitudinal axis of the rectum and extend deeply into the pelvis for a distance of several inches.

*Clinical Course.* So long as the primary external opening of an ischio-rectal fistula remains patent and the internal opening is small, the formation of offshoots from the main track is not likely to take place. When, however, the internal opening is large, faecal matter is apt to find its way into the main track and set up active inflammation, so that secondary abscesses are formed which discharge their contents through secondary openings upon the skin surface. Moreover, infection of the lymphatic channels of the opposite ischio-rectal fossa may ensue and lead to the development of an ischio-rectal fistula on that side. On account of the symmetrical disposition of the lymphatics, the

resulting fistula is a replica of the original fistula and communicates with it by means of a track which passes beneath the ano-coccygeal ligament.

*Operative Technique.* A blind external ischio-rectal fistula may in some instances be cured by laying open the main track from the external opening as far as its blind extremity, together with any offshoots that may exist, and then increasing the surface area of the wound to a suitable extent by means of relieving cuts. If, however, the blind end of the track passes beneath the external sphincter muscle so that only mucous membrane intervenes between it and the anal canal, time will be saved by converting the blind external fistula into a complete fistula, by forcing the point of a probe-director through the mucosa between the sphincters and then liberating the director by dividing the tissues covering it.

When operating upon a blind internal ischio-rectal fistula, the exact position and extent of the main track should be ascertained by introducing the finger into the rectum and palpating the ischio-rectal fossa between it and the thumb placed upon the skin surface. An incision is then made into the indurated area (occasionally fluctuant if active suppuration is progressing) radiating outwards to the required extent, as shown in figure 661, and then the main track is laid open in accordance with the method advised for a complete ischio-rectal fistula.

When the fistula has become complete, the primary external opening indicates the surface termination of the main track, so that, in order to ensure that the main track is laid open, the probe-director should be introduced into the primary opening and not into the secondary opening if one exists. Since the main track of a complete ischio-rectal fistula always takes a curved course backwards towards the internal opening (fig. 664, D or E), it is not possible to introduce the probe-director far enough along it in order to reach the internal opening, on account of the bend in its course. The probe-director should therefore be gently passed along the track as far as it will go, and when it is found that further progress is impeded, it should be forced through the skin at that point. The overlying tissues are divided and then the probe-director is re-introduced as far as it will go and again forced through the skin if it does not readily reach the internal opening.

After dividing the overlying tissues, if a portion of the track still remains undisclosed, the above manoeuvre is repeated until the point of the probe protrudes through the internal opening. When the main

track has thus been completely exposed from end to end the surface area of the wound should be increased by making relieving cuts at points where there is most tension on the edges of the main incision. A careful search is then made for offshoots from the main track. Superficial offshoots should be laid open from end to end, but when a deep offshoot is discovered, extending forwards beneath the inferior hæmorrhoidal vessels, it should not be laid open as, by so doing, the nerve supply of two-thirds of the external sphincter would be cut off, and some measure of incontinence will result. Under such circumstances the portions of the offshoot behind and in front of the vessels should be laid open, but the part beneath them must be kept intact.

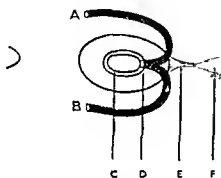


Fig. 669.

Fig. 669.—A COMPLETE BILATERAL ISCHIO-RECTAL FISTULA. NOTE THAT THERE IS ONLY ONE INTERNAL OPENING

A and B are the symmetrically placed external openings situated anteriorly to the transverse anal line  
C—Interval between sphincters. D—Internal opening E—Ano-coccygeal ligament. F—Coccyx.

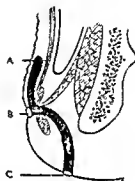


Fig. 670.

Fig. 670.—A COMPLETE ISCHIO-RECTAL FISTULA, WITH A SUBMUCOUS EXTENSION TERMINATING IN A CUL-DE-SAC

A—Cul-de sac of the submucous extension from the main track.  
B—The internal opening between the sphincters.  
C—The external opening upon the skin surface

When operating upon a bilateral ischio-rectal fistula (posterior horseshoe) all the existing tracks on both sides, as well as the track passing beneath the ano-coccygeal ligament, should be laid open completely, but the part of the main track, which passes beneath the external sphincter to the internal opening, should not be laid open until the greater part of the wound has been filled in. Lastly, a careful search should always be made for a submucous extension above the internal opening (fig. 670).

*After-treatment.* The after-care of an ischio-rectal fistula is practically identical with that of a para-rectal fistula. The packing in the wound is removed on the fifth day and the wound cavity is

thoroughly irrigated with a strong antiseptic solution. Surfaces of the wound, which tend to make contact with one another, should be kept apart by inserting a thin layer of cotton wool between them. On no account should the cavity of the wound be repacked.

At the expiration of ten days after the operation, the surface of the wound will usually be found to be covered by healthy granulations and there should not be any appreciable purulent discharge. Should pus collect in the wound, it should be regarded as evidence that active suppuration is taking place somewhere in the neighbourhood, and accordingly a careful search should be made for offshoots from the main track of the fistula, which may have escaped detection when the operation was being performed or had developed subsequently.

There are three situations where search should always be made under such circumstances, namely : (1) posteriorly, for a track extending to the opposite side beneath the ano-coceygeal ligament ; (2) anteriorly, for a track passing forwards and inwards beneath the inferior hæmorrhoidal vessels ; and (3) for a track extending upwards beneath the mucosa from the site of the internal opening, which is always situated in the middle line posteriorly, between the sphincters.

As soon as the presence of an actively suppurating track has been disclosed, immediate steps should be taken to deal with it so as to prevent further extension. Thus, when an offshoot is found to extend to the opposite side, it should be laid open completely from end to end, relieving cuts being made at suitable points along the edges of the resulting wound.

In the event of a submucous extension being discovered, the method recommended for dealing with a submucous fistula should be adopted. When, however, a track is found extending beneath the inferior hæmorrhoidal vessels, it should not be laid open completely to the skin surface, because the nerve supply of the anterior two-thirds of the external sphincter muscle would thereby be destroyed. A small crucial incision should therefore be made over the anterior extremity of the track, in order to provide a free exit for the pus, and then the portion of the track beneath the vessels should be thoroughly cauterised with nitrate of silver. Several such applications may be necessary, at intervals of three or four days, before the track beneath the vessels is obliterated.

## CHAPTER VI

### STRICTURE OF THE ANUS AND OF THE RECTUM

By the term *stricture* we understand that a *permanent* abnormal narrowing of the calibre of the bowel has been produced. Strictures, such as are here meant, are produced either by a malignant growth or by cicatricial tissue. Some authors designate a variety of stricture which they attribute to muscular spasm, *spasmodic stricture*; but, as this is not permanent, it is not taken into consideration here. Strictures due to malignant disease will be dealt with in the chapter on malignant growths. The following remarks relate solely to cicatricial or fibrous stricture.

*Cicatricial or Fibrous Stricture.* This form of stricture may be met with in any part of the rectum, but its lower margin is most usually found at a level of from one and a half to two inches above the anal margin. It is frequently observed, during a digital examination, that the lowermost margin of the stricture is felt when the finger has been introduced nearly as far as the second joint, i.e. about one and three-quarter inches above the anal orifice. The next situation in order of frequency is at the anal orifice itself, and the least common at the upper part of the rectum.

*Varieties of Stricture.* There are two distinct varieties of cicatricial stricture, namely, the *linear* or *annular stricture* and the *tubular stricture*.

#### THE LINEAR OR ANNULAR STRICTURE

This does not exceed a depth of a quarter of an inch, and is usually found either at the anal orifice or at the level of the upper border of the internal sphincter muscle, and is generally due to traumatism.

When a stricture of this nature is met with *at the anal orifice*, it has nearly always been produced by a too free removal of the anal skin. Consequently in such cases expansion of the anus cannot occur to a



natural extent during defæcation. The degree of limitation of expansion varies in different cases, in some instances being so marked that the little finger cannot be introduced through the anal orifice. The unyielding scar tissue does not extend deeper than the skin, and therefore the external sphincter is not directly concerned in the stenosis. In fact, the anal orifice is simply prevented from expanding by an unyielding covering.

When the stenosis is situated at the upper border of the internal sphincter, i.e. about one inch and a quarter from the anal margin, it is sometimes due to a puckering of the mucous membrane produced by the cicatrization of the granulating surfaces left after the separation of the sloughs of ligatured internal piles. Such puckering only occurs if cohesion of the lateral instead of the upper and lower margins of a granulating area be allowed to take place and is therefore readily preventable by careful treatment during the healing process, as mentioned in the treatment after the operation by ligature of internal piles (see page 1286).

When an annular stricture is located two and a half or more inches above the anal margin, it is sometimes due to the destruction of the mucous coat and submucous tissue by an excessive application of nitric acid or other caustic to the apex of a procidentia of the rectum.

*Symptomatology.* In all cases of annular stricture there is an increased frequency in the action of the bowels, very small quantities of feces being passed at a time. An obstruction to the passage of the feces is complained of, and, after an action has taken place, there is a well-marked sensation that the rectum has been incompletely evacuated. As a result, the colon is always full of fecal material, and the abdomen becomes greatly distended, especially after breakfast and afternoon tea, though the discomfort is increased after partaking of any kind of food. Digestion becomes impaired and the patient gradually loses weight. More or less complete loss of control over the rectal contents is manifest, a discharge consisting of liquid feces and rectal mucus more or less continuously escaping from the anus. The above symptoms are met with in strictures situated at the anal orifice and at or about the level of the upper border of the internal sphincter. When an annular stricture is situated higher up in the rectum, invagination of the rectum is generally produced and therefore the symptoms characteristic of the latter condition are superadded, the only difference being that when the intussusception protrudes from the anal orifice during

straining at defæcation, spontaneous reduction occurs after the straining effort has ceased.

When the stricture is situated at the anal orifice, impaction of fæces in the rectum is usually met with, the faecal mass distending the rectum and acting like a ball-valve, so that only liquid fæces are passed, and of this there is an almost constant escape. When this condition exists the patient sometimes describes his condition as that of continuous diarrhoea.

*Pathological Anatomy.* When the seat of constriction is *at the anal orifice*, inspection will reveal a scarcity of anal skin. The natural rugæ are absent and there is no redundancy. When the patient is requested to strain down, a characteristic tense appearance of the margin of the orifice is observed, showing that the limit of expansion has been reached. The integument covering the external sphincter muscle is of a whiter colour than the normal on account of the cicatricial tissue present. When an attempt is made to introduce the finger into the anal canal, the margin of the orifice is found to be firm and unyielding. Straining efforts on the part of the patient force the anal region down, making it prominent, but do not produce relaxation of the anal orifice. The degree of constriction varies in different cases, in some being so marked that it is impossible to introduce the finger through the orifice without causing pain. If the finger can be introduced into the cavity of the rectum, an impaction of fæces is nearly always found to be present.

When the constriction is situated *at the level of the upper border of the internal sphincter*, the anal orifice is generally somewhat patulous. There is often œdema of the anal skin. There is no spasm of the external sphincter, that muscle being relaxed. When the finger is introduced into the anal canal, a diminution of its calibre is met with as soon as the insertion has extended to the first joint. The diameter of the stenosed area is generally large enough to admit the tip of the finger, so that the length of the stricture can be ascertained. This usually measures from one-eighth to one-quarter of an inch, and feels like a stout piece of cord encircling the bowel. The mucous membrane both above and below the stenosed area is smooth, and there is no discharge unless the finger has been forced through the stenosis, thereby causing slight bleeding and much pain. The stricture itself is found to consist of a puckering of the mucous coat and submucous tissue only. The constriction yields to dilatation so that it is often possible to introduce the examining finger to its full extent. When this can be

done, feces will often be found above the constriction, the rectum dilated, and the mucous coat smooth and healthy to the touch, showing that the diseased area is very limited.

When the stenosis is met with *at a level of from three to four or more inches above the anal orifice* there is generally some invagination of the rectum, the stricture in such cases being situated at the apex of the intussusceptum. Such an intussusceptum may or may not be protruded from the anal orifice. The existence of a firm unyielding margin to the orifice of the intussusceptum is sufficient evidence to enable us to attribute its cause to the stricture.

*Treatment.* Annular strictures can often be permanently cured by the methods which will now be described, the particular procedure differing according to the seat of the stricture.

(a) *When the Stenosis is situated at the Anal Orifice.* The cicatricial tissue in this situation is of a particularly unyielding character, attempts at forcible dilatation being liable to cause one or more lacerations of the circumference, thus producing much pain and also predisposing to the formation of an abscess. Consequently, treatment by bougies is not advisable. The object to be kept in view is to increase the diameter of the contracted orifice, without causing any permanent damage to the part. I have found that the best way of effecting this is to increase the circumference by dividing it in one or two places, and ensuring that healing occurs by granulation and the formation of a wide scar. In this way the circumference may be increased by half an inch even with one incision, thus overcoming the stenosis which is partly due to the œdema caused by the irritation of the constricted orifice. An incision, therefore, should be made in the right posterior quadrant of the contracted anal orifice, and should extend completely through the external sphincter muscle but not beyond it. The tension at the circumference causes the wound to gape widely, the diameter of the passage being thus considerably increased. If additional increase be required an incision should subsequently be made in the left anterior quadrant (i.e. diametrically opposite to the first incision), but this must be confined to the skin and subcutaneous tissues only. It is quite unnecessary, and in women inadvisable, to divide the external sphincter in two places. The wounds should be made to granulate slowly from the bottom by careful packing, in order that the resulting scar shall be as wide as possible. In fact, the scar should be wedge-shaped, and, when healing has taken place, the base of the wedge should measure

from three-eighths to half an inch in width. In order to obtain a scar of requisite width, the wound must be firmly packed, which sometimes causes much discomfort, but this can generally be relieved by soaking the material used for packing in a 20 per cent solution of cocaine.

When healing has been completed the scar if left to itself will contract from side to side, so that a scar measuring a quarter of an inch in width may eventually measure only one-eighth of an inch. If, however, the contraction of the scar be made to take place vertically instead of transversely, an increase instead of a diminution in the width of the scar can be obtained. Such vertical contraction can be ensured by introducing a vulcanite tube three-quarters of an inch in diameter and two and a half inches long into the rectum every night at bed-time, and leaving it *in situ* for half an hour or as much longer as the patient is able to bear. The use of the tube should be continued every night for about a month, then twice a week for a similar period, and, finally, once a week until all tendency in the scar to contract has subsided, a result which is usually attained in from three to four months after the operation. In order to ascertain whether the result of this method of treatment has been satisfactory, the left index finger should be introduced into the anal canal as far as the distal interphalangeal joint, and then the index finger of the right hand passed along it for a similar distance, the palmar surfaces of the two fingers being in contact. If this can be done without creating a feeling of tension or causing pain to the patient, it may be safely assumed that the diameter of the anal orifice is sufficient for the purpose of natural defæcation and that undue straining to expel even firm fæces is no longer necessary. If, at the expiration of six months, such an introduction of the two fingers be still possible, a permanent cure will have been effected. Should some degree of stenosis have recurred during the interval, the treatment described above should be repeated.

(b) *When the Stenosis is situated at the level of the Upper Border of the Internal Sphincter.* A stricture in this situation, unless of long standing, yields readily to gradual dilatation. In recent cases this method is generally all that is necessary, though in some cases supplementary division of the scar tissue in one or more places may be required. The duration of the stenosis is an important factor in its amenability to dilatation. Originating as a result of cohesion of the lateral margins of the granulating surfaces left after the separation of the sloughs of ligatured internal piles, it is obvious that the bond of union becomes tougher and more resistant as time goes on. If stenosis

supervenes after the operation upon internal piles by ligature, symptoms referable to the condition may be manifested within two or three weeks after the operation, and therefore an early diagnosis of the condition can be made. In such recent cases the passage of the index finger through the stricture by slow and gentle insinuation is nearly always possible at the first examination. When the finger has been inserted into the rectum to its full extent it should be withdrawn, no further dilatation being made on that occasion. This procedure should be repeated every other day for a week, then once a week for a month, and, finally, once a fortnight for two months. At the end of this period the stenosis will have usually disappeared and, as a rule, will not then tend to recur. It is unnecessary to dilate such a stricture beyond the diameter of the index finger. Bougies or tubes are not often required in the treatment of these cases. When they are used the greatest gentleness must be employed in their introduction, lest the stricture be torn and an abscess result therefrom.

When the stricture comes under observation after an interval of three or four months from the date of the operation, it does not so readily yield to digital dilatation. Therefore, in such cases, either bougies or, still better, vulcanite tubes about two and a half or three inches in length and from half to five-eighths of an inch in diameter should be used, or the stricture should be divided on the right and left sides and in the middle line posteriorly, the angles of each incision being sutured together with either chromicised catgut or silkworm-gut. For this purpose either a rectal bivalve or a vaginal duck-bill speculum will greatly facilitate the application of the sutures. Each incision should not exceed half an inch in length. When silkworm-gut is used the ends should be left long enough to protrude from the anal orifice in order that they may not irritate the rectum.

(c) *When the Stenosis is situated at a level of three or more inches above the Anal Orifice.* The treatment to be adopted depends upon the presence or absence of invagination.

When invagination is present the stricture should be divided, if possible, on its right and left sides and in the middle line posteriorly. These three incisions should extend completely through the stricture and into healthy mucous membrane for about one-eighth of an inch both above and below the stricture. The angles of each incision should then be sutured together, thus increasing the circumference of the lumen of the rectum at the seat of stricture from one to one and a half inches. When the invagination in these cases frequently protrudes

through the anal orifice it is desirable, in order to prevent a continuance of the protrusion, to perform left iliac colostomy before operating on the stricture itself. After the stricture has been successfully treated the colostomy may be safely closed.

When invagination is absent and the stricture is so high that it cannot be reached for incision, its dilatation must be attained either with vulcanite tubes or with soft elastic bougies.

#### THE TUBULAR STRICTURE

In this variety the length of bowel involved is usually from a half to one inch, but occasionally the constriction may extend to three, four, or more inches. It is met with more often in women than in men, and is practically incurable, though much can be done to relieve it. Tubular strictures under one or two inches in length are chiefly met with in women during the child-bearing period of life. In these cases the constriction usually begins about one and a half inches from the anal orifice, that is, near the level of the reflection of the peritoneum from the anterior wall of the rectum.

The more extensive tubular strictures are met with as often in men as in women, and at any age (fig. 671). It is probable that they are always caused by infective ulceration of the rectum.

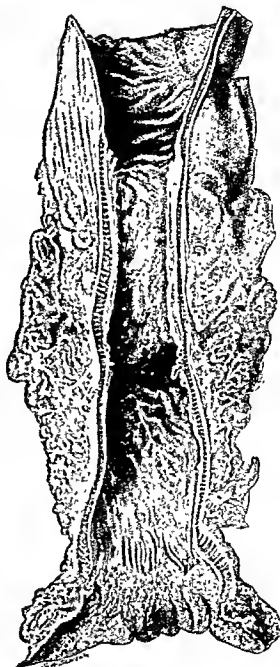


Fig 671.—A TUBULAR STRICTURE OF THE MIDDLE OF THE RECTAL AMPULLA. FOR A DISTANCE OF TWO INCHES THE MUCOSA HAS BEEN DESTROYED THROUGHOUT THE CIRCUMFERENCE.

*Ætiology.* Stricture is due to the contraction of fibrous tissue

deposited in the rectal wall. This fibrous tissue is either the medium by which some definite loss of tissue has been repaired, or the organised residue of an inflammatory infiltration. Consequently the precursors of this form of stricture are (a) *local tissue destruction*, and (b) *chronic inflammatory processes*.

(a) *Local Tissue Destruction*. This may be the result of *traumatism* or *ulcerative* processes. Of the former, especial mention must be made of too free removal of tissue during operative procedures. Thus, during the operation for internal piles a too free removal of the folds of perianal skin, or the too long continued application of the cautery when removing hæmorrhoids together with some peri-anal skin by that method, may result in definite narrowing of the anal orifice. In the same manner, when a complete ring of anal mucous membrane has been removed, stenosis may occur as a result of the healing process. This is liable to take place after Whitehead's operation for piles should the stitches, which unite the mucous membrane to the skin, cut out and allow the mucous coat to retract. The best example perhaps of stricture of traumatic origin is that which sometimes results from excision of the whole circumference of the lower part of the rectum when colostomy has not been previously performed. Here, a large surface has healed by granulation, with the result that a stenosis of a very unyielding character occurs. Sloughing of the rectum, either as a result of injury or of acute inflammatory processes, produces the same result, unless colostomy has been performed while the sloughing is in progress or very soon after the wound has begun to heal.

All ulcers heal by granulation and are therefore liable to cicatricial contraction. A small ulcer situated in the rectal wall may not cause an appreciable narrowing of the lumen when cicatrized, but when the ulcer has extended completely round the circumference of the bowel, cicatrization is certain to end in stenosis. The length of the ulcerated area determines the length of the strictured zone, and consequently the longer strictures are generally the result of extensive ulceration. The extensive destruction of the mucous and submucous coats of the rectum resulting from infective ulceration frequently gives rise to strictures extending to two or more inches in length.

(b) *Chronic Inflammatory Processes*. These include the manifestations of syphilis and inflammations occurring in neighbouring viscera, especially the uterus.

*Syphilis* is considered by many authorities to be the cause of extensive stricture of the rectum, but the accuracy of this view may be

questioned. In the first place, it is found that anti-syphilitic remedies have no effect whatever in either curing or relieving the stricture or the ulceration associated with it. One would not, perhaps, expect to find that the fibrous deposit became entirely absorbed under the influence of iodide of potassium, but the active ulceration which is often found to co-exist with stricture ought to be benefited by it. Large doses of iodide of potassium have been tried repeatedly, both alone and combined with mercury, but no improvement has taken place in the ulceration or the stricture. Again, though a certain number of patients with stricture are undoubtedly syphilitic, there are a considerable number in whom neither a history of syphilis nor any other evidence of the disease, such as a positive Wassermann reaction, can be obtained, and in these patients it does not seem justifiable to assume the presence of the syphilitic taint. Moreover, stricture occurs much more frequently in women than in men, while in syphilis the converse obtains. It seems, therefore, that a great many of the strictures that are set down as syphilitic are not so, but are the result of *infective ulceration*. At the same time it cannot be said that stricture of the rectum does not occur as a result of syphilis.

*Chronic inflammation of the uterus* is probably an important factor in the causation of stricture of the rectum. In this connection it has repeatedly been observed that cases of stricture occur at least five times as frequently in women as in men, and almost invariably during the child-bearing period, i.e. during the third and fourth decades of life. Moreover, in these patients, a history of either a pregnancy or a miscarriage is usually obtainable. It is probably due to the latter fact that syphilis is thought to play such an important part in the production of stricture of the rectum. Syphilis, however, is not the only cause of abortion, and therefore it by no means follows that because a woman has had miscarriages she is necessarily the subject of syphilis. Chronic endometritis is often the result of abortion, and consequently it is justifiable to assume that when a woman gives a history of miscarriages, she has also suffered from inflammation of the uterus.

It is a fact that the development of stricture of the rectum is often preceded by either a miscarriage or parturition, and therefore the question naturally arises, *What is the connection between the two events, and does stricture of the rectum result from inflammation of the uterus?* The following case throws some light upon this point. When performing abdominal hysterectomy for intractable metritis, it was noticed that the surface of the rectum, just above the line of reflection of the peritoneum, was thickened, and its coats for an inch or more in length felt



indurated. On making a subsequent rectal examination, a distinct narrowing of the lumen of the bowel at a level of two and a half inches from the anal margin could be felt. The mucous membrane of the rectum was healthy, and there was no ulceration in the strictured zone. From anatomical considerations, it appeared probable that a round-cell infiltration of the tissues had occurred along the course of the lymphatic vessels as they passed round the rectum from the uterus to enter the retro-rectal lymphatic glands. It is easy, therefore, to understand how chronic inflammation of the uterus may be one of the chief causes of stricture of the rectum in women. Moreover, this hypothesis explains why stricture of the rectum supervenes without previous ulceration or other loss of tissue.

*Symptomatology.* During the early stage of stricture the first symptom complained of is increasing frequency in the desire for an action of the bowels, followed by the passing of only a small quantity of fæces, and an after-feeling of incomplete relief. After an interval of a few minutes the urgent desire again returns, such a cycle of events often recurring three or four times in the course of the first hour or two after taking warm fluids. As contraction progresses these symptoms become more frequent, and the patient notices that the fæces are passed in short and round or flattened tape-like pieces. When the stricture has been preceded by ulcerative processes, the discharge of pus or pus and blood with mucus is associated with the above symptoms.

The symptoms indicating the presence of a well-developed stricture are: (a) *frequent desire for an action of the bowels*; (b) *alteration in the size and character of the fæces passed*; (c) *the passage of blood, mucus, and pus from the rectum*; (d) *abdominal distension*; (e) *loss of weight*; and (f) *œdema of the lower extremities*.

(a) *Frequent Desire for an Action of the Bowels.* In cases of stricture the desire to evacuate the rectum is urgent, and is relieved by passing either discharge or fæces, or both. The quantity of the evacuation is usually very small and the patient feels that the bowels have not been completely relieved. This is the most constant symptom of stricture of the rectum, and it is always present when the diameter of the lumen of the bowel has been reduced to less than half an inch. Its presence denotes that there is some degree of obstruction to the passage of fæces through the rectum, but throws no light upon the nature of the obstruction, that is to say, whether it is due to fibrous stricture, malignant stenosis, or a neoplasm blocking the lumen of the bowel.

The association of other symptoms, together with digital exploration, alone can decide that point.

The history is usually obtainable that an action of the bowels takes place from ten to fifteen times, or even more frequently, during a period of twenty-four hours. The majority of these actions occur during the day-time, but, as a rule, two or three take place during the night. These nocturnal actions are so common in cases of fibrous stricture that the question of their occurrence should always be raised when the presence of the disease is suspected, and, when they are complained of, the rectum should invariably be examined. The first action occurs as soon as the patient rises in the morning. Another takes place immediately after breakfast. During an hour or so after that meal there may be three, four, or even more actions at short intervals. Fæces are voided, as a rule, at the first and second actions only, the dejecta in the remainder consisting chiefly of mucus, or mucus mixed with pus and blood. Usually after each meal an action of the bowels occurs, and if warm fluids have been partaken of, there may be several during the first hour. With the evening actions fæces may be again voided, but of the total number of actions it is unusual for the dejecta to be faecal on more than three or four occasions during a period of twenty-four hours. The desire for an action of the bowels is always urgent, the contents of the rectum being usually expelled with considerable force.

(b) *Alteration in the Size and Character of the Fæces Passed.* In nearly all cases of stricture the fæces when firm are voided in short tape-like pieces, measuring from one inch to an inch and a half in length, and about half an inch in diameter. These differ from the characters of the firm fæces passed in cases of spasmodic action of the sphincters and levatores ani met with in hypertrophy of the external sphincter associated with pruritus ani and fissure. In those cases the fæces are passed in thin round or flattened pieces, varying from two to four or more inches in length and from about half an inch in diameter to the full size natural to the patient.

(c) *The Passage of Blood, Mucus, and Pus from the Rectum.* This is an almost constant symptom, the material evacuated at all actions of the bowels, excepting the first one or two in the morning and the one or two actions late in the afternoon, consisting of a combination of mucus, pus, and blood. The quantity evacuated is generally about one or two teaspoonfuls. If the quantity of pus suddenly increases, the probability is that a blind internal fistula, either of the submucous,

the para-rectal, or the ischio-rectal type has formed as one of the results of the stricture. As a rule, the quantity of blood lost is slight, a feature which serves to distinguish the fibrous from the malignant stricture. When a para-rectal abscess has formed, the purulent discharge will continue to be profuse, even after the stricture has been well dilated. In the majority of cases, however, the abscess is either of the submucous or the ischio-rectal variety.

(d) *Abdominal Distension.* The tendency of every fibrous stricture is towards obliteration of the lumen of its portion of the bowel. Consequently the obstruction to the passage of the contents of the colon becomes more and more marked the longer the stricture remains untreated. The large intestine becomes chronically distended from flatulent and faecal accumulation, and may reach an enormous size. As a result of this, the abdomen is nearly always considerably distended as soon as the lumen of the bowel has been sufficiently narrowed by the stricture to prevent healthy evacuation of the colon. One of the most marked features of this distension is the accumulation of faeces in the caecum. Flatulent distension is more complained of than faecal accumulation, and is very distressing to the patient, especially during the first two hours after food has been taken.

(e) *Loss of Weight.* As soon as the effects of a stricture begin to make themselves felt, the patient commences to lose weight. This is probably due to general digestive disturbances, with consequent mal-assimilation. The frequent actions of the bowels, and especially the disturbance of rest at night, have, however, a good deal of influence in producing loss of weight. In the more advanced stages of the disease, the patient may be greatly emaciated, especially noticeable in the arms and legs.

(f) *Oedema of the Lower Extremities.* The pressure of the faeces in the distended colon above the seat of stricture sometimes causes a gradually increasing oedema of either one or both lower extremities. The left lower extremity is more commonly affected than the right.

*Pathological Anatomy.* The anus is generally patulous and several more or less oedematous redundant folds of anal skin are usually present. The peri-anal region in some cases is natural in appearance, but in others there is extensive scarring, particularly when fistulae resulting from the stricture have been operated upon, or when the stricture is the result of infective ulceration.

On introducing the finger into the rectum, the sphincter muscles will be found to be deficient in contractile power. The lower margin of the stenosis will usually be met with at a level of about from one and a half to two inches above the anal orifice. If the finger can be passed into the stenosed area it may be possible to determine the length of the stricture. Occasionally the diminution in the lumen is so great that it is impossible to insinuate the finger into the stricture without using undue force. Under such conditions the length of the stricture must be determined by other means. In women, palpation of the recto-vaginal septum from the vagina will often greatly assist in the estimation of the length of a stricture. In men, olive-headed bougies or similar instruments may with advantage be used for this purpose.

If the finger can be introduced into the stenosed area, the bowel will be found to have been converted into a more or less rigid tube, the internal surface of which is firm and irregular. In all long-standing cases, and especially in those in which the stricture has been the result of previous ulceration, the mucous membrane of the bowel immediately above and below the stenosed region is ulcerated. From these surfaces there is a continuous secretion of pus, and, therefore, when the finger is withdrawn, it will generally be found to be smeared with pus and blood.

When the stricture is short, and the finger can be introduced through it into the rectum above, the cavity of the latter will generally be found to be distended with fæces. If there be much thickening in the vicinity of the rectum, either pelvi-rectal suppuration has probably occurred or an ischio-rectal abscess has formed as a result of the stricture. The co-existence of a para-rectal or an ischio-rectal abscess is substantiated if pus escapes in considerable quantity either during or after an examination.

*Treatment.* The treatment of a tubular stricture of the rectum should be both local and general.

(a) *Local Treatment.* The treatment of a tubular stricture is not radical, because a permanent cure cannot be effected. The fibrous deposit in the rectal wall cannot, by any known means, be entirely removed; and, therefore, unless active treatment be continuously persevered with, a recurrence of the stenosis results. In this respect tubular strictures of the rectum are comparable to strictures of the urethra. When, therefore, a patient presents himself for treatment and is found to be suffering from the *tubular* variety of stricture he

should he told that his disease cannot be cured though considerable relief of his symptoms and, perhaps, immunity from serious sequelæ can be obtained by persistent treatment throughout the remainder of his life.

In tubular strictures of the rectum the treatment by *dilatation* is the safest and best. Other methods, such as division of the stricture (*posterior linear proctotomy*), resection of the stenosed portion of the rectum, and excision of the rectum itself are practised by some but have not found favour with the majority of surgeons. In some of the extensive strictures resulting from infective ulceration of the rectum which cannot be much relieved by continuous dilatation, left iliac colostomy is the only measure which will prolong and possibly save the patient's life.

*Treatment by Dilatation.* Dilatation of a tubular stricture may be accomplished either forcibly or gradually. Therefore the two methods are : (1) *forcible or rapid dilatation* ; and (2) *gradual dilatation*.

(1) *Forcible or Rapid Dilatation.* This method cannot be recommended, and should never be resorted to. When attempted, either by inserting bougies of gradually increasing diameter, one after the other, as when dilating the cervix uteri, or by employing Todd's dilator or one of the powerful expanding metal instruments designed for the purpose, there is considerable risk of splitting the strictured portion of the bowel, and so permitting peri-rectal extravasation. The result of such an accident depends upon the situation of the stricture. If the bowel be ruptured below the level of the peritoneal reflection, extensive peri-rectal suppuration, terminating in a para-rectal abscess, may supervene ; but, if the stricture be situated above the peritoneal reflection, fecal extravasation into the general peritoneal cavity ensues and will be followed by diffuse peritonitis and death.

(2) *Gradual Dilatation.* This is by far the safest and best method, and should always be tried and persevered with before resorting to any other form of treatment. The dilatation is accomplished either by the use of bougies or tubes, and is in several respects comparable to the methods employed for the gradual dilatation of urethral strictures. The selection of the bougie or the tube for the purpose depends upon the nature of the stricture. In short straight strictures situated near the anal orifice, the tube possesses the distinct advantage of being retainable *in situ* for a considerable length of time without causing

discomfort to the patient, because of the ready escape afforded for flatus, discharge, etc.

When the stricture is long and tortuous, the tube cannot be used. In such cases a bougie of the flexible type is indispensable. The shot-bougie is, I think, the best for this purpose, as it can be easily made to follow the tortuosities of the stricture.

Whether tubes or bougies are used for dilating a stricture, the question of primary importance is: *To what extent should the dilatation be carried?* When a stricture has been dilated sufficiently to admit easily an ordinary index finger (i.e. about five-eighths of an inch in diameter), its lumen is quite large enough for the purposes of effective defæcation. Consequently it is seldom necessary to introduce either a bougie or a tube of a larger diameter than three-quarters of an inch, and when the lumen of the stricture is maintained at that size, the actions of the bowels are steadily reduced in number (the nocturnal actions completely ceasing), the abdominal distension is reduced, and the colicky pain resulting from the accumulation of fæces and flatus does not recur. Some surgeons recommend that the dilatation be persevered with until a diameter of an inch and a half has been attained. This is neither desirable nor beneficial to the patient because it is not possible to estimate the limit of possible expansion of a given stricture, and therefore a continuation of the dilatation may result in splitting the wall of the bowel and so setting up peri-rectal suppuration. When the dilatation is carried beyond five-eighths or three-fourths of an inch in diameter, the stenosed area is generally irritated by such further dilatation, and the patient is therefore subjected to much pain and more or less constant discomfort without receiving any compensating benefit.

As soon as a diameter of sufficient size to permit a free evacuation of the contents of the colon has been attained, further increase in the dilatation should be dispensed with and our efforts should be directed simply to preventing re-contraction. In order that the method of gradual dilatation may be successfully carried out, it is essential that the surgeon should be provided with sets of bougies and tubes, the diameters of which increase in the same ratio as in urethral instruments, the smallest measuring three-sixteenths of an inch and the largest five-eighths or three-fourths of an inch at its thickest part. In a given case, the diameter of the stenosis should first be ascertained by introducing successively the smaller instruments until the size that can just be passed easily is reached. This should be left *in situ* for twenty minutes or half an hour and then withdrawn. On the third day it

should be again introduced, and then the next larger size. If the latter passes easily the next larger size should then be introduced, left *in situ* for half an hour and then withdrawn. This procedure should be repeated every third day, commencing with the size that had been left in on the previous occasion, and then introducing one or two larger sizes. When the size of five-eighths or three-fourths of an inch has been reached, it should be reintroduced every other day for a week, then twice a week for a month, then once a week for two months, then once a fortnight for three months, and finally once in three months for the remainder of the patient's life.

(b) *General Treatment.* The general treatment consists in the careful selection of articles of diet which will give the maximum of nutriment with the minimum of excreta. A patient suffering from a tubular stricture should be induced to recognise the fact that errors in diet may at any time cause either impaction of fæces above the stricture or diarrhoea, either of which may lead to perforation of the bowel above the strictured zone. Potatoes and rice, especially the latter, and red wines of all kinds should not be taken. Cod-liver oil, in doses of one to four drachms, taken two or three times a day, is very useful for making the fæces soft. The injection of one or two ounces of olive oil into the rectum at bedtime, the oil being retained all night if possible, is very useful in causing the bowel to act freely and easily once or twice a day without the use of any other aperient.

*Results of Stricture.* The following are three of the most important sequelæ which result from the neglect of a stricture of the rectum, namely (1) *intestinal obstruction*; (2) *para-rectal suppuration*; and (3) *amyloid degeneration of the kidneys*.

(1) *Intestinal Obstruction.* This is usually of the chronic type, and is due to the gradually increasing difficulty in evacuating the contents of the colon. The cæcum, as a rule, is greatly distended with fæces and may, in protracted cases, slough on its anterior surface and give rise either to a localised abscess or to acute general peritonitis. Hyperdistension of the sigmoid colon in some cases leads to rupture of the bowel just above the stricture zone, causing localised pelvic suppuration.

(2) *Para-rectal Suppuration.* This is always due to a perforation of the rectal wall occurring as a result of co-existing ulceration. An

extensive abscess in the para-rectal space may then be formed which may extend and find an exit on the surface after having first invaded the ischio-rectal fossa, or it may rupture into adjacent hollow viscera, such as the vagina or bladder and so establish a communication between them and the rectum. The discharge of pus in these cases is usually profuse, and, if long continued, gradually debilitates the patient and threatens his life.

(3) *Amyloid Degeneration of the Kidneys.* This is always the result of prolonged suppuration. Its onset is marked by the appearance of albumen in the urine, attacks of diarrhœa, steadily increasing anæmîa, loss of weight, and, later on, anasarca. These changes usually begin about three or four years after the commencement of profuse suppuration.



## CHAPTER VII

### BENIGN TUMOURS OF THE ANUS AND RECTUM

THESE tumours are occasionally met with, and present the characteristics of similar growths found in other parts of the body, that is to say, they are of slow growth, they do not infiltrate surrounding structures, and do not, as a rule, tend to recur after removal. Structurally they consist of the more highly developed connective tissue and glandular elements. Thus we have the fibroma, the lipoma, the myoma, the myxoma, the papilloma, and the adenoma. For the sake of convenience, those tumours that are met with at the anus and in its vicinity will be described first, and subsequently those that originate within the rectum.

#### BENIGN TUMOURS OF THE ANUS AND ITS VICINITY

These arise from the skin and subcutaneous connective tissue, and consist of (a) the papilloma; (b) the soft fibroma or fibro-cellular tumour; and (c) the lipoma.

(a) *The Papilloma.* This is due to hypertrophy of the papillary layer of the cutis vera, consisting of elongated wedge-shaped masses of connective tissue, each containing a central artery and vein, and surmounted by ordinary stratified epithelium. The hypertrophied papilla is usually separated at its base from its neighbours by a narrow strip of healthy skin, though the free extremities are often in close contact. Sometimes they are found in separate clusters upon the perianal skin, or they may be so numerous as entirely to surround the anus and exclude it from view. An excellent specimen of this kind of growth is shown in figure 672. In this case the anus was quite hidden from view, though the aperture leading into it was situated in the centre of the mass. The papillæ measured from one-third to three-fourths of an inch in length, and being wedge-shaped (the free extremities corresponding to the base of the wedge), the surface of the tumour

appeared to be that of a minutely lobulated single mass. Upon close examination it was found that each papilla was separate from its neighbours, and that narrow strips of healthy skin intervened between their points of origin. The latter fact was of much value in enabling the diagnosis to be made from epithelioma (squamous carcinoma) of the anus.

The papilloma of the anus is apparently identical with that met with on the glans penis and the labia majora, as a result of the irritating

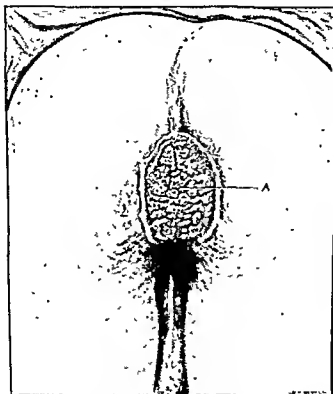


Fig. 672.—AN EXTENSIVE PAPILLOMA OF THE ANAL SKIN COMPLETELY SURROUNDING THE ANAL ORIFICE. THE POSITION OF THE ANUS IS INDICATED BY THE LINE MARKED A. IT IS CONCEALED FROM VIEW.

effect of gonorrhœal discharge (gonorrhœal warts); but there is no reason to believe that those met with in the region of the anus are due to gonorrhœal infection. Want of cleanliness and a continual moist condition of the peri-anal skin appear to be sufficient to produce the papillary hypertrophy. It is met with in both sexes, and usually in young adults.

*Symptoms.* These are chiefly due to the discomfort consequent upon the presence of the tumours, viz. constant irritation and dampness of the part, difficulty in cleansing the anus after defæcation, and

occasional slight bleeding. There is rarely any pain, except when the surface of the tumour is abraded.

*Treatment.* A complete cure can be effected by snipping through each papilla, close to its seat of attachment, with a pair of scissors. There is, as a rule, rather free bleeding from the central artery of each papilla, but this usually ceases under the influence of pressure. Should it continue, a solution of adrenalin may be applied, or the bleeding points may be ligatured. On no account should the skin be removed with the growth lest stenosis of the anal orifice should result. When the growths have been removed, the peri-anal region ought to be kept quite dry for some time by means of a dusting powder of starch and oxide of zinc. If the patient is subsequently careful in regard to cleanliness, and prevents constant moisture of the anus, recurrence does not take place.

(b) *The Soft Fibroma or Fibro-cellular Tumour.* These tumours, identical in structure with those known as molluscum fibrosum when occurring on the neck and trunk, are occasionally seen in the peri-anal region. They are often distinctly pedunculated, and may attain large size. I have seen one which, after removal, weighed 1 pound 11 ounces. This tumour was attached by a stout pedicle to the skin in the right posterior quadrant of the anal region, about  $1\frac{1}{2}$  inches distant from the margin of the anus. The patient, a female, stated that it had been gradually increasing in size for ten years. She would not have presented herself for treatment but for the foul smell and discharge caused by a large gangrenous ulcer situated upon its most dependent surface. Structurally these tumours consist of bundles of white fibrous tissue loosely arranged in a meshwork, the interstices of which contain a serum-like fluid, which readily drains away when the tumour is incised. The pedicle contains blood-vessels of considerable size, which bleed freely when the tumour is removed.

*Symptoms.* Beyond the inconvenience caused by the size of the tumour there are no symptoms, unless the surface becomes inflamed and excoriated through friction against the clothing. When the surface of the tumour becomes ulcerated, as in the instance cited above, there may be free bleeding, as well as an offensive discharge.

*Treatment.* This consists in removal of the tumour. When the pedicle is stout, a small flap of skin should be raised on each side, in

order that the margins of the wound may be brought together without undue tension. All bleeding vessels should be ligatured before the wound is closed. There is no liability to recurrence.

(c) *The Lipoma.* This form of tumour is sometimes met with in the peri-anal region. The tumour is due to hypertrophy of one or more of the lobules of fatty tissue contained in the loculi formed by the peculiar arrangement of the deep fascia in this locality. It increases slowly in size. It can be diagnosed from a collection of pus in the ischio-rectal fossa by the fact that dimpling of the surface is caused by attempting to raise the skin, by the edge of the tumour being felt to slip away from beneath the finger when pressure is made upon it, and by the absence of fluctuation and pain on pressure.

*Symptoms.* It may exist for years without giving rise to any symptoms that direct attention to its presence. Even when of considerable size, it causes inconvenience only by its presence in the vicinity of the anus. Should the surface become inflamed through friction against the clothing, the symptoms attendant upon such a condition make themselves manifest.

*Treatment.* The tumour should be removed by making a free incision over it extending through the capsule. The fatty tissue is then shelled out, care being taken not to leave behind any offshoots from the main mass. The wound should be allowed to granulate. If it be closed with sutures there is the risk of a fistula resulting.

#### BENIGN TUMOURS OF THE RECTUM

These arise in connection with the glandular and connective tissue elements of the mucous and submucous coats of the rectum. They grow slowly, and may exist for several months or even years before giving rise to symptoms indicating their presence. When in an early phase of development, the neoplasm forms a projection under the superficial layers of mucous membrane, and is therefore sessile, but in the course of time the downward traction exerted by the continued passage of feces over the tumour causes the base of attachment to be attenuated into a distinct pedicle which may vary from one or two lines to an inch or more in length (see fig. 674). The pedicle consists of a fold of mucous membrane enclosing the blood-vessels which supply the growth. The pedunculated character of the benign neoplasms of the rectum has

given rise to the term polypus being applied to them. Accordingly when we speak of a rectal polypus we do not mean to imply that the growth possesses any distinctive histological character, but simply that the growth itself, whatever its structure may be, is attached to the rectal wall by a pedicle or stalk.

Several distinct varieties of benign tumour occur in the rectum, namely (a) the adenoma or glandular polypus; (b) the fibroma or fibrous polypus; (c) the villous tumour or villous polypus; (d) the myxoma or myxomatous polypus; (e) the myoma or myomatous polypus; and (f) the lipoma or lipomatous polypus. As the last two of these are rarely met with, and may be regarded in the light of pathological curiosities, only the first four varieties will be described.

(a) *The Adenoma or Glandular Polypus.* This variety of tumour generally arises in connection with the tubular glands (crypts of Lieberkühn) of the mucous coat, being really a localised hypertrophy thereof. The microscopical appearance is that of a number of tubules, lined by ordinary columnar epithelium arranged upon a distinct basement membrane, and bound together by delicate connective tissue. In some cases the connective tissue elements preponderate, and then the growth is spoken of as a fibro-adenoma. A further variety of adenoma (the lymphadenoma), though very rarely met with, consists of lymphoid tissue, and arises from hypertrophy of one of the solitary lymph nodules met with in the mucous membrane.

The adenoma is usually single, but occasionally more than one may occur in the same patient, and in some instances they exist in large numbers (fig. 673). It varies in size from a quarter of an inch to one inch in diameter, is generally rounded in shape, and attached by a slender pedicle, which sometimes measures an inch or more in length. The growth may arise from any part of the rectum, but is usually situated in the lower two inches. When situated low down and particularly when furnished with a long pedicle, it is sometimes protruded through the anal orifice during defecation. As a rule, however, its presence may be undetected for a considerable length of time owing to the absence of symptoms. It is usually found in children under ten years of age, and is seldom met with in adults. The probable reason for this is that when the growth has existed for some time, the pedicle gradually becomes elongated, more and more slender, and is ultimately torn through during an action of the bowels. There can be little doubt that many of these growths are removed from the rectum in this way, a circumstance which explains the cessation of

repeated attacks of hæmorrhage in children as age advances. When these growths are met with in adults, especially in those beyond middle life, they exhibit a tendency to undergo carcinomatous degeneration, especially if they have been subjected to prolonged irritation. A careful microscopical examination of the growth should therefore be made, and if irregularity in the arrangement of the tubules be observed, the patient should be examined from time to time for signs of induration in the neighbourhood of the scar.

*Symptoms.* An adenoma of the rectum, when small, may exist for a considerable length of time before giving rise to any symptoms whatever. As the growth increases in size, it becomes more and more dragged upon during the passage of the fæces, especially when the bowels are constipated, when abrasions of its surface may occur from time to time and give rise to attacks of hæmorrhage. When the pedicle attains sufficient length to allow of the growth descending within the grasp of the sphincters, periodical attacks of tenesmus, accompanied by discharge of blood from the rectum, are complained of. In some instances the growth itself is extruded through the anus at defæcation, occasionally necessitating manual reduction.

*Physical Examination.* The passage of blood from the rectum in children should always lead the surgeon to suspect the presence of an

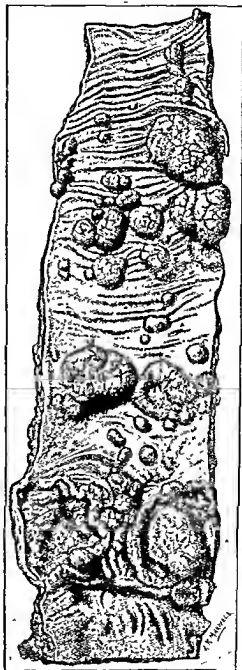
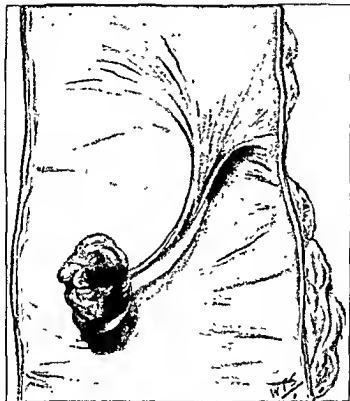


Fig. 673.—MULTIPLE ADENOMATA OF THE RECTUM, THREE OF WHICH (TWO IN THE LOWER AMPULLA AND ONE IN THE MIDDLE) ARE SIMULTANEOUSLY UNDERGOING CARCINOMATOUS CHANGE. SEVERAL OF THE TUMOURS ARE LOBULATED AND ARE ATTACHED BY A BROAD BASE SO AS TO RESEMBLE CLOSELY AN ADENOID CANCER.

(From a specimen in the Museum of the Cancer Hospital.)

adenomatous polypus. A careful exploration of the rectum should be made under the influence of anæsthesia, if necessary. A small growth is by no means easy to locate, as its tissue is of the same density as the mucous membranc. Moreover, a growth furnished with a long pedicle may be capable of such free movement that it may readily be mistaken for a small mass of feces (fig. 674). The best way of determining the presence of these growths is systematically to sweep the examining finger round



*Fig 674*—A PEDUNCULATED ADENOMA OF THE RECTUM. THE LONG PEDICLE PERMITS OF A CONSIDERABLE RANGE OF MOVEMENT SO THAT, WHEREAS AT TIMES THE GROWTH MAY BE WITHIN EASY REACH OF THE EXAMINING FINGER, AT OTHERS IT IS NOT. THE LIABILITY TO SHIFTING OF POSITION IS OF GREAT DIAGNOSTIC VALUE.

the whole circumference of the bowel as far as the highest point that can be reached. In this way a freely movable growth, after having been pushed in front of the finger, is at last arrested by its pedicle. The finger can then be hooked round the pedicle and the growth drawn down, and if possible brought out through the anus. The speculum is of little use for locating growths of small size. We have sometimes had much difficulty in finding a growth by this means, even after its presence had been previously determined by the finger. Examination with the proctoscope and sigmoidoscope should never be omitted.

*Treatment.* These growths should always be removed under the influence of anæsthesia. The sphincters should be stretched and the growth hooked down by the finger. The pedicle can then be clamped by a pair of pressure-forceps as close as possible to its point of attachment to the rectal wall, and a ligature applied on the proximal side of the forceps. The pedicle is then severed on the distal side of the forceps and the latter removed. The ligature, when tightly tied, separates on the fourth or fifth day.

*After-treatment.* The bowels should be confined for three or four days and then opened by means of olive-oil injections. In the case of adults at or over middle life, the seat of attachment of the growth should be examined from time to time for signs of induration. In one of my cases a typical carcinomatous ulcer developed in the vicinity of the scar within six months after an adenomatous polypus had been removed. In this case the microscopical appearance of the growth did not, at the time, indicate carcinomatous degeneration, but the patient, a female, was fifty-seven years of age, and, therefore, at a time of life when carcinoma of the rectum often occurs.

(b) *The Fibroma or Fibrous Polypus.* This type of growth is composed almost entirely of fibrous tissue, and is usually attached by a short tough pedicle. The tumour has a complete investment of mucous membrane when it arises from the rectum or the upper part of the anal canal, but is covered with stratified epithelium when it springs from the lower part of the anal canal. It is almost invariably situated in the lower two inches of the rectum. Microscopical examination of the fibromata attached to the edges of the Morgagnian valves shows that they contain numerous nerve end-plates and sometimes a cartilage cell. I have recently seen a fibrous polypus, measuring three-quarters of an inch in diameter, which was attached to the rectal wall at a level of one and a half inches above the anal margin. The size usually varies from that of a split pea to a walnut, and is seldom larger when consisting of fibrous tissue alone. Occasionally single, there are usually two, three, or more growths of varying sizes in the same case. When multiple growths are present, some of them usually spring from the free edges of the valves of Morgagni, and are, therefore, attached to the wall of the anal canal, about a quarter of an inch above Hilton's white line.

The fibromata met with in the rectum have three sources of origin, viz. (1) localised hypertrophy of the fibrous elements in the submucous



tissue; (2) from an internal hæmorrhoid, the dilated veins of which have become thrombosed and have subsequently undergone fibrous transformation (the fibrosed pile); and (3) hypertrophy of the papillæ or nodules normally met with on the free edges or surfaces of the valves of Morgagni. The growths, though at first sessile, soon become pedunculated by reason of the traction exerted upon them during defæcation. Consequently they are frequently protruded at stool and are generally associated with internal piles and a well-developed pecten band.

*Symptoms.* A small fibroma of the rectum may exist for a considerable time without giving rise to symptoms indicative of its presence. This is especially the case with those that arise in connection with the papillæ on the valves of Morgagni. Their presence, however, ultimately induces an unnatural spasmodic action of the external sphincter muscle, and is one of the chief factors in the causation of the pecten band. The difficulty in defæcation thus engendered causes the patient to seek a rectal examination, when the presence of one or more of these small fibromata may be disclosed. Larger growths are sooner or later protruded at each action of the bowels, and, if associated with internal piles in the third stage of their development, may be constantly protruded. It frequently happens that the pedicle of one of these fibromata is partially torn during the passage of a hard mass of fæces. This causes burning pain and a small loss of blood. The pain sometimes persists for several hours after the action of the bowels, and is suggestive of fissure. In such cases, if on examination there is no sign of a fissure, the finger should be gently passed into the anal canal for the purpose of ascertaining whether a fibrous polypus be present. If so, the possibility of a laceration of its pedicle should be suspected. The detection of such laceration is important because, in addition to the pain which it causes, a blind internal submucous fistula may arise therefrom.

*Physical Examination.* The presence of a fibroma in the rectum is, as a rule, readily detected by introducing the finger into the anal canal and sweeping it slowly round its entire circumference. The growths are firm to the touch and can be sometimes readily drawn down and exposed to view. In those cases in which a laceration of the pedicle exists, there is usually so much spasm of the sphincter muscles and levatores ani that it is difficult to conduct the examination satisfactorily without an anæsthetic.

*Treatment.* As soon as a fibrous polypus of the rectum is known to exist the patient should be urged to have it removed without delay,

because of the possibility of a laceration of the pedicle occurring and a blind internal fistula resulting therefrom. The growth is easily removed by applying a ligature to its base. When the polypus is semi-sessile it is best dealt with like an internal pile. When a laceration of the pedicle is present, the pecten band should be completely divided in the right posterior quadrant, and after that has been done the growth should be removed.

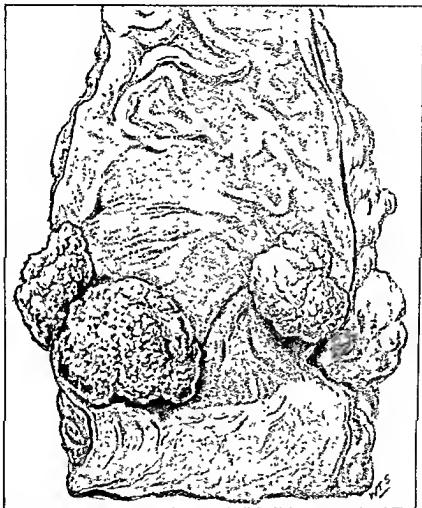


Fig. 675.—A VILLOUS PAPILLOMA OF THE RECTUM. THE TUMOUR IN THIS CASE CONSISTED OF THREE LOBES, BUT THE GROWTH WAS DIVIDED WHEN THE RECTUM WAS OPENED UP. THE TUMOUR IS ATTACHED TO THE WALL OF THE RECTUM BY A BROAD PEDICLE CONSISTING OF A FOLD OF MUCOUS MEMBRANE CONTAINING BLOOD-VESSELS OF CONSIDERABLE SIZE. THE TUMOUR IS SOFT AND VELVET.

(From a specimen in the Museum of the Cancer Hospital.)

(c) *The Villous Tumour.* The villous tumour is one of the rarest of all the growths met with in the rectum. This statement is warranted by the fact that but a small number of cases have been recorded in the literature of the subject. These growths when seen by the surgeon

vary in size from the dimensions of a walnut to that of a Jaffa orange, and are usually semi-oval in shape. The surface is minutely lobulated, resembling very much the appearance of a cauliflower (fig. 675). In the smaller growths this lobulated appearance is not so well marked, the surface being covered by elongated papillae. An excellent example of this kind of tumour exists in the Museum of St. Bartholomew's Hospital, the growth closely resembling in outward appearance the villous tumour of the bladder.

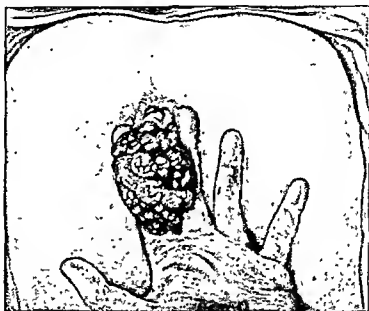


Fig. 676.—A VILLOUS TUMOUR OF THE RECTUM EXTRUDED THROUGH THE ANUS. IT WAS ATTACHED TO THE RECTAL WALL BY A BROAD PEDICLE CONSISTING OF A DOUBLE LAYER OF MUCOUS MEMBRANE CONTAINING BLOOD-VESSELS BETWEEN THEM. THE PEDICLE IS GRASPED BETWEEN THE INDEX AND MIDDLE FINGERS PLACED BEHIND THE TUMOUR.

The villous tumour appears to originate entirely from the mucous membrane of the bowel, the other coats not being involved, unless it is undergoing carcinomatous degeneration. When small the growth is as a rule sessile, but, as it increases in size, it becomes distinctly pedunculated. The pedicle in some cases is not well developed, while in others it is distinctly band-like, consisting merely of a double fold of mucous membrane extending either transversely or obliquely across one or other side of the bowel, usually obliquely. Such a pedicle is produced by the tumour being dragged away from the muscular wall of the rectum, either by virtue of its own weight or from its being pushed downwards during defæcation. When the pedicle is well developed the growth has the appearance of being slung to the rectal wall by a

mesentery, as it were, of mucous membrane. This pedicle is apt to become extremely lax in its attachment to the muscular coat, and in some cases will allow the growth to be protruded through the anal aperture. The accompanying illustration depicts such a protrusion from the anus (fig. 676). These tumours may be classed as benign, since they seldom show a tendency to recur after complete removal and do not infiltrate the rectal wall. At the same time it should be remembered that a carcinomatous change may take place in the growth, just in the same way as a simple adenoma of the breast may become malignant.

*Ætiology.* In the present state of our knowledge no light can be thrown upon the determining factor in the causation of this disease.

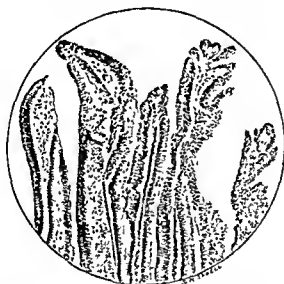


Fig 677.—ILLUSTRATING THE MICROSCOPICAL APPEARANCE OF THE HYPERTROPHIED VILLI OF A VILLOUS TUMOUR OF THE RECTUM. THE EPITHELIAL ELEMENTS SHOW NO IRREGULARITY IN GROWTH.

Pathologically the growth appears to consist of villous processes arising from the mucous membrane (fig. 677). Sex seems to have no influence, since males and females are affected with almost equal frequency. In both sexes the age at which the growth appears is beyond middle life.

*Symptoms.* The characteristic symptom of a typical villous growth in the rectum is the frequent escape of a thin watery fluid from the bowels, necessitating the patient going to stool many times during the day and night. This prominent symptom, for which the sufferer seeks relief, is described as continuing diarrhoea. It would be erroneous,

however, to suppose that all cases of villous growth present this single and characteristic symptom. There are others which make their appearance insidiously, their duration extending over many months, if not for several years. There are pains of a dull and aching character both in the rectum itself and over the sacrum, at first intermittent, but later becoming constant; hæmorrhage from the bowel, usually slight, but occasionally severe, especially when internal hæmorrhoids co-exist; loss of appetite; gradual and increasing diminution in weight; a general cachectic appearance, the skin assuming a pale and waxy colour; and, lastly, obstinate constipation, alternating with diarrhoea.

In the earlier stages of the disease, while the growth is yet small, none of the symptoms are well marked, beyond the escape of watery fluid, tinged or not with blood, but as it increases in bulk, the watery discharge to which it gives rise increases largely in quantity, and obliges the patient to evacuate the bowel many times during a period of twenty-four hours. This discharge is usually described as consisting of mucus, but such is not the case. The fluid is pale and watery in appearance, and contains only a few shreds of mucus. It is the rapid secretion of this watery fluid which occasions such frequent action of the bowels, and close inquiry will elicit from the patient that a natural fecal evacuation takes place only once or, at most, twice in the course of twenty-four hours.

Bleeding from the rectum occurs with sufficient frequency in cases of villous tumour to warrant its inclusion as a symptom, but it is very questionable whether the blood emanates entirely from the growth itself. So long as the growth remains inside the rectum, very little bleeding from its surface is noticed, and it is rare to find the exploring finger streaked with blood, as is the case in most instances of carcinoma. Should the growth, however, become prolapsed, bleeding readily takes place from its surface, but this ceases almost directly when it has been returned into the rectum. It is, of course, possible that abrasions of the surface of the growth, produced either by local examination or by the passage of hardened feces over it, may cause some hæmorrhage. The point to be emphasised is that villous tumours rarely bleed unless protruded, and that when bleeding exists, its cause must be looked for elsewhere. So far as my observations go, several of the patients suffering from villous growth had internal hæmorrhoids, which evidently were the source of the hæmorrhage. In one of the cases the patient, who was aged fifty-seven, had three of the largest piles that I had ever seen.

The pain complained of is readily explained by the constant dragging upon the rectal wall by the traction of the growth, and is referred to the rectum itself and through the sacral nerves to the skin over the sacrum. It is a noteworthy fact that pain is felt in the growth itself, so long as it is protruded through the anus, but ceases as soon as the tumour has been returned into the rectum.

The loss of flesh and general cachectic appearance is undoubtedly due to the constant discharge and accompanying mental anxiety.

It is rare to meet with complete intestinal obstruction due to a growth of this kind, as the patient usually seeks relief long before it has attained sufficient size to occlude the lumen of the bowel.

*Pathological Anatomy.* On inspection, the anus is generally found to present a sodden appearance, and there is an absence of folds of redundant skin unless well-developed internal piles co-exist. The anal orifice is somewhat loosely closed by the sphincters, marked relaxation being present only when there is a history that the growth protrudes at times from the anus.

When the growth is protruded at the time of the examination it will be found to be attached by a distinct pedicle which passes into the bowel, the pedicle being so much smaller in circumference than the growth itself that the fingers can be placed behind the growth, as is shown in figure 676. When there is no protrusion a digital exploration of the rectum should be next proceeded with. When there has been well marked hæmorrhage the right anterior pile, and perhaps others, is usually present. On introducing the finger to its full length and sweeping it round the interior of the rectal cavity a soft, slippery growth will be found occupying the rectum, and feeling very much like a redundancy of healthy mucous membrane. Careful manipulation will now show that the growth is attached to some part of the circumference of the bowel by a broad fold of mucous membrane, which generally extends obliquely or transversely to its longitudinal axis, unless the growth involves the anal canal, in which case it has not a well-marked pedicle.

In order that a more exhaustive examination may be made, it will be necessary to place the patient under the influence of an anæsthetic. When this has been done the sphincters can, if necessary, be thoroughly dilated, and the growth brought down through the anal orifice, unless the pedicle is too short or the growth situated too high up to allow of this. In this manner the exact size of the tumour can be ascertained, and an idea formed as to the measures to be adopted for its removal.

When the growth is seen, its colour is of a brighter red than the surrounding mucous membrane. There is no induration of its seat of attachment. The growth is as a rule single, though I have notes of one case in which there were two distinct growths. If the tumour is undergoing carcinomatous change, there will be alteration in its consistency, with, perhaps, ulceration of its surface, induration of its base, and a marked tendency to bleed freely when touched (fig. 678).

*Treatment.* Palliative measures are of no avail in the treatment of this kind of growth and should not be resorted to. As soon as its presence has been detected, early removal by operation should be



Fig. 678.—THE MICROSCOPICAL APPEARANCE OF A VILLOUS PAPILLOMA UNDERGOING CARCINOMATOUS CHANGE. THE HYPERTROPHIED VILLI APPEAR TO BE NORMAL FOR THE MOST PART BUT AT THE BASE THE EPITHELIAL ELEMENTS ARE PROLIFERATING IRREGULARLY AND ARE PENETRATING THE MUSCULARIS MUCOSÆ.

advised, firstly, because the free, watery excretion from the growth gives rise to frequent actions of the bowels, and the constitutional depression consequent thereon; secondly, because of the possibility of either invagination or procidentia of the rectum being produced by the downward traction of the growth; and lastly, because the growth may ultimately undergo carcinomatous degeneration and so infiltrate the muscular coat of the bowel or later on the peri-rectal structures (fig. 679). It has been pointed out that nearly all the primary villous growths of the rectum are furnished with a distinct pedicle and are therefore amenable to being drawn down and made to protrude from the anal orifice. In some cases, however, the pedicle is attached so high up as to render

this impracticable. When so situated removal of the growth from within the rectum is a matter of extreme difficulty, and it is better either to perform left inguinal colostomy and subsequently remove the rectum itself or in suitable cases to resect the portion of the bowel containing the growth and perform end-to-end anastomosis.

*Method of Operating.* The sphincters should, if necessary, be thoroughly stretched. The growth is then drawn down by the fingers and made to protrude through the anal orifice. A suitable clamp is then placed on the pedicle about half an inch from the margin of the tumour. The pedicle is then transfixed, on the proximal side of the clamp, in one or more places according to its breadth and ligatured in sections, as described on page 1313 for dealing with a fold of prolapsed mucous membrane. The pedicle is then divided on the distal side of the clamp. The ligatures are cut short, the clamp removed, and the stump allowed to retract into the rectum.

*After-treatment.* The bowels should be kept confined for four days and then an injection of 1 oz. of olive oil night and morning should be employed to make them act. Until the bowels have commenced to act freely only a light diet should be allowed.

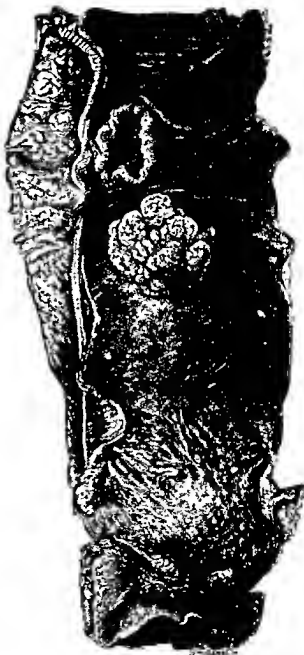


Fig. 679.—A BENIGN PAPILLOMA OF THE UPPER AMPULLA ASSOCIATED WITH A TYPICAL ADENOID CARCINOMA IN THE TERMINAL PELVIC COLON. THE PRIMARY SYMPTOM IN THIS CASE WAS A PROFUSE HÆMORRHAGE FROM THE RECTUM SO THAT IT IS PROBABLE THAT A SECOND PAPILLOMA EXISTED WHICH UNDERWENT CARCINOMATOUS DEGENERATION AND, BECOMING DETACHED, GAVE RISE TO THE BLEEDING.

(From a specimen in the Museum of the Cancer Hospital.)



*Recurrence of the Growth After Removal.* A recurrence of this kind of tumour occurs with sufficient frequency to render it advisable for the patient to present himself for examination at least once in every six months after the operation. The recurrent growth generally commences at the margin of the scar of the previous operation, which is invariably adherent to the muscular coat of the bowel. The recurrent growth is, therefore, nearly always sessile. If it be undergoing carcinomatous degeneration, infiltration of the muscular coat also occurs, and this may be suspected to have taken place if there be much attendant induration of the base of the tumour.

*Treatment of the Recurrent Growth.* As there is practically no pedicle, the growth cannot be completely removed without excising part of the muscular coat of the bowel as well. Accordingly the case should be treated as one of carcinoma pure and simple, and excision of the rectum should be performed by the abdomino-perineal method if the general condition of the patient is sufficiently good or by the perineal method after preliminary colostomy.

(d) *The Myxoma or Myxomatous Polypus.* The pure myxoma is very rarely met with in the rectum. Gant mentions one case in which the diagnosis of the pathological nature of the growth was verified by microscopical examination.

Growths consisting of a combination of mucoid and fibrous tissue (fibro-myxoma), however, are occasionally met with and may attain large size. There is an excellent specimen of this kind of tumour in the Museum of St. Bartholomew's Hospital. It was removed from the rectum of a woman aged twenty-four years, and weighed, when fresh, nearly two pounds.

In structure the fibro-myxoma consists of bundles of loose gelatinous connective tissue containing spaces which are filled with fluid. The surface of the tumour is smooth or slightly lobulated. It grows slowly, and may exist for a considerable time before giving rise to symptoms referable to its presence.

*Symptoms.* The growth gradually increases in size until it may almost completely fill the cavity of the rectum. Consequently the first symptom noticed is a steadily increasing difficulty in obtaining an action of the bowels and a sense of incomplete relief after defæcation. If the growth is attached near the anal orifice, protrusion during straining at stool may occur. The protruded mass, when of large size,

may be so firmly gripped by the sphincters that reduction becomes impossible.

*Treatment.* This consists in removing the tumour by transfixion of the pedicle and ligaturing it in two, three or more portions according to its breadth. There is no tendency to recurrence after removal.

## CHAPTER VIII

### MALIGNANT GROWTHS OF THE ANUS AND RECTUM

BOTH varieties of malignant growth, viz., the carcinoma and the sarcoma, are met with in the ano-rectal region. The former are so frequently seen that, according to statistics, they form rather more than one-third of the carcinomatous growths of the whole of the alimentary canal, including mouth, tongue, and fauces. The latter are extremely seldom met with.

#### CARCINOMA OF THE ANUS

This may occur either as a primary growth of the anal skin, in which case it is always a squamous carcinoma (epithelioma), or as a secondary invasion from a growth starting low down in the rectum, and then it presents the histological characters of an adeno-carcinoma. As the latter variety is but the advanced stage of a carcinomatous growth of the rectum it will be dealt with under that head.

The squamous carcinoma of the anus is seldom seen, and is nearly always met with in patients who have attained to the sixth or later decades of life. It is usually seen when the stage of ulceration has already been reached, and then it presents the characters of a typical squamous-cell carcinomatous ulcer, as met with in other parts of the body.

The histological characters of the growth are characteristic. The symptoms are those of an ulcer which steadily extends, without healing in any part, and bleeds more or less freely when touched. The inguinal glands on the corresponding side soon become enlarged.

*Treatment.* The ulcer cannot be too soon or too freely removed. The anal margin on the affected side should be completely removed, and no attempt made to preserve the corresponding half of the external and internal sphincters. Complete removal of one half of the anal margin, as advised, does not cause stenosis of the anal orifice. Should

the inguinal lymphatic glands he implicated, or subsequently become so, they should be completely removed. There have been a few cases successfully treated by radium, but on the whole more satisfactory results are obtained by surgical operation.

### CARCINOMA OF THE RECTUM

*Carcinoma* is the most grave of the diseases of the rectum. As in carcinoma of other organs, early recognition of the disease is of paramount importance. The laws which govern the onset of carcinoma are as yet unknown, and, for the present, early and complete removal of the growth holds out the only prospect of cure.

### CLASSIFICATION

The adeno-carcinoma is the only type of cancer that is met with in the rectum, the epithelial elements being derived from the cylindrical cells of Lieberkühn's follicles (fig. 680).

Although there is only one type of carcinoma of the rectum, four distinct clinical varieties can be recognised, differing in physical characteristics and in degree of malignancy. These are: (a) the papilliferous carcinoma; (b) the common adenoid carcinoma; (c) the colloid carcinoma; and (d) the melanotic carcinoma.



Fig. 680.—DRAWING MADE FROM A MICROSCOPICAL SECTION TAKEN FROM THE MUCOSA CLOSE TO THE APPARENT GROWING EDGE OF A RECTAL CARCINOMA AND SHOWING THE EARLY HISTOLOGICAL CHANGES THAT CHARACTERISE MALIGNANCY. THE DILATED LIEBERKÜHN FOLLICLE IS LINED FOR FIVE SIXTHS OF THE CIRCUMFERENCE BY A SINGLE LAYER OF WELL-FORMED COLUMNAR CELLS WHICH APPEAR TO BE NORMAL IN ALL RESPECTS. FROM THE REMAINING SIXTH OF THE CIRCUMFERENCE THERE IS AN INGROWTH OF IRREGULAR COLUMNAR EPITHELIUM SEVERAL CELLS IN THICKNESS.

(From the Pathological Department of the Cancer Hospital.)

*The Papilliferous Carcinoma.*  
The growth resembles an ordinary simple papilloma, but at the base of the tumour the epithelial elements proliferate irregularly and penetrate the muscularis mucosæ. Such a growth extends rapidly upon the surface and soon involves the whole circumference of the bowel

(fig. 681). Owing to the exuberance of the growth, the lumen of the bowel becomes obstructed long before infiltration of the

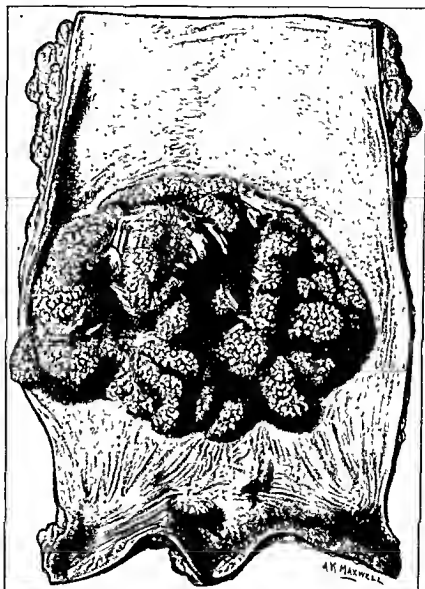


Fig. 681.—A PAPILLIFEROUS CARCINOMA INVOLVING THREE QUARTERS OF THE CIRCUMFERENCE OF THE RECTAL AMPULLA. THE SURFACE OF THE GROWTH IS NOT ULCERATED SO THAT THE PAPILLIFORM APPEARANCE IS WELL SHOWN. THE MUSCULAR COAT IS NOT DEEPLY INFILTRATED, AND THERE WERE NO EXTRA-MURAL METASTASES

(From a specimen in the Museum of the Cancer Hospital.)

muscular coat has progressed to any marked degree. Consequently these growths are not particularly malignant and seldom give rise to extra-mural metastases unless they have been in existence for a considerable time. When surface necrosis has occurred the retro-rectal lymph

glands may become enlarged from septic absorption, but rarely are they the seat of cancerous deposit. Hence the papilliferous carcinoma seldom recurs after removal, even by an operation of a most restricted type. As a rule, symptoms of obstruction, due to blocking of the lumen of the bowel by the exuberant growth, lead to early detection before extra-mural dissemination has had time to develop.

*The Adenoid Carcinoma.* The great majority of cancers of the rectum are of this kind. They are usually seen as sessile tumours

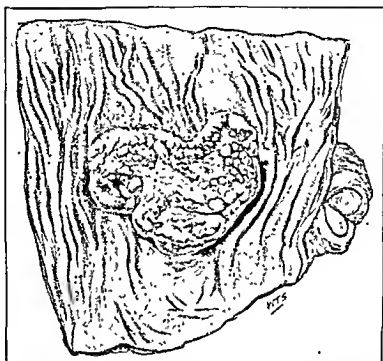


Fig. 662.—AN ADENOID CARCINOMA OF THE AMPULLA IN WHICH SURFACE DIS-INTEGRATION HAS RECENTLY COMMENCED. THE GROWTH EXTENDS OVER ONE-THIRD OF THE CIRCUMFERENCE OF THE BOWEL AND IS FREELY MOVABLE OVER THE SUBJACENT MUSCULAR COAT. THE INFILTRATION DOES NOT EXTEND BEYOND THE SUBMUCOUS TISSUE. A RETRO-RECTAL GLAND IS HEAVILY INVADIED, DESPITE THE FACT THAT THE MUSCULAR COAT HAS NOT YET BEEN IMPLICATED.

(From a specimen in the Museum of the Cancer Hospital)

involving the mucosa and submucosa. In the majority of cases the growth is flattened, the transverse diameter being greater than the longitudinal. The tumour increases in size in all directions, and, though at first freely movable upon the muscular coat, soon infiltrates and becomes adherent to it. It is impossible to say how soon after the inception of the neoplasm deep infiltration takes place, but probably it occurs within six months. As the growth increases in size, surface disintegration occurs and a definite ulcer, exhibiting the well-known

malignant characters, is formed. Even at this early stage the retro-rectal lymph glands are usually invaded, thus showing that extra-mural dissemination of cancer cells takes place while the growth is still in a clinically early stage of development (fig. 682). As more of the circumference of the bowel becomes involved, the ulcer deepens and assumes a crateriform appearance with nodular, everted, and indurated edges. When the ulcer has extended nearly all round the bowel, stenosis of the lumen is produced and then the stage is reached when symptoms of impending obstruction make themselves manifest. The adenoid cancer gives rise to extra-mural metastases which may be widespread while the growth is yet in an early stage clinically, and therefore must always be considered as highly malignant (fig. 682). It will inevitably recur after a restricted operation.

*The Colloid or Muroid Carcinoma.* This is merely a degenerative stage of the preceding varieties, both the epithelial elements and the connective tissue undergoing mucinoid change. Such growths are extremely malignant and are apt to recur rapidly even after the most comprehensive operation (fig. 683).

*The Melanotic Carcinoma.* This variety of rectal cancer is fortunately extremely rare, and is the most malignant of all. I have met with only three examples of it among nearly five hundred cases of cancer of the rectum which I have operated upon. The growth is found either in the anal canal or at the lowermost part of the ampulla and generally upon the posterior wall. Macroscopically it differs little in general appearance from the ordinary adenoid cancer, but microscopically the presence of pigmentation in the epithelial and connective tissue elements reveals the true nature of the growth. The pigmentation, however, may be absent from portions of the growth, so that unless serial sections of the tumour are made, the melanotic nature of the growth may escape detection. Rapid dissemination takes place giving rise to metastases throughout the body. The growth invariably recurs after removal.

Dukes classifies rectal carcinomata according to the depth of the local infiltration, that is, spread through continuity of tissue. He divides them into three groups, viz. : A, B and C, thus :

“ A cases are those in which the carcinoma is limited to the wall of the rectum, there being no extension into the extra-rectal tissues and no metastases in the lymph nodes.

"B cases are those in which the carcinoma has spread by direct continuity to the extra-rectal tissues, but has not yet invaded the regional lymph nodes.

"C cases are those in which metastases are present in the regional lymph nodes."



Fig. 683.—COLLOID CARCINOMA OF THE RECTUM. THE WHOLE OF THE MUCOSA OF THE LOWER TWO THIRDS OF THE RECTUM IS INVOLVED BY THE DISEASE. BOTH THE SUBMUCOUS AND THE MUSCULAR COATS PARTICIPATE IN THE MUCINOID CHANGE. THE RETRO-RECTAL GLANDS AND THE PERI-RECTAL TISSUES ARE THE SEAT OF NUMEROUS DEPOSITS.

(From a specimen in the Museum of the Cancer Hospital.)

The above classification is useful from the point of view of post-operative prognosis in regard to the likelihood of recurrence, but it is not of any value for indicating the type of operation suitable for a given case, because the group to which the growth belongs can only be determined by an examination of the specimen after the operation has been performed. It would not be very comforting to ascertain that a restricted type of operation had been performed for a growth that was subsequently proved to belong to Group C.



## ÆTIOLOGY

The cause of cancer of the rectum, as of cancer in other organs, is still unknown. The determining factor causing the epithelial elements of part of the circumference of a Lieberkühn's follicle (as shown in fig. 680) to undergo irregular proliferation remains obscure. We have evidence that benign adenomata undergo malignant degeneration, and when existing in large numbers two or three of them may become carcinomatous simultaneously, but it is extremely doubtful whether every carcinoma of the rectum is preceded by an adenoma. As the result, too, of chronic irritation it is possible that a carcinoma may arise in the stoma of a diverticulum of the terminal pelvic colon and subsequently extend to the rectum. The accompanying illustration (fig. 684) shows a carcinoma of the rectum associated with multiple adenomata of the ampulla and several diverticula of the terminal pelvic colon.

## SYMPTOMATOLOGY

It is of the utmost importance that the earliest symptoms indicative of the presence of a carcinomatous growth in the rectum should be known and recognised. The disease is insidious in its inception and early progress, and may exist for six months or more before giving rise to symptoms which are sufficiently pronounced to induce a patient to seek relief.

There is a distinct and considerable latent period during which there are no objective symptoms at all, the growth being in a pre-ulcerative stage. It is impossible to determine the duration of this latent period, but doubtless it extends over a period of several months, possibly six months or even a year. It is obvious, therefore, that if some reliable sign, indicative of the presence of carcinoma during the latent period of its evolution, could be recognised, a much larger proportion of growths in an operable stage would be discovered.

Careful enquiry into the antecedent history of patients found to be suffering from carcinoma of the rectum very often reveals the fact that, a year or so prior to the first appearance of objective symptoms, functional inertia of the colon existed, expressed by a prolonged and well-marked attack of constipation.

After a definite interval, varying from three to six months, the constipation is followed by diarrhoea which, though slight at first, gradually becomes more pronounced. The constipation resulting from

the inertia of the colon does not readily yield to treatment. Aperients appear to have little effect beyond producing small ineffective motions and the voiding of flatus. The inertia of the colon is not associated with the abdominal distension, which is always present during the later phases of the disease when the growth obstructs the lumen of the bowel.

The actual cause of the inertia is doubtful, but it is possible that it may be due to interception of the peristaltic wave by the growth. Definite proof of this is unfortunately wanting because patients do not present themselves for rectal examination during the latent period.

If a carcinomatous growth is capable of causing inertia of the muscular wall of the bowel in this way, it is possible that an attack of persistent constipation may be the earliest sign of the presence of the disease. At any rate, the history of an attack of intractable constipation



Fig. 684.—ADENOID CARCINOMA OF THE UPPER AMPULLA ASSOCIATED WITH DIVERTICULOSIS OF THE TERMINAL PELVIC COLON. THE STOMATA OF THE DIVERTICULA ARE INDICATED BY BRISTLES. IN THE LOWER AMPULLA THERE ARE SIX SMALL SESSILE ADENOMATA AND THERE IS A SMALL ONE ABOVE THE GROWTH. (From a specimen in the Museum of the Cancer Hospital.)

during the latent period preceding the onset of objective symptoms is so often obtainable that I attach much importance to it and strongly urge that a thorough rectal examination be made in every instance in which frequency of the action of the bowel is found to have followed a prolonged attack of constipation in an otherwise apparently healthy subject, since the possibility of carcinoma being the cause is very great and should not be lost sight of.

Sooner or later objective symptoms make their appearance. No particular train of symptoms can be ascribed to all cancerous growths of the rectum, because they vary considerably with the position of the growth. Accordingly, when dealing with the symptomatology of cancer of the rectum, it is necessary that the symptoms of growths occurring (a) at the recto-sigmoidal junction, (b) in the anal canal, and (c) in the ampulla, should be considered separately.

(a) *Symptoms of a Cancerous Growth at the Recto-Sigmoidal Junction.* Growths in this situation conform to the type usually met with in the colon in that they rapidly involve the entire circumference of the bowel, and produce obvious stenosis. Both the papilliferous and the adenoid varieties occur in this locality, and are attended by different trains of symptoms. A papilliferous growth gives rise to excessive mucous secretion and causes diarrhoea which, though slight at first, exhibits a tendency to become more marked. During the early stages the diarrhoea occurs only after meals, and particularly after the drinking of warm fluids such as tea or coffee; but, later on, as the stenosis becomes more pronounced, it increases in frequency and may recur at short intervals. The stools are frequently tinged with blood, though the bleeding is seldom copious unless a necrotic area on the surface of the growth becomes detached. After a variable interval the diarrhoea ceases and an attack of obstruction supervenes either from intussusception of the stenosed segment of bowel or from impaction of a mass of faeces above the stenosis.

The adenoid cancer, on the other hand, rapidly infiltrates the entire circumference of the bowel and causes such rigid stenosis that the first indication of its presence is an attack of absolute obstruction. Abdominal distension, associated with visible peristalsis and intermittent attacks of colicky pain, denotes the completeness of the blockage. An excellent example of this kind of growth is shown in figure 685.

The entire circumference of the bowel is involved and the lumen is greatly stenosed. The surface of the growth though irregular in outline is not eroded. The muscular coat has not been completely penetrated,

but a large metastatic deposit exists in the lower part of the pelvic mesocolon and the retro-rectal glands are extensively invaded.

Carcinomata situated at the recto-sigmoidal junction are usually operable because they come under observation at an early date on account of the urgency of the symptoms produced by stenosis.

*(b) Symptoms of Carcinoma arising in the Anal Canal.*

Cancers situated in the anal canal, even when quite small, give rise to great discomfort at an early date (fig. 687). Especially after an action of the bowels there is a sensation of incomplete relief. Being constantly in the grip of the sphincters the surface of the growth soon ulcerates, and thus both pain and bleeding are early indications of its presence. Pain is experienced whenever there is an action of the bowels and persists for some length of time afterwards. The painful phenomena closely simulate those produced by an ordinary fissure of the anus and may be mistaken for them. Bleeding though usually slight

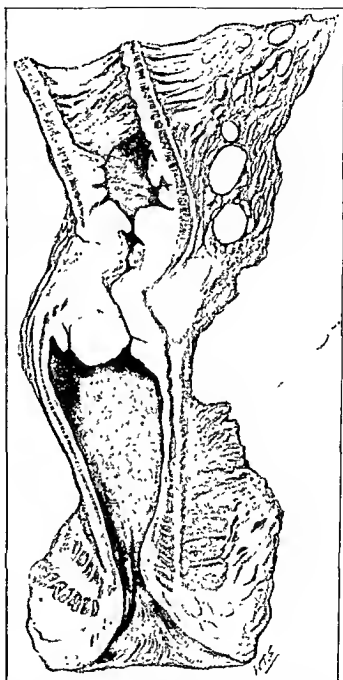


Fig. 685.—AN ADENOID CARCINOMA SITUATED AT THE RECTO SIGMOIDAL JUNCTION. THE GROWTH EXTENDS COMPLETELY ROUND THE BOWEL, PRODUCING MARKED STENOSIS. THE SURFACE OF THE GROWTH IS NOT ERODED. THE LOWERMOST PART OF THE PELVIC MESOCOLON IS INVADIED BY DIRECT EXTENSION THROUGH THE MUSCULAR COAT, AND THE RETRO RECTAL GLANDS ARE EXTENSIVELY INVADIED.

(From a specimen in the Museum of the Cancer Hospital.)

may sometimes be profuse if laceration of the surface of the growth occurs.

Anal canal cancers give rise to definite objective symptoms while they are still in an early stage, and their presence is usually detected before they have passed beyond an operable stage. It often happens, however, that the symptoms are mistaken for those of internal piles or fissure, and consequently these cases are often allowed to drift until perforation of the bowel wall has resulted in extensive ischio-rectal suppuration. As the result of compression by the spasmodic contraction of the sphincters deep excavation occurs early (fig. 687).

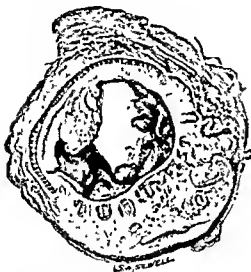


FIG. 686.—A TRANSVERSE SECTION OF AN ADENOID CARCINOMA AT THE RECTO-SIGMOIDAL JUNCTION. THE GROWTH HAS COMPLETELY ENCIRCLED THE BOWEL BY EXTENDING IN THE SUBMUCOUS TISSUE, AND IN HALF OF THE CIRCUMFERENCE HAS EXTENDED THROUGH THE MUSCULAR COAT INTO THE SURROUNDING TISSUE.

(From a specimen in the Museum of the Cancer Hospital.)

(c) *Symptoms of Carcinoma of the Ampulla.* Owing to the facts that the lumen of the ampulla is of considerable magnitude, and that the mucosa is not particularly sensitive, a growth may exist for several months without affording any indication of its presence. After a time surface disintegration begins and then blood is noticed in the stools. Thus the earliest objective sign of the existence of an ampullary growth is streaking of the motions by blood. As the disease progresses other symptoms develop, so that it is

convenient to describe the symptomatology of ampullary cancers as they occur during the successive stages of their development.

*Symptoms During the Pre-ulcerative Stage.* So long as the growth remains in the pre-ulcerative stage, objective signs are absent and patients have no reason for seeking advice. A papilliferous tumour, owing to the fact that the exuberant tissue soon fills the lumen of the bowel, may excite attention by giving rise to a sensation of fullness in the rectum and an impression that the bowel had been incompletely emptied after defæcation. Under these circumstances a growth of this kind may be detected before any objective sign, such as a copious

mucus discharge, has made its appearance. The adenoid cancer, however, may exist for several months without creating any disturbance beyond causing a prolonged attack of constipation which, as I have already pointed out, is probably due to inertia of the muscular wall of the howel.

Towards the end of the pre-ulcerative stage, frequency in the action of the howels appears and tends to become more marked as time goes

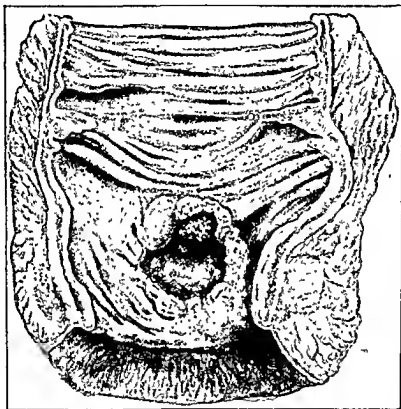


Fig. 687.—AN ADENOID CARCINOMA OF THE ANAL CANAL. THE GROWTH IS SITUATED UPON THE ANTERIOR WALL. ALTHOUGH IT INVOLVES LESS THAN A THIRD OF THE CIRCUMFERENCE, THERE IS DEEP EXCAVATION AND THE MUSCULAR COAT HAS BEEN COMPLETELY PENETRATED. THE DEEP EXCAVATION IS THE RESULT OF PRESSURE NECROSIS INDUCED BY PROLONGED SPASMODIC CONTRACTION OF THE SPHINCTER.

(From a specimen in the Museum of the Cancer Hospital.)

on. At first the consistency of the dejecta is semi-solid, the actions of the howels taking place after meals only. Eventually the stools become fluid in character and the actions of the howels are more frequent. Nocturnal movements seldom occur during this stage.

As a rule, cancers in this early stage of development are seldom seen because the symptoms to which they give rise attract so little attention that, unless careful enquiry he made, the patient does not mention them (fig. 688). In some instances he may seek advice in the belief that he is suffering from diarrhoea. Pain is entirely absent, and there is nothing

in the character of the stools, beyond frequency, to cause uneasiness. Hence it is that a rectal examination is not sought until symptoms of a more definite character develop.

*Symptoms During the Progress of Surface Ulceration.* So soon as the surface of the growth becomes abraded, either from the detachment of exuberant masses or from the separation of necrotic portions, an ulcer results and symptoms of a more definite and objective character

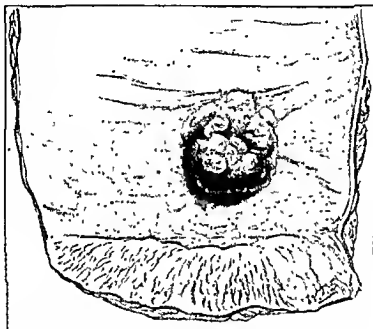


Fig. 689.—AN ADENOID CARCINOMA OF THE AMPULLA IN THE PRE-ULCERATIVE STAGE. THE TUMOUR IS ATTACHED BY A BROAD BASE AND IS FREELY MOVABLE UPON THE SUBJACENT MUSCULAR COAT. IT DID NOT GIVE RISE TO OBJECTIVE SYMPTOMS AND WAS DISCOVERED DURING A ROUTINE RECTAL EXAMINATION. THE RETRO-RECTAL GLANDS WERE INVADDED.

(From a specimen in the Museum of the Cancer Hospital.)

appear. As a direct result of the presence of an ulcerating surface the rectum becomes irritable and intolerant of contact with faeces (fig. 689). The bowels act frequently, a dozen or more movements occurring during twenty-four hours. The majority of the stools consist of mucus tinged with blood. The movements of the bowels take place during the day and very seldom at night. The appearance of blood in the stools becomes more marked as the ulcerating surface extends, but bleeding is seldom profuse unless portions of the growth become detached.

When repeated attacks of free bleeding occur during this stage of the disease they are generally the result of co-existent internal piles.

The presence of an excessive quantity of mucus in the stools is characteristic of the ulcerative stage, and is probably due to proctitis in the vicinity of the ulcerating surface. The mucus collecting in the rectum gives rise to frequent desire to evacuate. From being mixed with blood and disintegrating portions of the growth, the mucus possesses a peculiarly offensive odour which is characteristic of cancer of the rectum.

It is surprising how little pain accompanies an ulcerated cancer of the ampulla during the earlier stages. When the primary growth has penetrated the wall of the rectum, local suppuration, either beneath the mucosa or in the peri-rectal fatty tissue, may occur and then pain, occasionally of a severe character, is experienced, and becomes pronounced during a movement of the bowels. In those instances in which a growth situated in the lower part of the ampulla encroaches upon the anal canal, severe pain is felt during defæcation and persists for several hours afterwards on account of spasm of the sphincters and

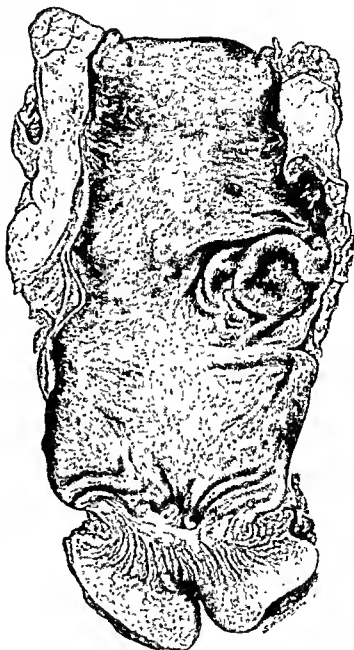


Fig. 689.—AN ADENOID CARCINOMA OF THE MIDDLE OF THE AMPULLA, LESS THAN AN INCH IN DIAMETER AND DEEPLY EXCAVATED. THE MUSCULAR COAT WAS COMPLETELY PENETRATED. THE RETRO RECTAL GLANDS WERE HEAVILY INVADDED.

(From a specimen in the Museum of the Cancer Hospital.)



levator ani muscles. When the growth is confined to the upper part of the ampulla, pain is usually absent, though a dull aching sensation in the region of the sacrum may be complained of.

During this stage of the disease, also, progressive loss of weight is noticeable. When there is excessive bleeding, and especially when suppuration is profuse, emaciation ensues rapidly.

*Symptoms when the Rectal Wall has been Penetrated and Infiltration of the Peri-rectal Tissues has Occurred.* When this stage has been reached the growth has involved more than three-quarters of the circumference of the ampulla. The surface is deeply excavated and the margins of the ulcer are irregular, nodular, and everted. By this time, also, extra-mural metastases are invariably found scattered along the course of the superior hæmorrhoidal vessels and probably along the inferior mesenteric vessels as well. The veins are especially apt to become compressed by the metastatic deposits, with the result that impediment to venous return causes passive congestion of the growth. Bleeding occurs readily both during digital examination and when the bowels act. Large quantities of blood may be lost suddenly, especially when a large and dilated vein has been invaded by the growth.

Frequency of the actions of the bowels is pronounced, the stools consisting almost entirely of mucus, blood, and pus. In some cases the quantity of pus voided is excessive, indicating the presence of peri-rectal suppuration. With the advent of peri-rectal suppuration, deep-seated pelvic pain of a continuous and distressing character supervenes. Apart from this, pain is not a prominent symptom, and is generally limited to tenesmus following every movement of the bowels. The patient loses weight, and here, too, if hæmorrhage and suppuration be profuse, the loss may be extreme.

*Symptoms when the Growth has Extended beyond the Fascia Propria and has Implicated Neighbouring Structures.* This stage is reached about two years after the appearance of the earliest objective signs and when the growth has involved the greater part or even the whole of the circumference of the bowel. Owing partly to the extent of the circumferential involvement, and partly to fixation of the diseased portion of the rectum to neighbouring organs, or to peri-rectal suppuration, increasing difficulty in evacuating the contents of the bowel manifests itself. The narrowed lumen of the bowel becomes obstructed, by the impaction into it of a faecal mass, so that neither flatus nor liquid stools can be voided. The abdomen is markedly distended and

visible peristalsis in the course of the colon is plainly discernible. The obstructive attack, unless relieved by colostomy, usually lasts for three or four days or, perhaps, a week, and then terminates with a profuse liquid evacuation. Alternating attacks of constipation and diarrhoea then supervening herald the onset of ultimate absolute obstruction.

Pain during this stage of the disease is of two kinds: (1) abdominal, due to paroxysmal efforts of the colon to overcome the obstruction in the rectum; and (2) pelvic, on account of the extension of the growth into neighbouring structures, especially the sacral plexus of nerves. Involvement of the sacral plexus causes pain over the sacrum and down the posterior aspects of the thighs.

Intermittent attacks of bleeding, often profuse if a large vein is involved, become frequent and cause profound anaemia. Rapid emaciation ensues from sepsis, disorders of digestion, and want of sleep.

Invasion of neighbouring organs, such as the prostate, the urinary bladder, the vagina, or the uterus, adds symptoms referable to disease of those organs; and extensive peri-rectal suppuration eventually leads to the formation of ischio-rectal abscesses and fistulae, which materially increase the sufferings of the patient. Finally, the appearance of oedema of the lower extremities, ascites, and jaundice indicate the onset of general carcinomatosis.

I have set forth the symptomatology of rectal carcinomata somewhat fully to emphasise the fact that both the position occupied by the neoplasm and its variety exert considerable influence upon the train of symptoms presented in a given case. It appears to be a general impression that cancer of the rectum presents a definite train of symptoms consisting of the passage of blood and mucus through the anus and the existence of alternating attacks of constipation and diarrhoea. Except in the case of growths situated at the recto-sigmoidal junction, alternating attacks of constipation and diarrhoea supervene only when the disease has passed beyond the stage of operability, and, therefore, if a rectal examination in search of cancer be postponed until this particular sign has developed, a large number of operable growths will be missed.

*Differential Diagnosis.* The typical papilliferous tumour of the ampulla and the adenoid carcinoma, when ulceration of the surface is well marked, present such characteristic features that they cannot very well be mistaken for anything else.

The papilliferous growth of the rectum, however, may be simulated

by (a) a similar growth of the colon which has become intussuscepted into the rectum, or (b) a villous papilloma of the rectum.

(a) When a papilliferous growth of the colon is intussuscepted into the rectum it forms a large mass which almost completely fills the cavity of the ampulla. The tumour is not attached to the wall of the rectum but is suspended from above by a thick round stalk covered with normal mucous membrane. The sulcus between the sheath and the returning layer of the intussusception extends upwards for several inches. The lumen of the bowel is situated in the centre of the growth. An elongated tumour is present in the left iliac fossa. The abdomen is distended and there are signs of impending obstruction.

(b) The villous papilloma forms a soft exuberant tumour, the surface of which is minutely lobulated. The growth feels like a mass of redundant mucous membrane, so that it is difficult to make out its connections. It is usually attached to the wall of the rectum by a narrow band-like pedicle extending from one extremity of the growth to the other. In some instances the pedicle becomes so elongated that the tumour appears to be attached to the wall of the bowel by a mesentery. The most characteristic feature of this growth is that it excretes a watery fluid which collects in the rectum and necessitates frequent evacuation.

An adenoid carcinoma in the pre-ulcerative stage may be mistaken for a simple adenoma, but the firmness of its texture and the induration of its base should serve to distinguish it. The simple adenoma is soft, finely lobulated, and devoid of induration. It is usually single and attached to the wall of the rectum by a slender pedicle (see fig. 674). Sometimes these tumours are not pedunculated but are attached by a broad base, thus closely resembling an adenoid cancer. When sessile they generally exist in large numbers (see fig. 673), and are apt to undergo carcinomatous degeneration, the surface becoming converted into an ulcer with hard, sinuous, and everted edges. A benign neoplasm which has been and may easily be mistaken for cancer of the rectum in women is an endometrioma. The growth arises in the endometrium, and may invade the recto-vaginal septum, producing a definite tumour which encroaches upon the lumen of the rectum as well as that of the vagina. It may invade the muscular coat of the rectum and extend into the submucous tissue forming a tumour, varying in size from a walnut to a hen's egg, which partially obstructs the lumen of the bowel. When the infiltration has not extended

beyond the muscular coat, the mucosa is freely movable over it ; but when the submucosa has become implicated the mucous membrane is adherent.

Microscopically the growth displays the characteristic features of an endometrioma of the uterus. The neoplastic tissue, consisting of bundles of unstriped muscle enclosing tubular glands surrounded by endometrial stroma, extends through the whole thickness of the muscular walls of both vagina and rectum, into the submucosa or even the mucosa itself. The invading tissue appears to displace rather than to destroy that of the part invaded. Though infiltrating, the tumour is a benign and not a malignant neoplasm. Deep-seated pain in the pelvis and in the rectum aggravated both during and after menstruation, an evidence that endometriomatous deposits participate in the menstrual cycle and themselves menstruate, is characteristic of the disease. The hard indurated nature of the growth and the fact that it infiltrates the wall of the bowel creates a suspicion of malignancy, but this can be dispelled by removing a portion of the tumour for microscopical examination. At a meeting of the Proctological Sub-Section of the Royal Society of Medicine I showed a patient suffering from this kind of tumour. The growth which was originally as large as a hen's egg involved the left wall of the rectum and was adherent to the cervix uteri anteriorly and to the sacrum behind. As the result of removing the uterus and both ovaries the growth has practically disappeared and the patient has been cured of her pain.

#### EXAMINATION OF THE CANCEROUS RECTUM

In all suspected cases of cancer of the rectum a thorough examination should be made both digitally and by means of the proctoscope and sigmoidoscope.

*Digital Examination.* For this purpose the patient should be lying on his side. I prefer the right side to the left because, as the pelvic colon enters the pelvis on the left side, the recto-sigmoidal junction tends to fall downwards and towards the anus, whereas when the patient is on his left side the upper part of the rectum tends to recede towards the abdomen. The finger should be introduced to its full extent and then swept round the whole circumference of the bowel from above downwards. With a finger of ordinary length the promontory of the sacrum, a point well above the recto-sigmoidal junction,

can be readily felt, and therefore the whole of the rectum can be palpated.

When a cancerous growth is found to exist in the rectum it is generally situated (1) at the recto-sigmoidal junction; (2) in the ampulla; or (3) in the anal canal.

(1) A growth situated at the recto-sigmoidal junction usually extends completely round the circumference of the bowel and produces stenosis (see fig. 685). If the tip of the finger can be introduced into the lumen of the stenosis the diseased portion of the bowel will generally be found to be freely movable in all directions. When the tip of the finger cannot be introduced owing to the narrowness of the lumen some degree of intussusception of the diseased portion of the bowel will be found to exist. When the intussusception is marked, as indicated by the depth of the sulcus between the sheath and the returning layer of the intussusceptum, the mobility of the tumour is impaired. Under these circumstances the fixation of the tumour is more apparent than real, and is not necessarily due to the growth having involved neighbouring structures.

(2) When the growth is situated in the ampulla it sometimes happens that the entire cavity of the rectum is filled by the tumour. The growth may be attached to part of the wall of the bowel or it may involve the whole of the circumference, when the passage way into the cavity of the bowel above the tumour will be found to be situated in the centre of the growth. The bulk of the growth and the extent of its attachment may create the impression that the disease is of long standing and too advanced for anything but palliative treatment. These exuberant growths are always papilliferous, and, although they extend rapidly upon the mucous surface of the bowel, seldom infiltrate deeply.

More commonly the growth takes the form of an excavated ulcer with sinuous, everted and nodular edges. The ulcer may involve the bowel to any extent from a quarter to the whole of its circumference (see figs. 690 and 691). It is important in these cases to determine the exact extent of the circumferential involvement, because, as we shall see when discussing the spread of cancer, this may be taken both as a measure of the deep infiltration that has taken place, and as an index of the duration of the disease. The diseased portion of the bowel preserves its mobility as long as the fascia propria of the rectum has not been penetrated by the direct extension of the growth, which does not occur until more than three-quarters of the circumference of the ampulla has been encompassed.

When the ulcerated surface involves the whole of the circumference of the rectum the howel is firmly fixed to the adjacent structures which have been invaded and the lumen of the bowel is considerably narrowed.

(3) When the growth is situated in the anal canal it is generally found upon the anterior wall, and is limited in extent (see fig. 687). The surface of the ulcer is usually deeply excavated owing to surface necrosis induced by the constricting effect of the sphincter muscles and the levatores ani. As the result of surface sloughing, perforation of the wall of the howel may occur, even while the growth is of limited extent, and may give rise to ischio-rectal suppuration. The association, therefore, of hrawny induration in the perineum with a carcinomatous ulcer of the anal canal does not necessarily imply that extra-mural extension of the growth has taken place.

When cancer affects other parts of the anal canal it is usually due to an extension downwards of an ampullary growth. Under these circumstances although the growth in the anal canal may be of limited extent the ampulla is extensively involved.

*Proctoscopic or Sigmoidoscopic Examination.* An examination by means of the pneumatic proctoscope or sigmoidoscope should always be made in order to verify the findings of the digital examination. When the patient is not under the influence of anæsthesia the genu-pectoral position is the best for the purpose, hut under an anæsthetic the left lateral position is the most serviceable. In cases of rectal cancer it is very rare to find evidence of inflammation of the uninvolved portion of the mucosa. In some instances, especially when the surface of the growth has become necrotic, the mucosa in the immediate vicinity of the growth may be inflamed when Lieherkühn's follicles are distended with mucus, hut these changes affect only a narrow area.

When the rectal mucosa generally is reddened, thrown into folds, and the appearances suggest hyperactivity of the mucous follicles, the cause is generally of an inflammatory origin, e.g. diverticulitis of the terminal portion of the pelvic colon. The proctoscope is especially useful for determining the extent of the circumferential involvement in ampullary growths. It is very difficult to determine this accurately by means of the finger because the uninvolved portion of the howel contracts considerably under the stimulus of the examination. When, however, the rectum is distended by inflation under an anæsthetic the relative proportion of involved and uninvolved parts of the rectal wall can be accurately determined.

## THE SPREAD OF CANCER OF THE RECTUM

Cancer of the rectum may spread in any of three distinct ways: (a) by direct extension through continuity of tissue; (b) by way of the venous system; or (c) by means of the lymphatic system.

(a) *Direct Extension through Continuity of Tissue.* This takes place in two directions: (1) on the mucous surface of the bowel progressively from its entire margin and (2) through the thickness of the bowel wall. The marginal increase is generally greater and more rapid in the transverse direction than in the longitudinal axis of the bowel. It is not uncommon to find that, whereas nearly the whole of the circumference of the ampulla has been invaded, the extent of the growth longitudinally is less than two inches. The growing edge undermines the more normal mucous membrane extending in the sub-mucous tissue deep to the muscularis mucosæ. Such surface extension is comparatively slow: thus, in the ampulla, for instance, it will take about six months for the growth to travel round a quarter of the circumference in an average case.

Whilst surface extension is slowly progressing, the more important deep infiltration of the muscular coat of the bowel is taking place. This infiltration probably begins at the centre or oldest part of the tumour, but owing to the fact that surface extension takes place unequally, it may happen that the most fixed and indurated portion is eccentric. The fact that the centre of the growth is opposite an important structure, such as the prostate or the base of the bladder, does not imply that penetration of the bowel is occurring at that point, and that actual invasion of the structure has taken place, although the rectum may appear to be adherent to it.

Direct extension through the muscular coat of the bowel appears to be a slow process. When the bowel wall has been penetrated the growth invades the peri-rectal fat, through which it extends until it reaches the fascia propria of the rectum. According to my observations upon this point the fascia propria is not usually invaded until the growth has existed long enough for more than three-quarters of the circumference of the ampulla to have been encompassed, thus indicating that the disease has existed for about eighteen months (figs. 690 and 691).

- It is only after penetration of the fascia propria that invasion of neighbouring structures such as the sacrum, uterus, or vagina, prostate

or bladder can take place; and it would appear that involvement of adjacent structures by direct extension does not occur until the expiration of at least a year after the first appearance of objective symptoms.

It will be seen, therefore, that the mode of spread through continuity of tissue is a comparatively slow process, and that direct



*Fig. 690* —AN ADENOID CARCINOMA OF THE AMPULLA INVOLVING THREE QUARTERS OF THE CIRCUMFERENCE. THE GROWTH IS DEEPLY ULCERATED WITH SINUSOUS, EVERTED AND INFLAMED MARGINS. THE WHOLE THICKNESS OF THE MUSCULAR COAT HAS BEEN PENETRATED BY DIRECT EXTENSION, BUT THE PERI-RECTAL FATTY TISSUE IS NOT YET INVADDED. SYMPTOMS HAD BEEN IN EXISTENCE FOR A YEAR SO THAT THE PROBABLE DURATION OF THE DISEASE IS ABOUT EIGHTEEN MONTHS.

*(From a specimen in the Museum of the Cancer Hospital.)*

invasion of neighbouring structures does not take place until the growth in the rectum has involved the greater part of the circumference of the bowel.

If extension of cancer through continuity of tissue were the only or even the usual mode of spread from the rectum, the surgical treatment of the disease would be quite simple because, unless the growth has extended beyond the confines of the fascia, a restricted operation, entailing nothing but the removal of the portion of the musculo-



membranous tube containing the cancer in its interior, would be all that was necessary to rid the patient completely of his disease. Unfortunately, however, other and more important modes of spread take



*Fig. 631.*—AN ADENOID CARCINOMA OF THE ANTILOA INVOLVING FIVE SIXTHS OF THE CIRCUMFERENCE. THE GROWTH HAS NOT ONLY PENETRATED THE ENTIRE THICKNESS OF THE MUSCULAR COAT, BUT HAS EXTENDED ACROSS THE PERI-RECTAL LYMPH SINUS AND HAS SPREAD DEEPLY INTO THE PERI-RECTAL FAT. THE GROWTH, HOWEVER, HAS NOT EXTENDED BEYOND THE FASCIA PROPRIA SO THAT THE RECTUM WAS NOT FIXED TO ADJACENT STRUCTURES. SYMPTOMS HAD EXISTED FOR A YEAR AND A HALF SO THAT THE DISEASE WAS PROBABLY OF TWO YEARS' DURATION.

(From a specimen in the Museum of the Cancer Hospital.)

place simultaneously and with greater rapidity, leading to distant dissemination even when the primary growth is still in an early phase of development.

(b) *Through the Venous System.* There is no doubt whatever that cancer cells sometimes penetrate into the interior of small veins and, becoming detached, are carried in the blood stream. The actual

invasion of a venous radical is occasionally seen in microscopical sections of carcinomatous tumours. As the rectal veins belong to the portal system, cancer cells penetrating them are carried straight to the liver. The metastasis thus produced by an embolus of cancer cells is single and is usually situated in the middle of the right lobe of the liver.

Upon two occasions, at the Cancer Hospital, small single metastases in the right lobe of the liver have been discovered during post-mortem examinations of patients who have died after an operation for removal of the cancerous rectum. In both instances the primary growth in the rectum had involved less than half the circumference of the bowel and, therefore, was in a comparatively early stage of development. In neither case was there evidence of extra-mural lymphatic spread nor was the liver enlarged or the seat of surface metastases. It is fortunate that dissemination by the veins is very rarely encountered, so that in practice we can disregard the possibility of its occurrence.

(c) *By means of the Lymphatic System.* Infinitely more important is the dissemination of cancer cells through the lymphatic channels, and a knowledge of the lymphatic system is essential to the performance of any radical operation on cancer.

#### ANATOMY OF THE LYMPHATICS OF THE RECTUM

Anatomists describe the rectal lymphatics in three groups, intra-mural, intermediary, and extra-mural.

(1) The *intra-mural* lymphatics are contained in the wall of the rectum and consist of two distinct networks, one situated in the submucous tissue and the other between the muscular layers. The two networks communicate freely with one another by means of short radial channels which pass through the circular muscular coat. The submucous network of the ampulla (fig. 692) is said to be continuous above with that of the pelvic colon and below with a similar network in the anal canal. The latter is also in communication with the lymph plexus in the subcutaneous tissue of the peri-anal skin, from which collecting stems pass forwards, in the furrow between the perineum and the inner aspect of the thigh, to terminate in the innermost of the horizontal set of inguinal glands. The intermuscular network of the rectum is also said to communicate above with that of the pelvic colon and below with the lymphatics of the external sphincter muscle. The

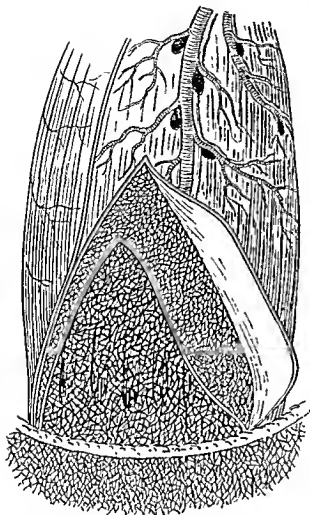


Fig 692.—DIAGRAMMATIC REPRESENTATION OF THE INTRA-MURAL LYMPHATIC SYSTEM OF THE RECTUM. THE SUBCUTANEOUS NETWORK COMMUNICATES FREELY AT THE MUCO-CUTANEOUS JUNCTION WITH THE SUBCUTANEOUS PERI-ANAL LYMPHATIC PLEXUS. THE INTER-MUSCULAR NETWORK, SITUATED BETWEEN THE CIRCULAR AND LONGITUDINAL MUSCULAR COATS, COMMUNICATES ON THE ONE HAND WITH THE SUBCUTANEOUS NETWORK AND ON THE OTHER WITH THE EXTRA-MURAL LYMPHATIC SYSTEM.

collecting stems traverse the external muscular coat of the rectum and terminate in the lymph channels of the intermediary system.

(2) The *intermediary* lymphatic system consists of two parts, namely, a subserous network in the portion of the rectum which is covered by peritoneum, and a lymph sinus, situated between the external muscular coat and the peri-rectal fat, in that part of the rectum which does not possess a peritoneal investment. This lymph sinus can always be demonstrated in any specimen of the rectum which retains its covering of peri-rectal fat. It is not a free space, but is occupied by a delicate wide-meshed reticulum consisting of fine strands

of connective tissue (fig. 693). It is possible that the connecting stems from the intermuscular network, after traversing the external coat, empty themselves directly into this lymph sinus instead of passing across it.

(3) The *extra-mural* lymphatic system is the most important of the three (fig. 694). The collecting stems from the lymph sinus form an extensive plexus and enter into relation with the ano-rectal glands of Gerota, which are scattered over the surface of the rectum among the

branches of the superior hæmorrhoidal vessels. The efferents from this plexus and from the ano-rectal glands pass to their ultimate destination in three directions, namely, downwards, laterally, and upwards.

The downward efferents traverse the fatty tissue of the ischio-rectal fossa in company with the inferior hæmorrhoidal vessels, enter into relation with a small group of lymph nodes situated close to the exit of Alcock's canal, then pass through the canal and empty themselves into the internal iliac glands. The lateral efferents enter a plexus situated between the levator ani muscles and the pelvic fascia, from which collecting vessels pass to the obturator gland situated at the upper border of the obturator foramen. The efferents from the obturator gland pass to the internal iliac glands and to the innermost of the group of glands accompanying the external iliac vessels.

The upward main efferents accompany the superior hæmorrhoidal veins, enter the lowermost mesocolic (retro-rectal) glands, then accompany the inferior mesenteric vein as it lies in the parietal border of the pelvic mesocolon and



Fig. 693.—THE PERIRECTAL LYMPH SINUS. IT IS SITUATED BETWEEN THE EXTERNAL MUSCULAR COAT OF THE RECTUM AND THE PERIRECTAL FATTY CONNECTIVE TISSUE. THE DRAWING HAS BEEN MADE FROM A MICROSCOPICAL SECTION TAKEN FROM A SPECIMEN OF CANCER OF THE AMPULLA. THE SMALL DARK CLUSTERS SEEN IN THE LOOSE CONNECTIVE TISSUE ARE CARCINOMA CELLS (IN THE SECTION THEY WERE SEEN TO BE STAINED BY MUCICARMINE) WHICH REACHED THE SINUS BY WAY OF THE INTRA-MURAL LYMPHATICS.

(Pathological Department of the Cancer Hospital.)

finally enter the group of glands situated at the bifurcation of the left common iliac artery and also the median lumbar (aortic) glands. Some of the efferents from the uppermost part of the plexus enter into relation with the paracolic glands situated along the mesenteric border of the pelvic mesocolon, from which collecting stems accompany the sigmoidal vessels to end in the median lumbar glands.

Such being the anatomical arrangement of the lymphatics in connection with the rectum, the tissues through which the lymphatics pass are liable to be invaded by cancer cells which have become detached from the primary growth.

Now, if there were a continuous submucous plexus of lymphatics such as has been described anatomically, we should expect, in cancer of the rectum, that visible metastases in the mucosa, either singly or in chains, ought to be of frequent occurrence both above and below the growth. On this point I can state positively that there is not a single specimen in the Museum of the Cancer Hospital that shows the existence of such metastases, and I doubt if anyone has ever seen them. Some years ago Mr. Sampson Handley, in his Hunterian lecture upon the spread of cancer, produced a specimen which he, at that time, thought showed continuous spread of cancer cells in the submucosa of a carcinomatous rectum for a distance of several inches above the primary growth.

These cells took up a mucicarmine stain, and, as it was believed that no other cells except those containing mucus were capable of being stained by mucicarmine, there was strong presumptive evidence that they were epithelial and malignant. Such an extensive spread of cancer cells in the submucosa of the rectum was considered to be of great importance and appeared to explain the clinical observation that, in some instances, recurrence took place in the proximal end of the bowel after excision of the rectum, even when the bowel had been divided several inches above the level of the growth. The fact, however, that metastases in the submucosa were never met with created doubt as to whether the cells with an affinity for mucicarmine were really cancer cells spreading in the submucosa.

With a view to determining this, Archibald Leitch investigated a considerable number of specimens of the cancerous rectum and failed to find mucicarmine-stained cells in the submucosa, though he encountered morphologically similar cells which are not specifically coloured. In a few instances, however, in non-cancerous rectums and other parts of the intestine, he found that these cells took up the mucicarmine stain but none of the other specific

mucin stains. He believed that the cells mentioned by Handley are the ganglionic cells of Auerbach's plexus which, for some reason or other, occasionally stain diffusely with mucicarmine. Whatever the origin of these cells may be there is no doubt that they are not cancer cells.

Observations carried out at the Cancer Hospital show that the spread of cancer cells in the submucosa of the rectum is very limited



Fig. 694.—SCHEMATIC REPRESENTATION OF THE EXTRA-MURAL LYMPHATIC SYSTEM OF THE RECTUM AND THE PELVIC COLON. THE EFFERENTS PASS IN THREE DIRECTIONS, DOWNWARDS THROUGH THE ISCHIO-RECTAL FOSSA, Laterally BETWEEN THE LEVATORES ANI AND THE PELVIC FASCIA AND UPWARDS IN THE PELVIC MESOCOLON.



Fig. 695.—SCHEMATIC REPRESENTATION SHOWING THE ARBORESCENT ARRANGEMENT OF THE SUBMUCOUS LYMPHATIC NETWORK. THE LYMPH CHANNELS OF SMALL CIRCUMSCRIBED AREAS ARE COLLECTED INTO SHORT RADIAL STEMS WHICH PASS BETWEEN THE FIBRES OF THE CIRCULAR MUSCULAR COAT TO THE INTER MUSCULAR NETWORK. THE ARBORESCENTS OF ADJACENT AREAS DO NOT COMMUNICATE WITH ONE ANOTHER.

in extent and does not extend more than a few lines beyond the microscopical margin of the growth. Leitch believed that the lymphatics of the mucosa do not exist as a continuous plexus, but are arranged as decussating arborescents from the collecting stems which pierce the circular muscular coat (fig. 695). Spread in the intermuscular lymphatics is just as limited. We may conclude, therefore, from clinical as well as histological findings that the intra-mural spread of cancer of the rectum is always of comparatively trivial extent.

These observations lead us to believe that detached cancer cells pass through the bowel wall somewhat rapidly by means of the

intra-mural lymphatic system, and gaining the external lymphatics give rise to extra-mural metastases scattered over a fairly wide area, long before the muscular coat has been penetrated by direct extension of the growth.

Cancer cells having passed through the submucous and the inter-muscular lymphatic systems gain access either to the subserous network or to the lymph sinus. Lodged in the subserous network, they may give rise to plaques in the peritoneal covering of the bowel; when, however, they enter the lymph sinus they soon involve the extramural lymphatic system. Cancer cells lodged in the lymph sinus are shown in figure 693.

The direction in which the cells travel is largely influenced by the direction of the current in the lymphatics which have been invaded. It must be remembered, however, that the path by which the cells advance is not entirely controlled by anatomical considerations. Thus it may happen that cancer cells derived from a growth in the rectum, wherever situated, may traverse the lymphatics in a downward, lateral, or upward direction, or in all three directions simultaneously. During the transit their progress may be arrested at any point in the region traversed by the lymphatics and so lead to the formation of nodules. The various tissues through which the extra-mural lymphatics pass, therefore, are prone to metastatic deposit which is either macroscopic or microscopic in character. It may be regarded as an axiom that whenever a visible metastasis exists, other metastases, which cannot be recognised without the aid of the microscope, also exist along the course of the lymphatics, at points more distant from the seat of the primary growth.

We may consider the extra-mural paths in three divisions, remembering that communications exist between them.

(1) *The Zone of Downward Spread.* The structures comprised in this zone are the peri-anal skin, the ischio-rectal fat, and the external sphincter muscle. Owing to the free intercommunication between the lymphatics of those structures and the efferents from the rectum, it is easy to understand how the progress of a detached cancer cell may be arrested at any point in that extensive network. It is also apparent that even an exhaustive microscopical examination of the structures above mentioned might fail to detect such isolated cancer cells. Nevertheless, the presence of minute masses of cancer cells has been revealed in microscopical sections often enough to establish the fact beyond doubt that these structures are liable to be invaded during the process

of dissemination. Additional evidence upon this point is forthcoming from clinical records in regard to post-operative recurrent growths.

In eight of my early operation cases in which a restricted type of perineal excision had been performed for primary growths, situated at a distance varying from half an inch to four inches above the anal margin, recurrence took place in the peri-anal skin. I have also met with sixteen instances of recurrent growths involving the peri-anal skin following peri-anal excisions performed by other surgeons.

The peri-anal skin, therefore, must be regarded as being liable to involvement even in cases where the primary growth is situated in the upper part of the ampulla or as high as the recto-sigmoidal junction, and should be widely removed in every case of excision of the rectum for cancer.

I have seen post-operative recurrence in the ischio-rectal fat on thirty-one occasions. Seven of these followed my own perineal excisions, one occurred after an abdomino-perineal operation, and twenty-three followed perineal excisions performed by other surgeons. In addition to this evidence of the liability of this tissue to invasion by cancer cells, the microscope revealed the presence of a minute metastasis in the ischio-rectal fat in a specimen which I removed by my abdomino-perineal method. I have seen recurrent growths involving the external sphincter muscle upon four occasions, once after one of my own resection operations, twice after similar operations performed by other surgeons and once after an abdomino-anal operation.

(2) *The Zone of Lateral Spread.* This area embraces the structures the lymphatics of which enter into relation with the extensive lymphatic network between the levatores ani and the pelvic fascia. These structures are the levator ani muscles, the coccygei, the pelvic peritoneum, the prostate gland, the base of the bladder, the cervix uteri, the base of the left broad ligament and the internal iliac glands.

The levatores ani are sometimes the seat of metastases. I have encountered such in the substance of the muscle close to its attachment to the rectum and also near the origin from the pelvic wall during the performance of excision operations, both primary and secondary. In one of the specimens which I removed by the abdomino-perineal method, two separate plaques of growth were found in the substance of the left levator (fig. 696). These muscles are exceedingly prone to invasion by cancer cells, which gain access to the extra-mural lymphatics before the deep infiltration of the primary growth has had





Fig. 606.—CARCINOMA OF THE LOWER PART OF THE RECTAL AMPULLA, WITH EXTENSIVE INVOLVEMENT OF THE THREE ZONES OF SPREAD BY METASTATIC DEPOSITS. THE RETRO-RECTAL GLANDS ARE EXTENSIVELY INVOLVED. A LARGE NUMBER OF HYPERPLASTIC LYMPH NODES ARE ALSO INVOLVED. IN THE SUBSTANCE OF THE LEFT LEVATOR ANI AND IN THE LYMPHATIC PLEXUS ABOVE THAT MUSCLE, AND ALSO IN THE ISCHIO-RECTAL FAT, THE HYPERPLASTIC LYMPH NODES WERE FOUND TO CONTAIN CANCER CELLS (Pathological Department of the Cancer Hospital.)

time to penetrate the muscular coat of the bowel, and they should, in all cases, be completely removed when a cancerous rectum is excised.

I have observed plaque deposits in the peritoneum of the pelvic floor upon so many occasions that it would not be an exaggeration to say that there is not a portion of it, from the middle line to the brim of the true pelvis, that has not been implicated. This is no doubt due to the fact that the peritoneum in this locality is in close relationship with the extensive lymphatic network situated between the levatores ani and the pelvic fascia. A metastatic deposit in this network, therefore, speedily penetrates the pelvic fascia and extends into the overlying pelvic peritoneum. Accordingly it is essential that the peritoneum of the entire pelvic floor, as far as the brim of the true pelvis on both sides, should be removed in every case of extirpation of the cancerous rectum in which immunity from recurrence is hoped for.

### (3) *The Zone of Upward Spread.*

The tissues of this zone are the retro-rectal (lowermost mesocolic) glands, the pelvic mesocolon in its entirety, the paracolic glands, the glands situated at the bifurcation of the left common iliac artery, and the median lumbar (aortic) glands.

Since the majority of the efferent lymphatics, which form the intra-mural lymphatic system, either pass through or terminate in the structures contained in this zone, it follows that these structures constitute the principal paths by which

cancer cells spread from primary growths in the rectum. In fact, this is the most constant and, therefore, the most important of all the routes of spread. We may find either macroscopic or microscopic metastases scattered throughout the zone in practically all cases of cancer of the rectum, even when the growth is in an early stage of development.

In some instances the retro-rectal glands are obviously enlarged and can be readily felt, but, in the majority, though the fresh specimen is carefully palpated, it is extremely difficult to be certain whether the glands are enlarged or not. Nevertheless, when these specimens have been hardened and dissected, the involvement of the glands is evident (fig. 697). Moreover, microscopic examination of the glands always shows one or more of them to be invaded by cancer cells.

'It sometimes happens that a gland may be free from deposit although the tissues surrounding it are definitely invaded by cells. In such a case, if the glands alone were examined and found to

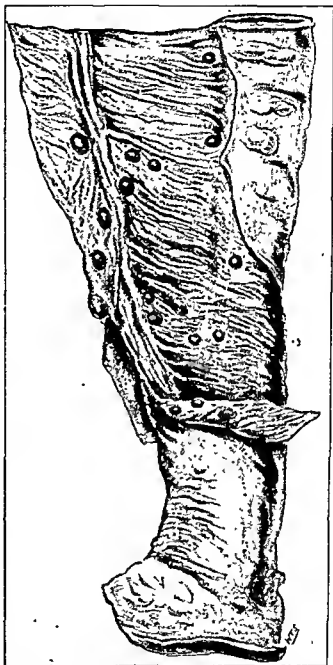


Fig. 697.—A DISSECTED SPECIMEN OF A CANCEROUS RECTUM SHOWING THE DISTRIBUTION OF INVADDED LYMPHATIC GLANDS. THE MAJORITY OF THE GLANDS ARE SITUATED ALONG THE COURSE OF THE SUPERIOR HÆMORRHOIDAL AND THE INFERIOR MESENTERIC VESSELS IN THE PARIENTAL BORDER OF THE MESOCOLON. THE AFFECTED PARACOLIC GLANDS ARE AT A CONSIDERABLE DISTANCE FROM ONE ANOTHER, INDICATING THAT THE GLANDS HAVE NOT BEEN INVADDED IN CONTINUITY OF THE SERIES. THREE WELL-MARKED PLAQUES EXISTED IN THE PELVIC PERITONEUM IN THIS CASE.

(From the Pathological Department of the Cancer Hospital.)

be free from invasion, an erroneous conclusion as to the absence of extra-mural spread might be arrived at and a more hopeful prognosis, in regard to the possibility of recurrence taking place in adjacent structures, might be given than would be warranted had a more thorough examination been made.

I have also seen carcinomatous deposits in this group of glands in a case of cancer situated at the middle of the loop of the pelvic colon, thus illustrating downward spread from that locality. The pelvic mesocolon, throughout its extent, is particularly liable to invasion. So often, indeed, is it found to be the seat of metastatic deposit that the removal of the whole of this structure in every operable case of cancer of the rectum is just as important as is thorough clearance of the axilla in breast cancer.

The part of the pelvic mesocolon which is invariably invaded is its parietal border, between the layers of which the superior hæmorrhoidal and the inferior mesenteric vessels are situated. The efferent lymphatics from the retro-rectal glands accompany these vessels on their way to the glands located at the bifurcation of the left common iliac artery, and constitute the principal route by which cancer cells from the rectum are carried by the lymph stream. At any point along this line metastases may occur, either singly or in chains. A well-marked chain of metastases situated along the line of the inferior mesenteric vessels is shown in figure 698.

In this instance the primary growth was situated at the upper part of the ampulla and involved three-quarters of the circumference of the bowel. The growth extended through the entire thickness of the bowel wall, and, having crossed the lymph sinus, invaded the peri-rectal fat; but did not involve the fascia propria, so that the diseased portion of the rectum preserved its mobility. The metastases in this case were not visible at the operation when the abdomen was opened because the peritoneum covering them had not been involved; but they could be felt distinctly, as a chain of nodules situated in the parietal border of the pelvic mesocolon, extending as far upwards as the promontory of the sacrum.

It does not often happen that metastases in this situation are as well developed as they were in this particular instance, but minute examination of a large number of specimens of cancerous rectums proves that they invariably exist, though they may be small enough to escape detection until the tissues have been hardened and a careful dissection made. I have been obliged to abandon an abdomino-perineal operation on account of a chain of palpable metastases extending upwards

along the inferior mesenteric vessels as far as the origin of that artery from the aorta.

There were no other visible metastases, either in the pelvic peritoneum or in the pelvic mesocolon, the primary growth involving not more than a third of the circumference, thus indicating that distant



Fig. 608.—AN ADENOID CARCINOMA IN THE UPPER PART OF THE AMPULLA WITH METASTASES IN THE ZONE OF UPWARD SPREAD. THE GROWTH INVOLVES THREE QUARTERS OF THE CIRCUMFERENCE OF THE BOWEL. IT HAS EXTENDED THROUGH THE ENTIRE THICKNESS OF THE WALL AND HAVING TRAVERSED THE LYMPH SINUS, HAS INVADDED THE LATTER, BUT HAS NOT YET REACHED THE FASCIA PROPRIA. SEVERAL METASTATIC DEPOSITS ARE SEEN ALONG THE COURSE OF THE INFERIOR MESENTERIC VESSELS. TWO PARACOLIC ISLANDS AT A CONSIDERABLE DISTANCE ABOVE THE PRIMARY GROWTH ARE INVADDED.

(Pathological Department of the Cancer Hospital)

extra-mural spread may occur during an early phase of the disease. There is scarcely a part of the pelvic mesocolon in which, from time to time, I have not observed metastatic deposits, so that the whole of the structure must be regarded as highly dangerous tissue.

The paracolic glands, which are in series with the ano-rectal glands and are situated along the mesenteric border of the pelvic colon, are often the seat of metastatic deposit. The involvement of these glands does not proceed from one to the other *seriatim* as might be expected, for a continuous chain of affected glands is never seen. When a paracolic gland is found to be involved it is generally isolated and may be situated at a considerable distance from the level of the primary growth.

It is not uncommon to find an invaded gland, several inches above the rectal growth, with no evidence of involvement of others of the same group. Instead of spread taking place from one paracolic gland to another, it is probable that the cancer cells first follow the course of the lymphatics accompanying the inferior mesenteric vessels and then, owing to blockage of the normal lymph stream by metastatic deposits, grow continuously or are carried by reversed flow along the lymphatic vessels of the pelvic mesocolon towards the colon to be arrested in one of the paracolic glands (fig. 699).

When, therefore, the rectum, together with part of the pelvic colon, has been removed by a perineal method of excision, it is quite possible that an invaded paracolic gland, above the level of the point of section of the bowel, may have been left behind, despite the fact that a careful examination of all the paracolic glands contained in the specimen has failed to demonstrate evidence of infection.

Even the extensive removal of the pelvic colon by means of the abdomino-perineal operation may sometimes fail to include an invaded paracolic gland when situated many inches above the level of the primary growth. The following case is a good example of this. A female, aged 50, was admitted into the Cancer Hospital with a carcinomatous stricture at the recto-sigmoidal junction. When the abdomen was opened the bowel, just above the peritoneal reflexion in Douglas's pouch, was found to be puckered by a narrow constricting band, the type of cancer so commonly met with in this situation. There was no macroscopic evidence of extra-mural spread. The radical abdomino-perineal operation was performed, the whole of the pelvic colon and its mesocolon being removed. Ten inches of bowel above the level of the growth was excised.

Subsequent dissection of the specimen showed that the retro-rectal

glands were the seat of metastatic deposit and that the paracolic glands were not involved. The operation was performed in December, 1909, and in July, 1911, nineteen months later, a recurrent nodule developed

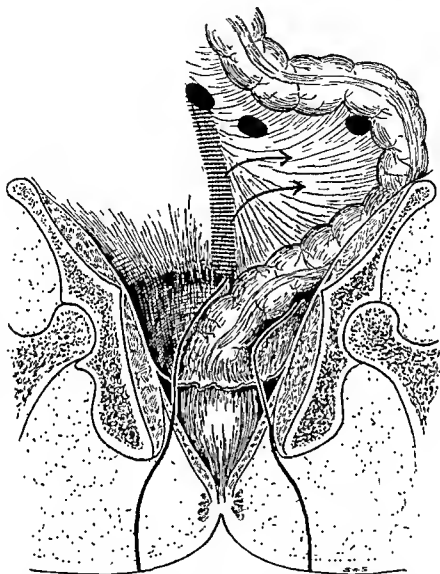


Fig. 600.—ILLUSTRATING THE PATHS OF THE UPWARD SPREAD OF CANCER CELLS FROM THE RECTUM. THE MAIN ROUTE OF THE SPREAD IS ALONG THE SUPERIOR HEMORRHOIDAL AND THE INFERIOR MESENTERIC VESSELS AS THEY LIE IN THE ROOT OF THE PELVIC MESOCOLON (INDICATED BY THE SHADED AREA). THE BLACK AREAS INDICATE THE RELATIVE POSITION OF THE METASTASES AS THEY EXISTED IN THE SPECIMEN FROM WHICH THE DRAWING WAS MADE. THE ARROWS INDICATE THE ROUTE BY WHICH CANCER CELLS, FROM THE MAIN LINE OF SPREAD, REACH THE PARACOLIC GLANDS.

(Pathological Department of the Cancer Hospital.)

in the lower margin of the colostomy opening. There can be no doubt that a paracolic gland, situated immediately above the point of section of the bowel, and already the seat of an invisible metastasis, escaped removal at the time of the operation.

There is ample evidence, then, that the dissemination of cancer cells by means of the extra-mural lymphatic system is more widespread and of much greater consequence than that which takes place in the intra-mural lymphatics. Moreover, it appears that, of the three zones of possible extra-mural spread, the upper zone is the most important because secondary deposits are always present, visible to the naked eye, or discernible by the microscope.

It occasionally happens that cancer cells are disseminated along the tissues of all three zones simultaneously, as shown in figure 696. This specimen was removed by the abdomino-perineal method from a female patient, who was admitted into the Cancer Hospital with a recurrent villous papilloma of the rectum that underwent carcinomatous change. The retro-rectal glands were full of cancer cells, and in the pelvic mesocolon a large number of hyperplastic lymph nodes existed along the course of the blood-vessels. Several of the paracolic glands were invaded by cancer cells. In the substance of the left levator ani muscle there were two nodes containing cancer cells and a similarly invaded node was found in the lymphatic plexus situated upon the upper surface of the muscle. In addition to these a minute metastasis was discovered in the ischio-rectal fat.

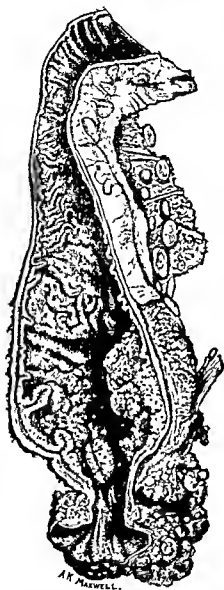


Fig. 700.—AN INTERIOR VIEW OF THE SPECIMEN SHOWN IN FIG. 696. THE GROWTH HAS NEARLY ENCIRCLED THE BOWEL; BUT HAS NOT EXTENDED THROUGH THE MUSCULAR COAT. THE WIDESPREAD DISSEMINATION IN THE EXTRA-MURAL TISSUES IS IN MARKED CONTRAST TO THE LIMITED DEGREE OF INVASION OF THE BOWEL WALL. (Pathological Department of the Cancer Hospital.)

This specimen is particularly interesting because it illustrates invasion of the three zones of spread by a cancer, which was still in such a clinically early stage of development that the direct extension

of the primary growth, through continuity of tissue, had not involved the whole thickness of the wall of the howel (fig. 700).

As a result of (a) noting the position of metastases during the performance of operations; (b) observing the locality affected by recurrent growth either clinically or during the performance of secondary operations; (c) recording the extent and the position of metastases in cases in which proposed operations had been abandoned on account of their presence; (d) studying the post-mortem findings in regard to the spread of cancer in patients, who had died from advanced and inoperable cancer; and (e) following the detailed examinations, macroscopic and microscopic, of specimens removed by operation, I have been able to construct a map indicating the tissues that are liable to metastatic deposit during the progress of the disease and to recurrence after restricted operations.

It will be seen that the ischio-rectal fat, the levatores ani muscles, the pelvic peritoneum, and the pelvic mesocolon are the tissues which are chiefly concerned in the spread of cancer from the rectum. Pathology teaches us that they may be the seat of metastatic deposits even when the growth in the rectum is in a clinically early stage, and that unless these highly dangerous tissues are completely removed in every case in which an operation for the removal of the cancerous rectum is undertaken, post-operative recurrence will be a rule to which there will be few exceptions.

#### OPERABILITY OF CANCER OF THE RECTUM

It is a common experience that a very large proportion of the cases of cancer of the rectum that one sees have passed beyond the stage of useful operation. Of the cases in which the decision in regard to operability rested with me, I have operated upon about 35 per cent. This somewhat low rate of operability agrees with that of most surgeons in this country, who place it at from 25 to 30 per cent. Opinions, however, differ considerably. Thus, among Continental authorities we find that Boas regards 19, Witzel 25, Czerny 71, Bergmann 80, Eiselberg 65 per cent as cases suitable for operation. This marked discrepancy in opinion is no doubt due to individual ideas in regard to the criteria of operability.

Those who maintain that it is not advisable to attempt to remove a growth unless uninvolved mucosa above it can be reached by the examining finger must of necessity limit the number of operations they perform. On the other hand, those who consider that a growth, where-



ever situated in the rectum, is amenable to operation, provided that the diseased portion of the bowel preserves its mobility, must operate upon a larger proportion of cases.

In estimating operability several things must be taken into consideration in addition to the condition of the growth itself. The operation for removal of the cancerous rectum is a severe one, and should not be undertaken unless the patient's general condition is good. Age is an important point. Patients over 65 years of age do not stand severe operations well, and every additional year adds to the risk of the operation. More important perhaps than actual age is the general condition of a patient's tissues. Fat subjects are not good operation risks, nor are those in whom there is evidence of arterio-sclerosis. Freedom from such diseases as tuberculosis, diabetes, Bright's disease, etc., is essential. Provided, then, that there is nothing in a patient's general condition to render an operation inadvisable, operability is governed by the local condition of the growth and by the spread of the disease extra-murally.

The local condition of the growth can be recognised by digital examination, but the extra-mural spread can only be determined by abdominal exploration.

*Determination of Operability by Digital Examination.* It is generally supposed that so long as the part of the bowel involved by the growth remains mobile, definite proof exists that the disease has not extended beyond the rectal wall. We have seen, however, that fixation to surrounding structures cannot take place until the fascia propria of the rectum has been penetrated, and that long before that happens extensive dissemination may have taken place in the tissues of the three zones of spread. Mobility of the diseased portion of the bowel, therefore, means nothing except that it is possible to remove the rectum.

When the growth is situated at the recto-sigmoidal junction, on account of the stenosis produced by circular infiltration, it is generally found to be operable because symptoms of impending obstruction cause the patient to seek advice during the early stages of the disease.

Similarly a growth located in the anal canal, provided that it has developed there and has not extended downwards from the ampulla, is practically always in an operable state.

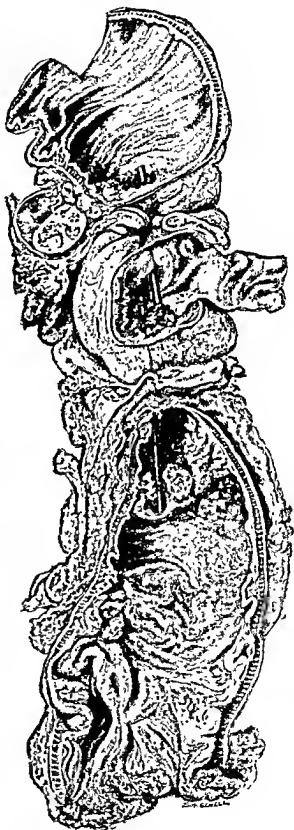
When, however, the growth is in the ampulla it is impossible, even when it appears to be in an early phase of development, to determine by digital examination alone that it can be removed with a reasonable

prospect of the operation conferring immunity from recurrence. This is due to the fact that the disease has been in existence for a considerable time before objective symptoms make their appearance.

I have so often seen metastases scattered in the pelvic peritoneum and in the pelvic mesocolon in cases in which the primary growth did not involve more than a quarter or a third of the circumference of the bowel and was freely mobile, that I am convinced of the impossibility of determining the operability of ampullary cancers without abdominal exploration.

Fixation of the diseased part of the rectum, either posteriorly to the sacrum, anteriorly to the prostate or bladder, or to the uterus or vagina, or laterally to the wall of the pelvis, indicates that the growth has extended beyond the confines of the fascia propria, and that operation is out of the question.

*Determination of Operability by Abdominal Exploration.* It having been decided from



*Fig 701.*—ADENOID CARCINOMA OF THE RECTO-SIGMOIDAL JUNCTION EXTENDING COMPLETELY ROUND THE BOWEL AND PRODUCING MARKED STENOSIS. A GROWTH OF SIMILAR TYPE EXISTS IN THE TERMINAL PORTION OF THE PELVIC COLON. IN BOTH SITUATIONS THE LUMEN OF THE BOWEL ONLY JUST ADMITTED A FINE METAL ROD. A PARA-COLIC GLAND OPPOSITE THE UPPER GROWTH IS HEAVILY INVADIED.

(From a specimen in the Museum of the Cancer Hospital.)

digital exploration that it is possible to excise the rectum, the final decision as to operability should be left until the abdomen is opened at the time of the proposed operation. A paramedian incision should be made from the umbilicus to the symphysis pubis with the patient in the Trendelenburg position, so that a complete exposure of the pelvis may be obtained. The condition of the pelvic peritoneum and the whole extent of the pelvic mesocolon can then easily be ascertained.

If the growth is situated in the portion of the rectum above the peritoneal reflexion, the peritoneal surface of the bowel should be carefully examined for plaques. Search should also be made for plaques in the pelvic peritoneum by carefully palpating the whole of its surface, especially at the point of reflexion on to the bladder or vagina, as the case may be. The parietal border of the pelvic mesocolon is then palpated throughout its extent for enlarged glands or metastatic deposits. The mesenteric border of the pelvic colon should be examined for enlarged para-colic glands throughout its extent, and finally the mesocolon itself for metastases.

If neither plaques nor metastases are either felt or seen it must not be assumed that they do not exist, because they may be present in a microscopical state, but, nevertheless, the case may be considered to be operable.

If plaques and metastases are felt but the peritoneum is intact over them, provided that they are not situated too near the limits of the operation field, the case may be deemed to be operable. Should, however, the peritoneum covering existing plaques or metastases have been penetrated so that the actual growth is discernible on the surface, then the case is inoperable because general peritoneal carcinomatosis will soon develop. It occasionally happens that a growth in the pelvic colon co-exists with one in the rectum, a condition which can only be determined by abdominal exploration (fig. 701).

#### THE BEARING OF PATHOLOGY UPON THE OPERATIVE TREATMENT OF CANCER OF THE RECTUM

The efficacy of an operation for cancer is measured by its recurrence rate. If a post-operative recurrent growth be found in the neighbourhood of the operation field, then either the disease had advanced beyond the possible limits of the operation or the scope of the operation was not sufficiently comprehensive to embrace the whole of the invaded removable tissue. We have seen that the chief path by which extramural spread from a cancerous growth in the rectum takes place is

along the course of the inferior mesenteric vessels and in the substance of the pelvic mesocolon. These structures are out of reach of any operation undertaken solely from the perineum, no matter what method of exposure be employed, so that it is not to be wondered at that a high recurrence rate has prevailed after perineal methods of excision.

My early perineal excision operations were of the restricted type in vogue at that time. They consisted of an exposure by means of excision of the coccyx, narrow removal of the peri-anal skin, scanty removal of the ischio-rectal fat, division of the levatores ani close to their attachments to the rectum, and division of the bowel an inch or

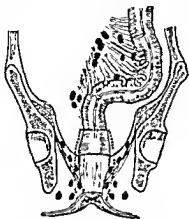


Fig. 702.—SHOWING THE RESTRICTED NATURE OF THE OPERATION CARRIED OUT BY EARLY METHODS OF PERINEAL EXCISION (KRASKE'S, BARDENHEUER'S, CRIPPS'S, ALLINGHAM'S). THE RECTUM WAS MERELY DISSECTED OUT AS A TUBE CONTAINING A CANCER AND THE DANGEROUS TISSUES OF THE UPWARD, LATERAL AND DOWNWARD ZONES OF SPREAD, WERE LEFT BEHIND.

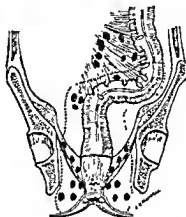


Fig. 703.—SHOWING THE SCOPE OF THE MOST COMPLETE REMOVAL OF THE RECTUM AND PELVIC COLON TOGETHER WITH TISSUES OF THE ZONES OF SPREAD THAT IS POSSIBLE BY AN OPERATION UNDERTAKEN FROM THE PERINEUM ALONE. IT WILL BE SEEN THAT THE GREATER PART OF THE PELVIC MESOCOLON IS LEFT BEHIND BECAUSE IT IS OUT OF REACH.

so above the upper margin of the growth (fig. 702). Recurrence ensued in every instance, so that the recurrence rate of that type of operation was exactly 100 per cent.

By extending the scope of the operative procedure in successive series I ultimately arrived at a removal which was the most complete that it was possible to carry out from the perineum. The type of operation was as follows: Preliminary colostomy, subsacral or Kraske's or Bardenheuer's method of exposure; wide removal of peri-anal skin; free excision of ischio-rectal fat; complete removal of the rectum encased in its covering of fascia propria; removal of the lowermost part of the pelvic mesocolon together with the retro-rectal glands; free incision of the pelvic peritoneum and the peritoneum of the pelvic mesocolon so as to permit of several inches of the pelvic colon being

drawn down ; section of the bowel at or near the middle of the loop (fig. 703).

Although this type of operation was the most extensive that it was possible to carry out from the perineum it did not suffice to reduce the recurrence rate to any appreciable extent, recurrence taking place in 94.4 per cent of the cases. Failure to prevent recurrence was due to the fact that tissues of the upward zone of spread, already invaded, lay beyond the reach of any operation carried out solely from the perineum. These tissues, representing the axilla in cases of cancer of the breast, can be approached only through the abdomen, and therefore it is obvious that an attempt to excise the cancerous rectum, in which spread has taken place in the upward zone, from the perineum alone is as futile as amputating a breast affected by cancer without also clearing the axilla of invaded lymphatic glands.

#### TREATMENT

It appears to be a fairly common belief that cancer is at first a local disease and remains localised for a considerable time. Although this may be true in certain forms of squamous carcinomata, I am quite convinced that it does not hold good for the majority of the adeno-carcinomata, and especially for adenoid cancer of the rectum. In the latter it is a common experience to encounter widespread dissemination in the peri-rectal tissues and even in the abdominal cavity in clinically early examples of the disease ; so that it seems probable that these carcinoma cells become detached from the primary growth almost synchronously with its inception and, finding their way by means of the lymph channels into the surrounding tissues, form more or less distant metastases.

The assumption that cancer remains localised for a definite period of time after its inception is no doubt responsible for the faith reposed in radium as a curative agent by those who are interested in radium therapy. So far as the rectum is concerned, the treatment of cancerous growths by radium has as yet been extremely unsatisfactory, it being my experience that, whereas local improvement may sometimes result, rapid and widespread dissemination takes place in the surrounding tissues owing to stimulation of the distant metastases. Thus it happens that despite the competition of radium therapy in the treatment of malignant disease, rectal carcinoma still remains within the domain of surgery. It behoves us, therefore, to endeavour to ascertain the best means by which surgical treatment can be carried out.

An operation undertaken for the cure of cancer, to be effective, must be based upon pathological findings in regard to the spread of cancer from the primary growth to surrounding tissues.

If the field of operation does not embrace the tissues pathologically known to be prone to invasion by cancer cells which have become detached from the primary focus, then the operation will be doomed to failure, because it will not prevent recurrence.

The whole question, therefore, of the surgical treatment of cancer of the rectum hinges upon the knowledge we have been able to obtain : first, of the manner in which cancer of the rectum spreads, and, second, of the paths along which that spread takes place.

The treatment of cancer of the rectum, therefore, is surgical. Cases are divisible into (1) those in which an operation for removal of the rectum is possible, and (2) those in which it is not.

*Surgical Treatment of Operable Cases.* Whenever the rectum is attacked by cancer it must be completely excised, together with the tissues comprised in the three zones of spread. The problem confronting us demands that an operative measure be adopted that will embrace the whole of the tissues which are known to be liable to involvement in the spread of cancer cells from the primary focus. The rectum, owing to the fact that the main line of advance in the invasion of the extra-mural tissues takes place in structures which are not only removable, but are sufficiently remote from the primary disease to necessitate the lapse of a certain length of time before the invading cells have progressed beyond the limits of the field of possible operation, is especially amenable to radical extirpation.

No other organ in the body is so favourably placed in this respect, and yet the results, so far as post-operative recurrence is concerned, have hitherto been extremely bad. Literature is replete with the various operative procedures that have been devised in the endeavour to elaborate a technique that will confer a reasonable immunity from recurrence. These procedures may be classified as follows :

- (1) Perineal excision.
- (2) Perineal resection.
- (3) Resection through the vagina.
- (4) Combined abdominal and perineal excision :
  - (a) The abdomino-anal operation.
  - (b) The radical abdomino-perineal operation.
  - (c) The perineo-abdominal operation.

*Perineal Excision.* By this method the rectum and several inches of the pelvic colon can be excised, but the greater part of the pelvic mesocolon is out of reach. We have seen that the main line of spread from a cancer of the ampulla of the rectum, wherever situated, is in an upward direction along the superior hæmorrhoidal vessels as they lie in the root of the pelvic mesocolon. Even after the most complete removal that can be carried out by an exposure from the perineum the greater part of the tissues forming the upward zone of spread is of necessity left behind. The operation, therefore, is not radical unless the spread happens to be confined to the lateral and downward zones.

There is no means, however, of ascertaining, even by preliminary abdominal exploration, whether the pelvic peritoneum or the pelvic mesocolon are free from invasion because it may exist in the microscopical state. It should not be assumed, therefore, that because there are no visible metastases present in the tissues of the upward zone, microscopical spread does not exist, and consequently that removal from the perineum alone is all that is needed for complete eradication of the disease. It is highly probable that a microscopical metastasis may exist for a considerable time, perhaps for several months, before attaining sufficient size to be discernible by the naked eye.

The efficiency of an operation for cancer is measured by its recurrence rate. If a particular method shows that recurrence follows in a large proportion of cases, then the method should be abandoned as inadequate for the purpose of eradicating cancer. I found that the most complete perineal excision that it was possible to carry out (fig. 703) had a recurrence rate of over 90 per cent, the recurrent growths taking place chiefly in the tissues of the upward zone of spread which were beyond reach. Consequently, I now only perform perineal excision as a palliative measure in patients whose general condition is not sufficiently good to enable them to stand the radical operation.

*Perineal Resection.* By this procedure the segment of the rectum in which the growth exists is resected and an end-to-end anastomosis effected. From the point of view of preserving natural sphincteric control it is ideal, but as a means of preventing recurrence it is useless. The scope of the operation is shown in figure 704, in which it will be seen that the tissues of the three zones of spread are left behind.

*Vaginal Resection.* This operation is practically the same as the foregoing, except that the rectum is exposed by splitting the posterior wall of the vagina.

*The Abdomino-anal Operation.* The scope of the operation is shown in figure 704. It aims at preserving sphincteric control, but does so at the risk of recurrence. The tissues of the downward, the lateral, and the greater part of the upward zones of spread are left behind. It is useless for preventing recurrence.

*The Abdomino-perineal Operation.* This is the radical operation for cancer of the rectum and aims at removal, as completely as possible, of the tissues comprising the three zones of spread. It may be carried out in one stage, namely, the one-stage abdomino-perineal

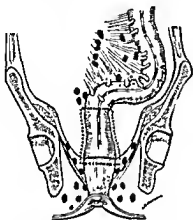


Fig. 704.—SHOWING THE EXTENT OF REMOVAL OF THE BOWEL BY RESECTION AND END TO END ANASTOMOSIS. THE DANGEROUS TISSUES OF THE THREE ZONES OF SPREAD ARE LEFT BEHIND.

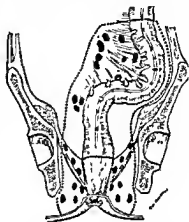


Fig. 705.—SHOWING THE SCOPE OF THE RADICAL ABDOMINO-PERINEAL OPERATION. BY THIS MEANS THE DANGEROUS TISSUES OF THE UPWARD ZONE OF SPREAD ARE COMPLETELY REMOVED. THE PELVIC MESOCOLON IS VERY FREQUENTLY FOUND TO BE THE SEAT OF METASTATIC DEPOSIT, WHICH MAY EXIST IN THE MICROSCOPICAL STATE SO THAT IT IS JUST AS IMPORTANT TO REMOVE IT COMPLETELY AS IT IS TO CLEAR THE AXILLA IN CASES OF BREAST CANCER.

(the Author's) operation, or it may be done in two stages with an interval of ten days or a fortnight between them, namely, the two-stage abdomino-perineal operation (Coffey's operation).

There appears to be a doubt, in the minds of some, as to what is exactly meant by a two-stage abdomino-perineal operation. I have heard it said that a preliminary colostomy followed after an interval of ten days or so by perineal excision is a two-stage abdomino-perineal operation. It is nothing of the sort because the tissues of the upward zone of spread are left behind. The real two-stage abdomino-perineal operation is nothing more or less than the one-stage operation performed in two stages with an interval of ten days or so between them.



The first stage ends with the reconstruction of the pelvic floor and the establishment of a terminal colostomy. The isolated portion of the pelvic colon together with the part of the rectum which has been freed, anteriorly as far as the upper part of the prostate in the male or half way down the vagina as the case may be, laterally down to the upper surfaces of the levatores ani and posteriorly down to the sacro-coccygeal articulation, is left *in situ* below the new peritoneal floor of the pelvis. The isolated bowel, having been deprived of the greater part of its blood supply from ligation of the inferior mesenteric artery, necroses and at the expiration of the interval between the stages is in an advanced state of decomposition so that in order to prevent absorption of toxic material a suprapubic gauze-wick drain is established.

Coffey maintained that the mortality of the operation in his hands was considerably reduced by his two-stage method, and that by doing so the radical abdomino-perineal operation has been brought within the compass of the surgeon of average ability.

As the result of my observations upon the paths by which the spread of cancer takes place from the rectum, and in consequence of the failure of the most complete operation carried out from the perineum alone to prevent recurrence, I planned the radical abdomino-perineal operation so as to embrace the tissues of the zone of upward spread in addition to those of the lateral and downward zones.

By this means the following are removed: the whole of the pelvic colon (with the exception of the portion to be utilised for the colostomy), together with the whole of the rectum encased in its sheath of fascia propria; the whole of the pelvic mesocolon; the peritoneum lining the floor as well as the walls of the true pelvis; the whole of the levator ani and coccygeus muscles; the external sphincter muscle, as much as possible of the ischio-rectal fat, and a wide area of peri-anal skin (fig. 705).

Although the operation is comprehensive in its aim, it should not be reserved for advanced cases only. It should be the procedure of choice for early cases; in fact, the earlier the better because then we may hope to circumvent the invisible spread of the disease. Should it be reserved for advanced cases only, as advocated by some, then the invisible spread will have advanced beyond the confines of the operation field and recurrence will be inevitable.

The operation is a surgical procedure of the first magnitude and importance, and should not be undertaken unless the patient's general condition is satisfactory. In order to ensure a successful result attention

should be paid to (1) preparation of the patient before operation ; (2) choice of anæsthesia ; and (3) details of after-treatment.

*Preparation Before Operation.* It is most important that a week or ten days be devoted to preparatory treatment. A large number of patients suffering from cancer of the rectum also suffer from intestinal stasis, due in most instances to increasing obstruction to the evacuation of the contents of the colon. In consequence, the contents of the colon are often exceedingly septic. Daily purgation and lavage not only empties the colon but reduces sepsis. In those cases in which the growth is situated at the recto-sigmoidal junction, the lumen of the bowel may be so narrowed that it is not possible to wash the colon out satisfactorily. Under these circumstances, temporary cæcostomy should be performed and the radical operation postponed for a fortnight or three weeks. The plan which I have followed for several years past, and which I have found to be very satisfactory, is as follows :

(1) If on examination of the abdomen there are no signs of intestinal obstruction the patient is given on the morning of admission 1 ounce of a mixture consisting of 2 drams of magnesium sulphate, 1 dram magnesium carbonate, and chloroform water to make 1 ounce, followed at hourly intervals by half an ounce of the same mixture until the bowels act freely. As a rule five or six doses are required. If during this treatment there should develop signs of intestinal obstruction the mixture is discontinued,  $\frac{1}{2}$  grain of morphia is injected hypodermically, and cæcostomy is done at once.

(2) On each succeeding morning until two days before the radical operation 1 ounce of the mixture is given. A colon wash-out of  $1\frac{1}{2}$  pints of plain water is administered every morning and evening, and 5 grains of dimol is given twice daily as an intestinal antiseptic. The last dose of the mixture is given on the penultimate morning before the operation, and the last wash-out on the morning before the operation, so that the intestinal canal should have complete rest for 24 hours. Several hours of sound sleep should be ensured by means of a suitable soporific.

(3) During the whole of the pre-operative period the patient is kept on a generous nourishing diet until the morning of the operation.

(4) The following investigations should be carried out before the operation : (1) Blood examination : (a) hæmoglobin, (b) blood group, (c) blood count, and (d) blood urea. (2) Urine examination : (a) twenty-four hourly specimen, (b) catheter specimen, (c) urea concentration test.

*Blood-Pressure Estimation.* Systolic and diastolic pressures are taken while the patient is at rest in bed. I regard it of very great importance that these pressures be accurately taken in order that cardiac energy can be determined by working out the Moots-McKesson "pressure-ratio percentage." This is one of the most valuable tests of operability and should never be omitted. If the pressure-ratio is below 25 per cent the operation will be fatal, and if above 75 per cent the risk to life is increased. A cardiac energy index of 50 per cent is the best possible.

A thorough examination in regard to general conditions should be made, particularly of the heart and lungs.

The patient should be kept in bed during the whole of the preparatory treatment.

*Choice of the Anæsthetic.* Ether or chloroform anæsthesia should never be employed as they produce a marked fall in blood-pressure. So far as my experience goes, the best results are obtained from gas and oxygen anæsthesia supplemented by intrathecal percaïne (15 cc. of 1-1500 solution), preceded, three-quarters of an hour before the operation, by an injection of omnopon and scopolamine.

#### TECHNIQUE OF THE RADICAL ABDOMINO-PERINEAL OPERATION

It should be borne in mind that one of the most important factors determining the success of the operation is the rapidity with which the various stages of the procedure are carried out. A definite system of working should be adopted, each step being completed in sequence. In an uncomplicated case the abdominal portion of the operation should be completed in forty-five minutes and the perineal part in fifteen minutes. Under no circumstances, however, should care be sacrificed for speed in the performance of an operation.

*The Abdominal Portion of the Operation. Position of the Patient.* Involving, as it does, an extensive and deep pelvic dissection, the most convenient and the best position for the purpose is the high Trendelenburg. It is important, therefore, to be provided with a good type of operating table. Most hospitals nowadays are equipped with these, but when operating at some nursing homes the tables provided are incapable of giving the high position, and add immensely to the difficulties of the operation.

*Incision.* The best incision is a right paramedian, half an inch from the middle line, extending from the crest of the pubis to a point an inch or more above the umbilicus. The sheath of the right rectus muscle is incised throughout the extent of the skin incision and the muscle displaced outwards. All bleeding points having been secured the peritoneum is opened in the middle line from one end of the wound to the other. This position of the incision is greatly to be preferred to the one sometimes adopted in the left linea semilunaris, for two reasons: first, because it affords greater facilities for carrying out the pelvic dissection of the right side; and, second, because it permits of the incision for the colostomy being made at some distance from the main incision, so that the latter can be adequately protected from faecal soiling during the subsequent progress of the case.

*Exposure of the Pelvic Cavity.* A self-retaining abdominal retractor having been placed in position the edges of the wound are widely retracted. Several patterns of these retractors are in use. A rapid survey of the pelvis and the rest of the abdominal cavity is now made with a view to ascertaining whether extra-mural spread of the disease exists or not. The pelvic mesocolon should be carefully examined for nodules or plaques of growth. The most common positions in which these are to be found are: (a) along the parietal border, in the course of the inferior mesenteric and superior hæmorrhoidal vessels; (b) along the margin attached to the colon, where the paracolic lymph glands exist; and (c) in the substance of the mesentery itself, anywhere between these two lines. If even quite minute nodules are discovered in the upper part of the mesocolon, it is direct evidence that widespread extra-mural extension of the disease has taken place, and the case had better be deemed inoperable; because recurrence is almost certain to ensue higher up in the median chain of the lumbar glands, or in the small intestine as a result of contact.

The failure to find evidence of visible spread in these situations does not necessarily mean that there is an absence of extra-mural extension, because such spread may exist in a microscopical state; though even in such a contingency a wide removal of the mesocolon may succeed in circumventing it; whereas if visible spread exists close to the boundaries of the operation field the widest possible removal may not get beyond the area of upward microscopical extension.

At this stage opportunity may be taken to ascertain the condition of the liver. In my experience, however, there is not much to be

gained from this. If there is recognisable disease in the liver, there is nearly always obvious extra-mural disease in the pelvis or in the peritoneum, and if the latter is still in the microscopical stage, then any existing disease in the liver is too small to be recognised, except by post-mortem examination.

The condition of the diseased part of the bowel should next be enquired into. If the growth is situated upon the anterior wall of the bowel, especial attention should be paid to possible involvement of the urinary bladder in the male, or the posterior wall of the vagina in the female. In either of these circumstances, the case is, in my opinion, inoperable. If the bladder is involved, it is not possible to remove the growth completely; and if the vagina is implicated, the additional operation of removal of the uterus and posterior wall of the vagina entails, in the majority of cases, too great a strain upon the patient's endurance.

Lastly, the peritoneum lining the recto-vesical pouch should be carefully examined for plaques. Very often they are not visible but can readily be palpated. The existence of plaques and metastases, unless situated near the periphery of the operation field, does not contra-indicate an attempt being made to perform the radical operation so long as the peritoneum covering them has not been penetrated. If only a small plaque exists, but its peritoneal covering has been penetrated so that the growth is exposed, the outlook is extremely bad because general peritoneal carcinomatosis will probably ensue at an early date.

In the absence of contra-indications the operation is performed as follows:

First of all, the pelvic cavity is cleared of small intestine. In nearly all cases there is pronounced enteroptosis, the majority of patients being at or beyond middle life. If the patient is taking the anæsthetic comfortably, and especially when the abdominal muscles are completely relaxed by spinal anæsthesia, the coils of small intestine usually drop out of sight into the upper abdomen; but in some instances this does not happen and the operator is constantly embarrassed by loops of small intestine being forced down into the pelvis during deep respiratory movements. Under these circumstances I do not hesitate to pull the small intestine out through the wound and cover it with a warm moist towel. I have resorted to this plan on several occasions, and I can confidently say that I have never seen any harm result therefrom, either from the intestine losing temperature or from it becoming temporarily congested as a result of its dependent position.

*The Pelvic Portion of the Operation. First Stage.* The pelvic colon is drawn through the wound and the position of its vessels noted. Occasionally there is some difficulty in doing this, owing to the bowel being adherent to the floor or lateral wall of the pelvis. Except when the colon is the seat of diverticulosis, the adhesions are not inflammatory in character, but are due to altered peritoneal attachments consequent upon coloptosis. When such adhesions are found, they should be freely divided on the outer side of the pelvic mesocolon so as to mobilise the adherent part of the bowel, and thus permit of its being delivered outside the abdomen.

In my earlier cases I was in the habit of dividing the pelvic colon at the seat of election as the first step in the operation. For this purpose a point in the pelvic colon was selected between the areas of distribution of the first and second sigmoidal branches of the inferior mesenteric artery. To this point an intestinal crushing clamp was applied. For some time past, however, I have left the division of the bowel to the end of the pelvic part of the operation and ligature the inferior mesenteric artery, at the seat of election, before doing anything else. The ligature is applied between the first and second sigmoidal branches.

When the pelvic mesocolon contains little fat the position of these vessels can be seen readily, and there is no difficulty in applying the ligature at the correct spot; but in obese subjects the vessels cannot be seen. Under these circumstances it is advisable to place the ligature on the inferior mesenteric artery at the level of the bifurcation of the abdominal aorta, as that point is half an inch below the origin of the first sigmoidal branch, and well above the origin of the second branch.

It is never necessary to expose the vessels by dissection, as, by so doing, somewhat free bleeding may ensue from branches of the sigmoidal veins which obscures the view and embarrasses the operator. The simplest way is to transfix the pelvic mesocolon by passing an aneurysm needle behind the inferior mesenteric vessels at the level of the bifurcation of the aorta and then to ligature the mesocolon *en masse*. When applying this ligature the position of the left ureter must be borne in mind.

At the level of the bifurcation of the aorta the ureter is from three-quarters of an inch to an inch to the left of the inferior mesenteric artery, but at the level of the promontory of the sacrum they are close together, so that if the mesocolon is transfixed too low down the ureter is in danger of being included.

Ligature of the inferior mesenteric artery, as the first step in the

operation, ensures a practically bloodless field during the subsequent steps of the pelvic portion of it, and is therefore essential. This having been done a second ligature is placed on the vessels about an inch below the first in order to control venous bleeding from the distal part of the mesocolon when it is divided.



Fig. 706.—AFTER THE INFERIOR MESENTERIC VESSELS HAVE BEEN LIGATURED, THE PERITONEUM IS INCISED FROM BEHIND FORWARDS ALONG THE BRIM OF THE PELVIS EXPOSING THE LEFT URETER AS IT CROSSES THE COMMON ILIAC VESSELS FROM WITHOUT INWARDS.

*Second Stage.* The pelvic mesocolon is divided completely immediately below the first ligature, and then the peritoneum, on either side of the line of origin of the mesocolon, is incised downwards as far as the promontory of the sacrum. When this is being done on the left side, the position of the left ureter should be carefully ascertained lest it be divided or otherwise injured (fig. 706). As soon as the peritoneum has been divided on both sides as far as the promontory, the cellular space, between the anterior surface of the sacrum and the terminal part of the pelvic mesocolon, comes into view.

By thrusting the fingers of the left hand into this space the terminal portion of the pelvic colon and the rectum can be stripped readily from the anterior surface of the sacrum as far as the sacro-coccygeal articulation. At the latter point the fascia propria of the rectum is closely adherent to the periosteum of the lower border of the sacrum and cannot be stripped away from it, thus indicating that the level of the articulation has been reached.



*Fig. 707.*—SHOWING THE RECTUM, ENCASED IN THE FASCIA PROPRIA, SEPARATED FROM THE HOLLOW OF THE SACRUM AS FAR AS THE SACRO COCCYGEAL ARTICULATION AT WHICH POINT THE FASCIA PROPRIA BLENDS WITH THE PERIOSTEUM OF THE LOWER SEGMENT OF THE SACRUM.

Occasionally one or two dense bands of connective tissue extend from the fascia propria to the sacrum. These should be divided with scissors rather than torn from their sacral attachments lest by so doing a presacral vein be lacerated and give rise to troublesome bleeding. The left hand is now introduced into the presacral space thus opened up and the rectum is pressed forwards and upwards in order to raise and render prominent the peritoneum lining the floor and the lateral walls of the pelvis (fig. 707). The pelvic peritoneum thus raised is divided forwards on either side parallel to the rim of the true pelvis as far as the base of the bladder, care being taken not to injure the ureters which



often adhere closely to the peritoneum. These incisions in the peritoneum, along the brim of the true pelvis, are continued until they meet anteriorly behind the base of the bladder in the male or the upper part of the vagina in the female.

*Third Stage.* The separation of the anterior wall of the rectum from its connections is next proceeded with. In the male, a well-marked stratum of loose areolar connective tissue exists between the fascia propria of the rectum and the layer of recto-vesical fascia which binds the vesiculæ seminales to the base of the bladder. When this line of cleavage is found the separation of the rectum from the vesiculæ seminales and the base of the bladder is easily effected, but unless care be exercised the space containing the vesiculæ may be opened up, and then the dissection becomes extremely difficult, so that the vesiculæ or the vasa deferentia may be injured. It is important that the separation of the rectum anteriorly should extend as far as the upper border of the prostate gland.

*Fourth Stage.* As soon as the rectum has been freed from its connections both anteriorly and posteriorly down to the points indicated above, the lateral attachments of the rectum can be readily made out. These consist, on either side, of a broad band of dense connective tissue, varying in depth from two to three inches, which passes forwards and outwards from the lateral walls of the rectum towards the base of the bladder at the point where the ureters terminate. These bands, the lateral ligaments of the rectum (fig. 708), must be completely divided down to the upper surfaces of the levator ani muscles. The middle hæmorrhoidal artery, which passes in the substance of the ligament to its distribution, is always divided but is seldom of sufficient size to need a ligature.

*Fifth Stage.* The rectum having thus been freed from its connections, anteriorly as far as the upper border of the prostate, posteriorly down to the level of the sacro-coccygeal articulation, and laterally to the upper surfaces of the levator ani muscles, the pelvic colon is crushed at the seat of election, that is, at a point situated about three inches from the termination of the descending colon. A broad-bladed crushing clamp (author's pattern) is applied to the colon at the point indicated, and then the crushed area is ligatured in two places. The bowel is then divided between the ligatures and the ends of the bowel protected by tying a piece of green protective over them. In

my earlier cases I invaginated the ends by means of a purse-string suture but have abandoned the practice in favour of the more speedy method of covering them with green protective.

*Sixth Stage.* The occluded end of the distal part of the pelvic colon is next pushed downwards into the presacral space until it rests at the

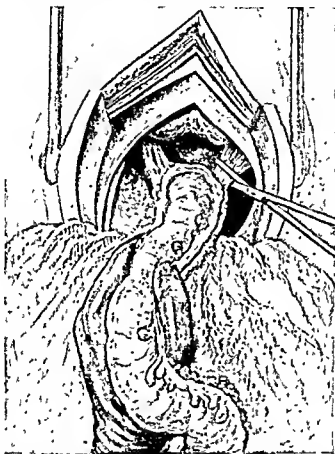


Fig. 708.—SHOWING THE SEPARATION OF THE ANTERIOR CONNECTIONS OF THE RECTUM AS FAR AS THE UPPER BORDER OF THE PROSTATE, AND DIVISION OF THE LATERAL LIGAMENTS. THE LATERAL INCISIONS IN THE PERITONEUM HAVE BEEN EXTENDED ON EITHER SIDE SO AS TO MEET ANTERIORLY BEHIND THE BASE OF THE BLADDER.

level of the sacro-coccygeal articulation where it can be easily reached when the perineal portion of the operation is being performed. The remainder of the distal part of the pelvic colon is then crowded down into the cavity of the pelvis and preparations are made for re-establishing the floor of the pelvis by peritoneum.

Owing to the free removal of the peritoneum lining the floor and the lateral walls of the pelvic cavity, a large gap remains. On no account should the pelvic mesocolon be left *in situ* with a view to facilitating the closure of the gap, because this structure is largely

concerned with the extra-mural spread of the disease, and is therefore to be regarded as highly dangerous tissue.

I cannot emphasise too strongly the necessity of completely removing this structure in every case, together with a wide strip of the adjacent peritoneum, if immunity from recurrence is to be hoped for. The gap cannot be closed by dissecting up the peritoneum from the lateral walls of the pelvis. However much the peritoneum be mobilised

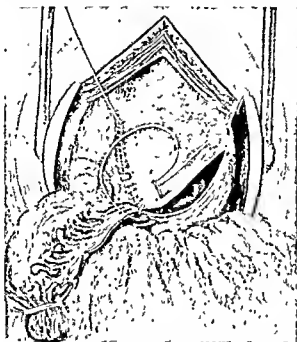


FIG. 709.—SHOWING THE METHOD OF RESTORING THE PELVIC FLOOR IN THE MALE. A FLAP OF PERITONEUM HAS BEEN DISSECTED UP FROM THE BLADDER AND DRAWN BACKWARDS UNTIL IT MEETS THE CUT EDGE OF THE PELVIC MESOCOLON, TO WHICH IT IS SUTURED.

at the sides of the gap the edges can only be approximated posteriorly in front of the promontory of the sacrum and sutured to the stump of the pelvic mesocolon.

The large pear-shaped gap remaining after this has been done can be readily filled in, however, by dissecting up the peritoneum from the base of the bladder in the male and stretching it backwards across the gap and suturing it there (fig. 709); or by dissecting up the innermost layers of the broad ligaments in the female and utilising them to fill up the space.

It is of the utmost importance that the suture line in the new peritoneal floor should be intact, and therefore, when the peritoneum is thin and likely to tear, an omental graft should be used to reinforce it.

It will be observed that the new peritoneal floor of the pelvis differs from the normal in that it is situated at a much higher level, that is, at the brim of the true pelvis. Consequently, during the healing process, a new recto-vesical pouch is formed partly by stretching of the peritoneal floor and partly by dragging down the peritoneum of the iliac fossæ. On the right side the terminal ileum is adherent to the peritoneum of the iliac fossa, and therefore a drag upon the peritoneum in this situation may produce a pronounced ileal kink. Accordingly, as soon



Fig. 710.—DISPLAYING THE ILEO CECAL ANGLE AND THE APPENDIX TO ASCERTAIN WHETHER THEY ARE FREE FROM ANGULATION AND ADHESIONS.

as the new peritoneal floor has been completed, any tendency to kinking of the ileum at the point where it enters the cæcum should be corrected (fig. 710).

*Seventh Stage.* The proximal end of the pelvic colon is now utilised for establishing a colostomy. The best position for this is at a point situated one and a half inches internally to the left anterior superior spine of the ilium, along a line extending from that bony prominence to the umbilicus. A circle of skin, one and a half inches in diameter, is excised, the centre of which is at a point at the junction of the outer and middle thirds of a line drawn from the anterior superior spine of the ilium to the umbilicus. The object of excising the circle of skin

is to prevent subsequent stenosis of the stoma. The aponeurosis of the external oblique muscle is now divided to the extent of one inch, and then the muscular fibres of the internal oblique and transversalis muscles are separated in the direction of their fibres by blunt dissection.

An opening just large enough to admit the index finger is then made through the transversalis fascia and the peritoneum. Through this small opening the stump of the proximal end of the pelvic colon is drawn, and fixed in position at the upper and lower angles of the wound by means of silkworm-gut.

I do not think there is any advantage in bringing the bowel out through the fibres of the left rectus abdominis, as is sometimes advocated in the performance of colostomy. The chief point to be borne in mind is to make the opening just large enough to allow the stump of the bowel to be drawn through it and no larger, lest herniation of small intestine between the bowel and the edge of the wound should occur.

The abdomen is now closed and a temporary dressing applied. The Trendelenburg position having been dispensed with, the patient is placed in the right dorsal and semi-prone posture in order that the perineal portion of the operation may be proceeded with.

*The Perineal Portion of the Operation. Incision.* The enus having been closed by means of a purse-string suture, a transverse incision about four inches in length is made at the level of the sacro-coccygeal

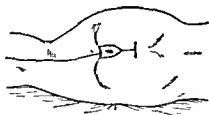


Fig. 111.—SHOWING THE OUTLINE OF THE INCISIONS IN THE PERINEUM. ONE TRANSVERSE INCISION IS AT THE LEVEL OF THE SACRO-COCYGEAL ARTICULATION AND THE OTHER IMMEDIATELY BEHIND THE BULB OF THE URETHRA.

articulation. From the centre of this a longitudinal cut is made in the inter-natal furrow, and carried down to a point one inch from the posterior margin of the anus. From the inferior extremity of this, incisions are carried to the right and to the left of the anus in the shape of a horseshoe, and the anterior extremities of these are joined by a

transverse cut (fig. 711). It is important that the arms of the horse-shoe should embrace as wide an area of peri-anal skin as possible, because the skin in this region is especially prone to develop recurrent growth. The gluteal skin flaps are then reflected and retracted out of the way, thus laying bare the coccyx.

*Removal of the Coccyx.* The sacro-coccygeal joint is opened and the coccyx dissected out; the incisions surrounding the anus are then deepened so as to include as much as possible of the ischio-rectal fat (fig. 712). It is never necessary to remove a piece of the sacrum, as

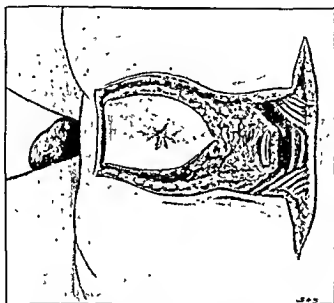


Fig 712.—SHOWING THE REFLECTION OF THE SKIN FLAPS AND THE INCISION THROUGH THE SACRO COCCYGEAL JOINT.

ample room is provided by removal of the coccyx alone. In fact, sufficient room for the completion of the operation can be obtained without even removing the coccyx; but I think it is best to remove it, because as the coccygeus muscles must be removed, the bone would be left without any lateral attachments.

*Exposure of the Presacral Cavity containing the Isolated Bowel.* A small transverse incision is made into the dense connective tissue immediately below the sacrum, where the attachment of the fascia propria recti can readily be detached from the ventral aspect of the lowermost piece of the sacrum. The index finger is then thrust into this, when, supposing that the separation of the rectum from the front of the sacrum has been carried down to the level advocated above, it

readily passes into the space containing the isolated bowel. A transverse incision is then made through the coccygeus muscle on either side, extending outwards as far as the great sacro-sciatic ligaments. Through the ample opening thus made the isolated bowel is drawn down to its full extent (fig. 713).

When the separation of the anterior connections of the rectum have been carried down to the prostate during the abdominal part of the operation, the base of the bladder and the vesiculæ seminales, with the vasa deferentia and the upper part of the prostate, come into view. In the female, the upper half of the posterior vaginal wall can be plainly seen.

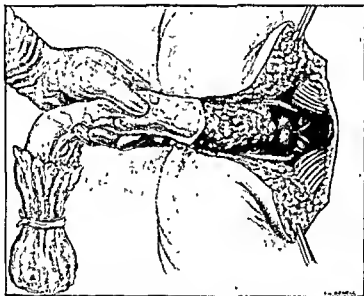


Fig. 713.—SHOWING THE PELVIC COLON AND THE ISOLATED UPPER PART OF THE RECTUM WITHDRAWN, FROM ABOVE DOWNWARDS, FROM THE CAVITY OF THE PELVIS. THE BACK OF THE BLADDER, THE VESICULÆ SEMINALES, WITH THE VASA DEFERENTIA AND THE PROSTATE, ARE READILY EXPOSED TO VIEW.

*Division of the Levatores Ani Muscles and Severance of the Remaining Connections of the Rectum.* By making traction upon the bowel with the left hand the levatores ani are put upon the stretch. If they do not come into view, it is because the lateral ligaments of the rectum have not been completely divided from above. In that case considerable difficulty may be experienced in delivering the loosened bowel through the perineal wound, and until the lateral ligaments have been completely severed the levatores cannot be divided. The levatores are now divided at their origin from the lateral wall of the pelvis, the puho-prostatic fibres being detached from the prostate.

In those instances in which the growth is situated on the anterior wall of the ampulla of the rectum, I always make a practice of dissecting away the prostatic capsule as well lest invasion of it may have commenced.

All that now remains to be done is to dissect away the anterior wall of the anal canal from the tissues forming the central point of the perineum, great care being taken not to wound the membranous portion of the urethra in so doing. It is not necessary to introduce a sound into the bladder for the purpose of indicating the position of the urethra.

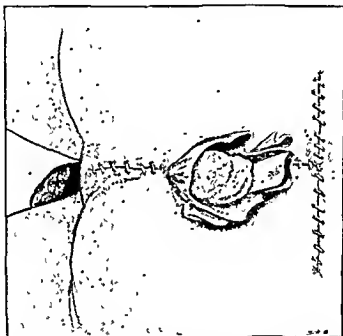


Fig. 714.—SHOWING THE PERINEAL WOUND PARTIALLY CLOSED. THE PELVIC CAVITY IS LINED BY "GREEN PROTECTIVE" INTO WHICH GAUZE PACKING IS INTRODUCED TO SUPPORT THE NEW PELVIC FLOOR CONSISTING OF PERITONEUM ONLY.

*Completion of the Operation.* After the removal of the rectum and the isolated portion of the pelvic colon, usually about sixteen inches in length, a large cavity is left. This cavity is surrounded by bony structures behind and at the sides, and it is absurd to suppose that it can be sewn up so as to obtain healing by primary intention. The cavity must heal gradually by granulation, portions only of the skin incision being brought together by sutures.

I always pack the cavity with long strips of gauze, so as to afford support to the new pelvic floor formed only by peritoneum. It is not advisable to allow the gauze to be in direct contact with the walls of the cavity, because it becomes firmly adherent to them and gives considerable trouble and pain to the patient when it is removed.



In one of my earlier cases, the peritoneum of the pelvic floor was torn when the gauze was removed, and a coil of small intestine became herniated through the opening. I always use a sheet of green protective, two feet square, for lining the cavity, and then pack the gauze into it, as shown in figure 714, the subsequent removal of the gauze thus being rendered easy and painless. Dressings and bandages are then adjusted, and the patient is turned upon his back so that the abdominal wounds may also be dressed. Before the patient leaves the table the ligatures closing the stump of the proximal end of the pelvic colon are removed, and the open end of the bowel is covered with green protective and a pad of gauze.

*Post-Operative Treatment.* Immediately after the operation has been completed a blood-transfusion (500 cubic centimetres) should be given. There is no doubt that this is the best means of combating post-operative shock. I have adopted blood-transfusion as a routine measure with the result that the mortality has fallen considerably.

As soon as the patient returns from the theatre the foot of the bed is raised and an electric radiant heat cradle is fitted over him, the temperature being carefully regulated.

Continuous subcutaneous saline is administered during the first forty-eight hours.

No fluid is given by the mouth during the first forty-eight hours so as to lessen the risk of acute gastric dilatation. Careful watch for the onset of this complication should always be kept, and as soon as the subxyphoid depression becomes effaced the stomach should be emptied by passing the stomach tube.

The perineal wound is dressed for the first time 72 hours after the operation. After the gauze packing has been removed, the cavity is irrigated with (1) hydrogen peroxide (10 vols.) 1 pint, followed by (2) perchloride of mercury (1/500) 1 pint, followed by (3) 2 pints of normal saline solution. Irrigation with these solutions is carried out twice daily during the first week, after which period a solution of iodine or of dettol (2 drams to the pint) is substituted.

On the morning of the fifth day an enema is administered by the colostomy, and on the following day a saline purgative is given. The colostomy wash-out is repeated daily.

At the end of a week the excess of bowel, forming the colostomy, is removed. Digital dilatation of the colostomy stoma is carried out daily until the tendency to contract, during the healing process, has ceased. The patient is allowed to get up for the first time on the eighteenth day for twenty to thirty minutes.

*Operability Rate of the Abdomino-perineal Operation.* As might be expected, a larger proportion of cases are operable by the abdomino-perineal route than is possible by perineal methods of excision. This is largely due to the fact that high-lying growths, at or near the recto-sigmoidal junction, are considered to be out of reach by an operation undertaken from the perineum alone. I find that, after excluding not only the cases in which the local disease is too far advanced, but those, at any stage of the disease, in which age, general condition, or low cardiac energy index, indicate had operation risks, the operability rate among the cases that come under my observation is about 35 per cent.

*Mortality of the One-Stage Abdomino-perineal Operation.* The death-rate attributable to the operation has steadily decreased in successive series of cases until at the present time it is remarkably low.

I attribute this to three main factors: (a) the employment of more suitable anaesthesia; (b) the estimation of cardiac energy when assessing operability; and (c) the adoption of blood-transfusion as a routine measure immediately after the completion of the operation.

The anaesthetic employed in the early cases was ether administered by the open method, and it was found that the patients seldom left the operating table with a pulse-rate of less than 120, clearly indicating a marked lowering of blood-pressure. This loss of blood-pressure due to the anaesthetic, when added to the fall associated with shock following the operation, was more than a great many of the patients were able to withstand, and as a consequence the mortality rate proved to be as high as 32 per cent. Although ether anaesthesia could not be held responsible for all the deaths, some of which were clearly due to other causes, there seemed to be no doubt that it accounted for the greater proportion of them.

In the next series, therefore, intrathecal stovaine with gas-and-oxygen inhalation were substituted. It was found, however, that the anaesthesia produced by stovaine did not last longer than three-quarters of an hour, sufficing only for the abdominal portion of the operation and that it was necessary to resort to ether inhalation during the perineal part of the procedure. Marked improvement resulted from the change, the mortality in this series falling to 17 per cent.

In the succeeding series in order to dispense entirely with ether, a caudal block, consisting of 30 cc. of a 2 per cent solution of novocaine introduced extrathecally into the sacral canal, was resorted to in addition to the stovaine and gas-and-oxygen. The anaesthesia of the perineum produced in this way was highly satisfactory and persisted

for three hours afterwards. More recently I have employed intrathecal percaïne (15 cc. of 1/1500 solution), which does away with the necessity of a caudal block as the anaesthesia persists for about three hours.

In my latest series, by eliminating those patients whose cardiac energy was estimated to be below 25 per cent as calculated according to the Moots-McKesson pressure-ratio percentage test, and by adopting blood-transfusion as a routine measure immediately after the completion of the operation, the mortality has been still further reduced to the neighbourhood of 10 per cent.

The marked reduction in the mortality of the operation has robbed it of most of its terrors, so that it may now be possible for those who have consistently regarded the abdomino-perineal operation, either from unfortunate experience or from timidity, as too dangerous a procedure to be undertaken, to be induced to try again.

*Recurrence Rate of the One-Stage Abdomino-perineal Operation.* It is interesting to ascertain whether the procedure has justified the hope entertained for it by a pronounced reduction of recurrence rate. For the purpose of this study I have taken the survivals of the operations performed by myself during a six-year period. It is customary, when compiling statistics concerning cancer of the rectum, to ignore the fact that there are several varieties of the disease. I think that this is a mistake because, although there is only one type—the adeno-carcinoma—there are four distinct varieties, namely, the papilliferous, the infiltrating adenoid, the colloid, and the melanotic, each of which differs considerably in the degree of malignancy displayed.

Malignancy is determined by the rapidity with which dissemination takes place in the neighbouring and outlying tissues. The papilliferous carcinoma disseminates very slowly, the infiltrating adenoid invades progressively and gives rise to distant metastases, the colloid infiltrates with great rapidity, and the melanotic growth not only disseminates by means of the lymphatics with extreme rapidity, but spreads through the vascular system as well. It is obvious, therefore, that when comparing results in regard to recurrence each variety should be considered separately. The recurrence rate for the different varieties was as follows :

<i>Variety.</i>	<i>Recurrence rate.</i>
(a) Papilliferous . . .	Nil.
(b) Adenoid . . .	14 per cent.
(c) Colloid . . .	87.5 „
(d) Melanotic . . .	100 „

It appears that the recurrence rate for colloid and melanotic growths is so high that they are not worth operating upon if their histological characteristics are known beforehand. It must be borne in mind, however, that even when a portion of a melanotic growth has been removed for microscopical examination, it may not be always possible to detect its melanotic nature, because the pigment may not be uniformly distributed. Melanotic carcinoma, however, almost invariably involves the posterior portion of the anal canal, so that growths in that situation should be regarded with suspicion.

Recent statistics compiled by my colleague, Mr. Lawrence Ahel, on a five-year basis from cases, including all varieties, operated upon at the Cancer Hospital, show a survival rate of 69.3 per cent.

*The Perineo-abdominal Operation.* This is a radical procedure as it includes the tissues of the three zones of spread and is carried out in one stage. It differs from the one-stage abdomino-perineal operation in the respect that the perineal portion of the operation is done first and then the isolated rectum and pelvic colon is delivered through the abdomen. It seems that the die-hard adherents of the perineal method of excision have become dissatisfied with the recurrence rate and have extended the operation field upwards into the abdomen so as to include the tissues of the upward zone of spread which had been left behind by the less radical procedure.

An excellent account of the technique of the operation is given by Mr. Gabriel in his book, to which the reader is referred. (*The Principles and Practice of Rectal Surgery*—H. K. Lewis, 1932.)

#### TREATMENT OF INOPERABLE CASES

It is unfortunately a common experience that a very large proportion of the cases of cancer of the rectum that come under the notice of the surgeon have passed beyond the stage when an operation for removal of the disease can be undertaken. In this class of case, if the disease is allowed to pursue an unhindered course, obstruction of the lumen of the bowel sooner or later occurs, either from exuberance of the growth, invagination of the affected segment of the bowel, or fixation of the rectum to neighbouring structures.

The surgical treatment of inoperable cancer of the rectum, therefore, resolves itself either into adopting measures for the relief of existing intestinal obstruction or for preventing its onset.

When obstruction exists, an effort first of all should be made to

relieve the obstruction by encrusta, as it may have been caused by impaction of a hard mass of feces in the stenosed area of the bowel. For this purpose injections of olive oil are extremely useful. Should, however, the obstruction remain unrelieved, an opening into the colon above the seat of the obstruction must be established without delay.

In those cases in which the abdomen is considerably distended, and especially when the attack of obstruction has supervened after previous abortive attacks, the operation of cæcostomy should be performed for the purpose of emptying the colon of its contents, and then, at a later date, left iliac colostomy can be established with subsequent closure of the cæcostomy. If left iliac colostomy be primarily attempted in cases of gradually supervening obstruction, both the descending colon and the sigmoid are often so distended by fecal material that it is impossible to bring the bowel up to the surface to permit of an opening being made into it with safety.

When the stage of obstruction has not been reached the operation of choice, for preventing its onset, is left iliac colostomy. We have seen that the natural termination of cancer of the rectum is an attack of intestinal obstruction, and that the means at our command for obviating obstruction is the establishment of colostomy. Colostomy, therefore, is inevitable, and the question arises as to whether it should be postponed until a later time. The matter is one which calls for careful consideration.

*How Soon should Colostomy be Performed in Cases of Inoperable Cancer of the Rectum?* By an inoperable case is meant not only one in which the condition is so advanced that complete removal of the disease is impossible, but also one in which, even though the growth be comparatively restricted, the age of the patient or his unfavourable general condition renders a radical operation too great a risk to life. Under the circumstances we are often called upon to decide whether the operation of colostomy should be deferred until the patient experiences pronounced difficulty in evacuating the contents of the bowel.

The matter is not easily disposed of, for not only the patients themselves but also their medical advisers as well regard colostomy as a calamity which should be postponed as long as possible. The argument usually advanced is that there will be time enough to resort to it when life is in danger from an impending attack of absolute obstruction. Such a contention as this would be reasonable if an attack of absolute obstruction supervened at a definitely known period during the course of the disease, and if the premonitory symptoms were always

gradual in development. Unfortunately, however, in some cases the attack occurs rapidly and without warning, though in others its advent may be long delayed. Moreover, in the interim, be it short or long, many complications may arise during the progress of the disease which materially increase the suffering of the patient and which a timely colostomy would greatly relieve or entirely obviate.

Since the site of the growth in the rectum has considerable influence both on the time of onset of absolute obstruction and upon the nature of the other complications, it is necessary that we should consider whether colostomy ought to be performed early or late in cases of cancer situated (a) at the recto-sigmoidal junction, (b) in the ampulla, and (c) in the anal canal.

(a) *Growths situated at the Recto-Sigmoidal Junction.* When carcinoma occurs at the recto-sigmoidal junction it conforms to the type generally met with in the colon, in that it spreads rapidly round the whole of the circumference of the bowel and produces stenosis. In these cases the earliest symptom to attract attention is increasing difficulty in the movement of the bowels. Alternating attacks of constipation and diarrhœa, which herald the early onset of absolute obstruction, soon make their appearance.

When such a case, for some reason or other, is found to be inoperable, should colostomy be done at once, or should it be deferred until it becomes absolutely necessary to avert death from complete obstruction? The answer is to be found in the records of our experience of the complications that may arise during the progress of these cases before the onset of obstruction. In the first place, diarrhœa may be so persistent as to necessitate relief of the bowels many times during the day and night. The patient suffers great distress and rapidly loses ground from having his rest disturbed.

Quite recently I saw a patient with a growth in the lower part of the pelvic colon which caused the most persistent diarrhœa. During the previous twenty-four hours he had had twenty-three actions of the bowels. As an operation for removal of the growth was entirely out of the question, I advised immediate colostomy. He refused to submit to it and died a month afterwards. I subsequently learned that on seeking further advice, he was told that colostomy was unnecessary as obstruction did not exist. I have no doubt whatever that had colostomy been performed when I suggested it, the diarrhœa would have ceased, the patient would have lived longer than he did, and the remaining days of his life would have been free from much misery. Again, a growth

of this type, besides causing stenosis, may penetrate the wall of the bowel and produce a weak spot which gives way under the strain of violent peristalsis.

Lastly, an attack of absolute obstruction may be precipitated suddenly and without previous warning, either from the stenosed segment of the bowel becoming intussuscepted into the lumen of the bowel below, or from a large mass of faeces having become impacted above the growth. In either case colostomy must be performed under the most disadvantageous circumstances, and, in some instances, may not be possible at all owing to distension of the colon with solid faeces.

During the progress of carcinoma at the recto-sigmoidal junction it is not possible to predict when the complications, to which I have alluded, may arise; and therefore much the safer plan is to perform colostomy as a preventive measure at the earliest possible moment.

(b) *Growths situated in the Ampulla of the Rectum.* Owing to the large diameter of the bowel in this situation an attack of absolute obstruction does not take place until the disease has reached a very advanced stage, and, indeed, in a considerable number of cases it does not occur at all. Hence, if colostomy is postponed until the signs of obstruction are impending, the patient will have endured much misery from various complications which earlier intervention would have spared him.

It is not only in advanced cases, however, that the expediency of early colostomy has to be considered. It may happen that, owing to an unfavourable general condition of a patient, a carcinoma in quite an early phase of development cannot be removed.

Let us take the case of a growth, for example, that has involved one half of the circumference of the bowel. At this period the process is not sufficiently extensive to cause much narrowing of the lumen of the bowel, and the risk of the occurrence of an attack of absolute obstruction is very slight; the bowels act freely every day and there is no evidence of abdominal distension. Still, by the time a growth has advanced to this stage surface disintegration is well marked and a state of active ulceration has been reached. Submucous suppuration is apt to occur in the vicinity of the margin of the ulcer, causing the patient considerable discomfort. He complains of pain in the rectum itself and over the lower part of the sacrum, and is troubled with severe tenesmus at each action of the bowels. The ulcerative process is also accelerated by the frequent passage of faecal material over it, and the growth often becomes covered with sloughing tissue. When these

sloughs separate, free and copious hæmorrhage occurs. Such attacks of bleeding, when repeated, soon sap the patient's vitality.

By the time, too, that a growth has encompassed half of the circumference of the howel, penetration of the whole thickness of its coats has probahly occurred, and leakage into the peri-rectal tissues takes place. This leads to the formation of pus in the pelvi-rectal space. Such abscesses usually evacuate themselves into the rectum, but sometimes they invade the ischio-rectal fossæ, not infrequently terminating in fistulæ with numerous external openings in the perineum from which pus continually escapes. These fistulæ require repeated operation, but seldom heal, and often become the seat of fungating growth, which adds immensely to the misery of the patient. The pain and the repeated hæmorrhages during this stage of an ampullary carcinoma are practically abolished, and the formation of abscess and fistula is entirely prevented by early colostomy.

Even during a later stage, when the growth has entirely encircled the howel, an attack of absolute obstruction may not occur. By this time the carcinoma has extended into the peri-rectal tissues, has invaded neighbouring structures, and has involved the sacral plexus of nerves. The condition of the patient is now truly miserahle, and colostomy if now performed holds out little prospect of relief. Moreover, though actual obstruction may not exist, the pelvic colon is probahly so distended with fæcal material that it is impossible to withdraw a loop of gut from the abdomen in order to obtain an efficient spur.

(c) *Growths involving the Anal Canal.* A carcinoma in this situation is from the outset accompanied by severe pain at every action of the bowels. This is due partly to greater sensitiveness of the mucosa of the anal canal, and partly to spasm of the sphincter and levator ani muscles. It is true that this pain can be relieved considerably by the operation of linear proctotomy, but in this case the misery of incontinence is substituted. The growth, too, very rapidly penetrates the coats of the bowel when ischio-rectal suppuration with external fistulæ results. Colostomy in these cases affords complete relief, especially if combined with linear proctotomy.

Thus, when a case of cancer of the rectum, in whatever position it may occur, is considered to be inoperahle, the sooner colostomy is performed the better it will be for the patient. By so doing he is saved much unnecessary suffering from the various complications that may arise before an attack of obstruction supervenes and renders the



operation necessary. From what I have seen of these cases I am convinced that, as soon as carcinoma of the rectum is found to be inoperable, every day lost before resorting to colostomy is a day to the bad.

#### FURTHER TREATMENT OF CASES UNSUITABLE FOR REMOVAL OF THE PRIMARY GROWTH

This consists in treating palliatively the various symptoms as they arise during the later stages of the disease. The operations of colostomy and cæcostomy are efficacious purely from the point of view that they prevent the occurrence or recurrence of obstruction and also the distressing symptoms due to the passage of faeces through the diseased rectum. The growth itself, however, pursues its course, and sooner or later extends by continuity of tissue into adjacent structures and gives rise to metastatic deposits in distant parts, notably the aortic lymphatic glands and the liver. The symptoms due to such extension are not relieved by an existing colostomy and accordingly call for treatment. These symptoms are: (a) an increased secretion of the rectal mucus; (b) hæmorrhage; (c) pain due to pressure upon or involvement of nerves of the sacral plexus; (d) pelvi-rectal and ischio-rectal suppuration; (e) recto-vesical and recto-vaginal fistula; and (f) ascites.

(a) *Increased Secretion of Rectal Mucus.* This accumulates in the rectum and causes frequent desire for an evacuation, often disturbing the patient's rest at night. I have used injections of a solution of alum (10 grs. to 1 dram) with benefit, the excessive secretion having been checked considerably for a time. Should it continue, the best treatment is to provide free drainage for the mucus by dividing both the external and the internal sphincter muscles (linear proctotomy). After so doing, it is true that there is a constant discharge from the anus, necessitating the patient wearing a pad of absorbent dressing, but at the same time he is not disturbed at night.

(b) *Hæmorrhage.* This usually emanates from blood-vessels of new formation in the growth itself which have been eroded by the ulcerative process. At first the application of solutions of adrenalin chloride, hemiscine, or perchloride of iron may check the bleeding, but later on, the best plan is to scrape away with a Volkmann's spoon all the soft portions of the growth. This method is efficacious in preventing further losses of blood for several weeks. Should a large venous trunk or

artery, such as the internal iliae, be involved by the ulcerative process, an extremely rare result, the patient's life is speedily terminated by profuse hæmorrhage.

(c) *Pain Due to Pressure Upon or Involvement of Nerves of the Sacral Plexus.* The onset of pain during the later stages of the disease is quite distinct from that produced by the passage of fæces through the diseased rectum, and is due to the extension of the growth into the peri-rectal structures. Scraping away the growth relieves the pain for a time, when due to pressure upon the nerves in the pelvis, but when the nerves themselves are involved in the growth little can be done beyond affording respite from suffering by the judicious use of morphia. Commencing with small doses of one-sixth grain every eight hours, the quantity of the drug may be increased until as much as two or three grains every three or four hours has been reached. The combination of cocaine with morphia for internal administration is especially serviceable in the treatment of the later stages of carcinoma. In prescribing preparations of opium in the treatment of these cases, the increase in the strength and the frequency of the dose should be made as slowly as possible, because patients so quickly become tolerant of large doses which soon impair their digestion and general health.

(d) *Pelvi-rectal and Ischio-rectal Suppuration.* The indication that this has taken place is generally an escape of pus from the anal orifice. A careful examination, under these circumstances, should be made for the purpose of locating the collection of pus, which, when found, should be freely evacuated. If the pus has already reached the surface, the resulting fistula should be thoroughly laid open, at least externally. In those cases in which a growth involving the lower portion of the rectum has given rise to fistula, the best way of relieving the patient is to excise the lower portion of the rectum together with as much of the growth as possible.

(e) *Recto-vesical or Recto-vaginal Fistula.* This is due to the direct extension of the growth into the bladder and vagina respectively. In those instances in which colostomy has not been performed the condition of the patient is most distressing, because both flatus and fæces, and sometimes blood, pass into the bladder on the one hand, causing intense cystitis, and on the other they escape into the vagina, producing incontinence of both flatus and fæces. In such cases the best treatment consists in performing colostomy as soon as possible. When colostomy

has already been performed, blood and discharge are all that escape into the bladder and vagina and are best dealt with by means of frequent irrigation.

(f) *Ascites*. The onset of ascites generally shows that the liver has become infected by secondary deposits. When the quantity of fluid is large enough to produce pressure symptoms, much temporary relief can be afforded by repeated tapplings.

PART IV  
X-RAY DIAGNOSIS

by  
H. CECIL BULL

SECTION 1  
RADIOLOGY OF THE ALIMENTARY TRACT

SECTION 2  
RADIOLOGY OF THE URINARY TRACT

## SECTION I

# RADIOLOGY OF THE ALIMENTARY TRACT

## I. THE STOMACH

THE stomach itself is invisible to X-rays ; but filled with a radio-opaque substance—barium sulphate—a shadow outline of the interior is seen. The stomach is an organ of volume in constant motion, and each film shows it only in a certain projection at a certain phase. It is not yet possible to take cinematograph films for long enough periods, but we can and do examine and palpate the stomach under the fluoroscopic screen, which is the most important part of an X-ray examination. The films taken are but records of what the screen examination revealed. Films without preliminary screen examination are inaccurate, whereas the combination of the two has a greater diagnostic accuracy than any other single procedure.

### THE NORMAL STOMACH

For any interpretation or understanding of the pathological it is essential first to recognise the normal. No single description can be given of the normal stomach since in size, shape and position, tone, mobility, etc., individual stomachs vary ; they may differ widely in form and functional activity but still be normal for the individuals to whom they belong. Stomachs do in fact vary with the build and temperament of the individual, and they may therefore be divided for the purposes of classification into three types : two extreme and dissimilar types—*hypersthenic* and *hyposthenic*—and a *common average* partaking of the characters of both and representing the majority of mankind.

(1) "*Hypersthenic*" is the polite term for "fat" : the chest of the hypersthenic patient is short, and the abdomen relatively long and fat. The stomach is high in position, lying more or less transversely

across the abdomen, so that it has been called "steer-horn." The fundus presents somewhat forwards, and the first part of the duodenum—called the duodenal cap—lies behind the pylorus.

The hypersthenic stomach is also hypertonic—or at least of good tone; it therefore seems small in comparison with the hypotonic stomach of the hyposthenic since it contracts round the meal which fills it: peristalsis is active and begins directly food enters the stomach; barium pours out freely and rapidly through the pylorus (fig. 715).

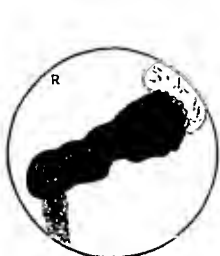


Fig. 715.—"STEER HORN" STOMACH—THE HIGH STOMACH OF THE HYPERSTHENIC. TONE IS GOOD, PERISTALSIS SHALLOW, THE DUODENAL CAP LIES BEHIND THE PYLORIC END OF THE STOMACH.

(From Bull's "X ray Interpretation")

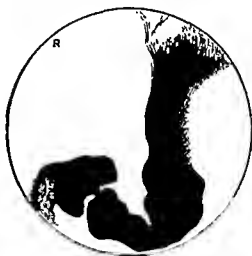


Fig. 716.—STOMACH OF THE "HYPOSTHENIC." LOW IN POSITION AND POOR IN TONE THIS STOMACH APPEARS LARGE. THE DUODENAL CAP IS ALSO LARGE AND HYPOTONIC.

(2) "*Hyposthenic*" implies visceroptosis: the stomach is large in size, the fundus low in position—dipping down into the pelvis—tone is poor, barium pools in the fundus of the hyposthenic stomach, and peristalsis begins slowly; the emptying time of these stomachs is therefore longer than that of the hypersthenic (fig. 716).

(3) *The common average* is somewhere between these two extremes; the stomach is of J shape, lying in the mid-abdomen, and showing a discreetly moderate tone, peristalsis and resulting emptying time. This is the commonest, and therefore perhaps regarded as the most normal type, but it is, in fact, only normal for the individual just as the hyper- and hypo-sthenic stomachs are normal for individuals of their own build. To find a large hypotonic stomach in a hypersthenic patient, or a small contracted stomach in a hyposthenic, is primary evidence of pathology.

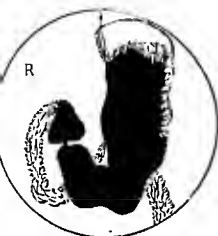


Fig. 717.—NORMAL STOMACH OF THE AVERAGE TYPE. NOTE THE GAS-BUBBLE, GREATER AND LESSER CURVATURES, PYLORIC CANAL, DUODENAL CAP AND SECOND AND THIRD PARTS OF THE DUODENUM.

(From Bull's "X-ray Interpretation.")



Fig. 718.—THE RUGAE OF THE STOMACH AND DUODENUM SEEN COATED WITH A THIN LAYER OF BARIUM.



Fig. 719.—RUGAE OF THE PYLORIC END OF THE STOMACH SHOWN BY PRESSURE.

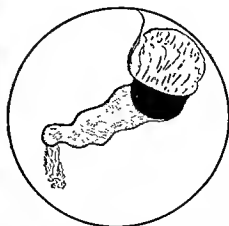


Fig. 720.—CASCADE SPASM—POSTERO-ANTERIOR VIEW. THE LOWER PART OF THE FUNDUS IS DRAWN UP BY CONTRACTION OF THE LONGITUDINAL FIBRES—FORMING A POUCH.

(From Bull's "X-ray Interpretation.")

Other points in the X-ray appearance of the normal stomach are the quantity of *secretion* and the pattern of the folds of *mucous membrane*. Finally there is the irregular muscular contraction—*spasm*—which, although not a feature of the normal, does not necessarily indicate organic disease in the stomach.

### *Secretion.*

The fasting stomach contains so little secretion that it passes unnoticed when the stomach is filled with *barium*, but a large quantity of fluid when examined after a 12-hour fast is either due to organic obstruction, or is a reflex delay from peritoneal irritation, or a central effect from a general toxæmia; the mildest and commonest cause of this form of delayed emptying is headache.

### *Mucous Membrane.*

The thick mucous membrane is thrown into more or less longitudinal folds to meet the changing volume of the stomach, and the pattern which these mucous membrane *rugæ* make is valuable as supplementary evidence of the normal or of the pathological. Their value is supplementary; in any large organic change in the stomach, deformity of the mucosal lines is inevitable and is clearly seen in films taken with suitable technique, but in a very small lesion—such as the small gastric ulcer with little or no surrounding inflammation the parallel lines are not appreciably deformed. The little spot of *barium* held in the base of the ulcer crater is thus the determining point in diagnosis.

### *Spasm.*

Rhythmic peristaltic waves are the normal muscular contractions of the stomach, but under the stimulus of inflammation affecting the peritoneum—whether of the stomach or elsewhere in the abdomen—the stomach may develop regional contractions which are difficult to distinguish from organic disease, and which indeed are the principal causes of difficulty in diagnosis.

There are three forms of spasm affecting the stomach:

- (1) Cascade spasm.
- (2) Spastic incisura, or “hour-glass” stomach.
- (3) Spasm of the pyloric end of the stomach.



(1) *Cascade spasm*. This is a spasm of the longitudinal muscle-fibres causing the fundus to be drawn up in front of the cardiac end, so that in the erect position the stomach is in two loculi—the upper one filling and spilling or “cascading” into the lower (fig. 720).

It is usually caused by irritation outside the stomach, often by distension of the colon—from whatever cause: less commonly it is reflex—from gastric or duodenal ulcer.

(2) *Spastic incisura* nearly always indicates gastric ulcer; it is a localised indrawing of the greater curvature towards the lesser, and points like a finger to the ulcer crater (fig. 721). When an ulcer is healing and the crater no longer visible, the incisura persists, and it is then



Fig. 721.—SPASTIC INCISURA—CONTRACTION OF THE CIRCULAR FIBRES—A COMMON ACCOMPANIMENT OF GASTRIC ULCER BUT SOMETIMES REFLEX FROM AN EXTRA GASTRIC LESION.  
(From Bull's "X-ray Interpretation.")



Fig. 722.—SPASM OF THE PYLORIC END OF THE STOMACH—A CIRCULAR SPASM WHICH SEEMS TO PINCH OFF THE END OF THE STOMACH—THIS CAN BE DUE TO PYLORIC ULCER OR TO EXTRA-GASTRIC IRRITATION.  
(From Bull's "X-ray Interpretation.")

impossible—without the evidence of the history—to say whether the spasm is due to a healing ulcer or is reflex from some extra-gastric cause. Like the other forms of spasm an incisura may be reflex from outside, but of the three forms it is the one most associated with inflammatory disease in the stomach, i.e. gastric ulcer.

(3) *Spasm of the pyloric end of the stomach*. This is the commonest spasm and, from the X-ray point of view, the most confusing since it can closely mimic the filling-defect of cancer, and since it may also be the only manifestation of ulcer at the pyloric end of the stomach. It is seen thus with pyloric ulcer, or as a result of direct irritation from without, such as inflammatory adhesions. It may also be a reflex spasm from peritoneal irritation elsewhere in the abdomen.

Pyloric spasm differs from the other forms of gastric spasm in that all the muscle groups—longitudinal, oblique and circular—are affected, so that this part of the stomach becomes a narrow tube of irregular outline from which barium is for the most part expressed (fig. 722). The spasm may so distort the rugæ of the mucous membrane that this is of no help in differential diagnosis, but the absence of a palpable tumour at screen examination favours spasm. However, a pyloric ulcer with adhesions and inflammatory induration can cause a palpable tumour. The most important and, indeed, the only certain point of differentiation is that spasm due to ulcer relaxes to some extent after a period of medical treatment. Such intense and fixed spasm of the pyloric segment is invariably due to ulcer, but spasm of varying degree in this area is the most common manifestation of reflex irritation from some other intra-abdominal organ.

#### FILMS

Films of the stomach are taken with the patient standing or lying prone; the prone position shows the best uniform filling of the stomach and is the one most commonly used; the erect position is good for showing the hypertonic stomach, particularly when the duodenal cap is behind the pylorus.

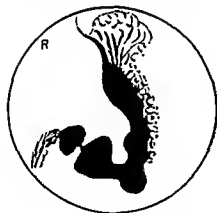


Fig. 723.—PRESSURE FROM THE COLON ON THE GREATER CURVATURE EMPHASISING THE CURVED LINES OF THE RUGÆ. NOTE THE CLEAR CUT LINE OF THE LESSER CURVATURE AND ANTRUM (From Bull's "X ray Interpretation.")

There are certain constant features about the shadow of the barium-filled stomach. The top of the shadow is irregular in outline and uneven in density owing to froth and gas-bubble. The lesser curvature and the pyloric end of the stomach are more or less clear-cut curving lines since the weight of the stomach contents is directed inwards and downwards in the prone position. On the other hand, the greater curvature is always

irregular and shows the curving folds of the mucous membrane rugæ; this is because the stomach contents tend to fall inwards away from the greater curvature, an appearance which is much exaggerated if the descending colon is distended with gas and pressing upon the stomach (fig. 723).

## DISEASES OF THE STOMACH

## 1. DIVERTICULUM

There is only one diverticulum from the stomach, and this is, in fact, a diverticulum from the epicardial portion of the œsophagus. It is seen projecting from the lesser curvature side of the cardiac end of the stomach—high in position, so that actually it appears to be a diverticulum from the gas-bubble.



*Fig. 724.*—DIVERTICULUM FROM THE EPICARDIA.

These diverticula are not common, they are usually small and do not give rise to symptoms; they do, however, retain barium after the stomach is empty.

The X-ray appearance is that of a smooth, evenly rounded projection in which barium pools at the bottom and shows a bubble of gas above (fig. 724).

They are distinguished from the somewhat similar projection of gastric ulcer by their high position—ulcers do not occur so high—their smooth outline, and the absence of spasm.

## 2. ULCER

A gastric ulcer burrows into or erodes the mucous membrane, and the little niche so formed fills with barium and shows as a projecting bud from the gastric outline. This is the direct sign of gastric ulcer (fig. 725).

## (a) ULCERS ABOVE THE INCISURA ANGULARIS

The great majority of gastric ulcers—95 per cent—occur on the lesser curvature of the stomach between the cardiac opening and the incisura angularis—an imaginary point where the vertical portion of the stomach changes direction and becomes horizontal or turns upwards. Such ulcers are usually easy to see and can be reproduced on X-ray films, provided that the rays pass tangentially to the curvature of the stomach on which the ulcer lies. Since this may be more on the posterior wall—or sometimes more on the anterior wall—than on the lesser curvature, and since gastric ulcers may be very small, they can be missed on films unless a preliminary screen examination has been made and the position of the ulcer determined.



Fig. 725.—GASTRIC ULCER ON THE LESSER CURVATURE SHOWING THE PROJECTING BUD AND THE SPASTIC INDRAWING OF THE GREATER CURVATURE.

(From Bull's "X-ray Interpretation.")

Two types of gastric ulcer which are most easily missed—because of their small size and the slight deformity they cause in the gastric outline—are the "slit" type of ulcer, a very narrow slit-like projection from the lesser curvature, and the flat "erosion" type of ulcer. Each of these makes a small and localised projection from the lesser curvature, and neither shows associated gastric spasm.

## (b) ULCERS BELOW THE INCISURA ANGULARIS

The ulcers which occur in the pyloric portion of the stomach, that is to say below the incisura angularis, are few in number, amounting to 5 per cent or less. They occur either on the lesser curvature and show as projection buds similar to those described above, or they occur on the anterior wall and cause intense spasm of the pyloric segment of the stomach (fig. 727).



*Fig. 726.*—PYLORIC ULCER, SHOWING SMALL BARIUM FILLED CRATER.



*Fig. 727.*—FILLING-DEFECT IN THE PYLORIC END OF THE STOMACH WITH PALPABLE TUMOUR SIMULATING CANCER BUT DUE TO AN ULCER WITH MUCH INFLAMMATORY INDURATION AND ADHESIONS. MICROSCOPIC SECTION SHOWED PEPTIC ULCER.

The niche type of ulcer below the incisura angularis is uncommon—less than 1 per cent—the commoner type being the ulcer on the anterior wall which shows no niche, but causes pyloric spasm.

In this latter type of ulcer, even when we X-ray the stomach with a thin layer of barium under pressure to show the outline of the mucous membrane, we are not always able to show the direct sign of an ulcer crater; we have then to rely on indirect signs.



#### INDIRECT SIGNS OF GASTRIC ULCER

(1) *Spasm.* Spasm accompanies an active gastric ulcer. With a gastric ulcer above the incisura there is almost invariably a spastic contraction of the greater curvature, drawing it towards the ulcer; sometimes this indrawing of the greater curvature is narrow and very local, so that it seems to point like a finger to the crater of the ulcer (fig. 725). At other times with a larger ulcer and considerable inflammatory reaction, the spasm may be so intense as to divide the stomach into two parts—the so-called “hour-glass” stomach. Such hour-glass deformity is entirely spastic, it does not relax with anti-spasmodic drugs, but does relax under anæsthesia (fig. 728).

The ulcers of the pyloric segment give rise to intense and fixed total spasm of this area of the stomach. At screen examination no relaxation can be obtained by massage, neither will the spasm relax with anti-spasmodics; barium can be forced past the spastic area and into the duodenum, but there is invariably delay in emptying of the stomach. The only effective way of relaxing—even partially—this spasm is by treating the patient in bed with milk and alkalis.

(2) *Alterations in contraction of mucous membrane folds.* Inflammation of the gastric mucous membrane leads to localised contraction around the site of an ulcer, and the rugæ of the gastric mucous membrane, which normally run in wavy lines along the axis of the stomach, are deflected and drawn towards the ulcer (fig. 729).

In the same way the rugæ of the mucous membrane are distorted by the spasm of an ulcer in the pyloric end of the stomach, but here the spasm is more general, involving all the muscle groups, and the course of the lines of the mucous membrane rugæ is so totally distorted that it is not often one can recognise rugæ deflected to a localised point, nor can a localised point always be demonstrated as a barium-filled crater.

(3) *Delayed emptying time.* Possibly due to inhibition of the normal neuro-muscular rhythm, the stomach with a gastric ulcer

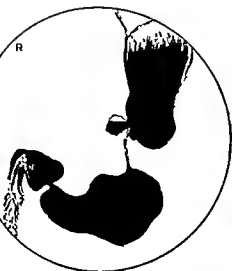


Fig. 728.—LARGE ULCER PERFORATED INTO THE LIVER FORMING AN ABSCESS CAVITY. BARIUM POOLS IN BOTTOM OF THE CAVITY, THE TOP CONTAINS GAS. THE INTENSE SPASTIC CONTRACTION OF THE GUT BELOW THE ULCER—SPASTIC HOUR-GLASS.



Fig. 729.—GASTRIC ULCER ON THE LESSER CURVATURE, SHOWING THE LINEAR RUGA DEFLECTED BY INFLAMMATORY CONTRACTION TOWARDS THE CRATER.



Fig. 730.—DOUBLE GASTRIC ULCER: CONFIRMED BY OPERATION.

usually shows some delay in emptying. In ulcers above the incisura the delay is slight—unless the area of inflammation is large and the “hour-glass” spasm extreme.

Ulcers below the incisura which show the total spasm of the pyloric segment of the stomach usually have an emptying time of over six hours.

### (c) INACTIVE GASTRIC ULCER

The visible crater of a gastric ulcer will disappear entirely in a few weeks under successful medical treatment, the last sign to persist being spasm—a certain amount of indrawing of the greater curvature remaining for a period after the niche is no longer visible. The persistence of this spastic contraction probably indicates that the lesion is not yet inactive.

X-ray examination at intervals, therefore, affords a valuable guide to the progress of a gastric ulcer under treatment. The same is unfortunately not true of duodenal ulcers, the deformity persisting during the inactive period.

The fact that the visible crater of a gastric ulcer disappears temporarily under treatment should be remembered in X-raying the stomach of a patient who has been under treatment before coming for examination.

### (d) MULTIPLE ULCERS

Gastric ulcers are usually single; it is exceptional to find two independent peptic ulcers in the same stomach. Gastric and duodenal ulcers are occasionally seen together, but this also is rare. Duodenal ulcers occur primarily and with greater frequency on the anterior wall of the duodenum; contact ulcers on the posterior wall are not uncommon, but they are found by examination of the duodenum at operation—the double location of the ulcers not being recognised by X-ray examination.

## 3. CANCER

The X-ray picture of cancer of the stomach is the converse of that of ulcer; cancer causes a “filling-defect,” or encroachment into the gastric outline, whereas ulcer is an extension of the gastric shadow into an inflammatory pocket. The different appearances are just those which would be expected from consideration of the pathology of the two diseases.



Cancer of the stomach may be divided into three types :

- (a) *Localised polypoid, cauliflower, or encephaloid growth.*
- (b) *Carcinomatous ulcer.*
- (c) *Diffuse infiltrating, scirrhus, or "leather-bottle."*

(a) POLYPOID GROWTH

This is the commonest form and the one which shows most typically the "filling-defect" in the barium shadow. Rather more than half the

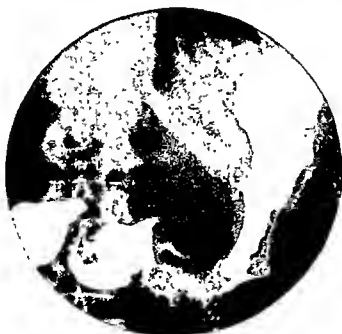


Fig. 731.—EXTENSIVE CARCINOMA OF THE BODY OF THE STOMACH.  
THE FILM SHOWS—GROSS FILLING-DEFECT, DIMINISHED CAPACITY,  
OBLITERATION OF FOLDS OF MUCOUS MEMBRANE

gastric cancers arise from the pyloric end of the stomach ; they tend to infiltrate the gastric walls so that the pylorus becomes rigid and patent, and barium passes freely through into the duodenum (see fig. 735). Less commonly—in encephaloid growths near the pylorus—the cauliflower projections of growth actually obstruct the pylorus and cause delayed emptying of the stomach.

The X-ray film of the barium-filled stomach shows a constant filling-defect in the stomach shadow ; the characteristic of this filling-defect is its constant form and outline in all positions. Other causes of filling-defects are intense spasm, and pressure from outside due to an extra-gastric tumour or to anatomical projections such as the

spine, ribs, or pelvis. Extra-gastric pressure is usually determined without difficulty at the screen examination, but fixed spasm—as from a gastric ulcer on the anterior wall of the pyloric end—may mimic cancer so closely that the diagnosis cannot be made with certainty without re-examination after a period of treatment (fig. 727).

The filling-defect of cancer is irregular in form and fades off gradually into the normal outline. When the rays pass tangentially to the growth the appearance is that of a piece bitten out of the stomach (fig. 731), and where the rays hit the growth full face there are multiple



Fig. 732.—CARCINOMA OF THE CARDIAC END OF THE STOMACH. THERE IS SLIGHT DELAY AT THE ESOPHAGEAL OPENING, BARIUM SPLASHES OVER THE GROWTH AND THERE IS AN IRREGULAR FILLING-DEFECT ON THE LESSER CURVATURE.

(From Bull's "X-ray Interpretation.")



Fig. 733.—THE "MENISCUS" TYPE OF CARCINOMATOUS ULCER ON THE LESSER CURVATURE. BARIUM IS HELD UP BY THE CRATER, AND THE THICK MALIGNANT EDGE OF THE ULCER MAKES A SEMILUNAR FILLING DEFECT.

(From Bull's "X-ray Interpretation.")

projections through the barium shadow, like coral at low tide. A characteristic form of uneven density which cancer shows through the gastric shadow is the "finger-print" filling-defect (see fig. 209).

Finally, the stomach affected with cancer is smaller than normal unless there is pyloric obstruction, and even then it never reaches the enormous size seen with cicatrising duodenal ulcer.

#### (b) CARCINOMATOUS ULCER

Whether a malignant ulcer is malignant from the beginning, or whether carcinoma develops on the site of a chronic gastric ulcer, is a question that cannot be answered on radiological evidence; there is evidence to support both views and perhaps both are correct.

From the X-ray point of view there are certain gastric ulcers which by their size alone are probably malignant, and there are ulcers which from other radiological features are almost definitely malignant.

Although there is nothing in the size to indicate malignancy, experience has shown that a large ulcer—anything from an inch or more in length—is frequently malignant on microscopic section. A large ulcer, therefore, may be regarded as potentially malignant.

Irregularity of outline is not necessarily an indication of malignancy, but the converse—a smooth outline—is not malignant. The little filling-defect in the gastric outline above and below the ulcer crater due to thickened rolled edge of an ulcer is suggestive of carcinoma.

The ulcer which is invariably malignant is the ulcer which holds up barium under its rolled edges as it lies across the lesser curvature. This sign of carcinomatous ulcer was first described by Carman (*Journ. Am. Med. Assn.*, p. 990, Vol. 77, 1921), and named by him the “*meniscus sign*” (fig. 733).



Fig. 734.—“LEATHER-BOTTLE” TYPE OF CARCINOMA. TOTAL INFILTRATION OF THE GASTRIC WALLS REDUCING THE CAPACITY TO A FEW OUNCES. BARIUM REGURGITATES INTO THE ESOPHAGUS AND POURS FREELY THROUGH THE PYLORUS.

### (c) “LEATHER-BOTTLE” STOMACH

Uniform infiltration of the walls of the stomach with cancer leads to a small contracted stomach with rigid walls and having a rapid outflow through the pylorus. The hard wall of the stomach can be palpated under the screen. The X-ray film shows the small stomach of limited capacity. It is, in fact, a total filling-defect, but there is none of the local filling-defect characteristic of the commoner form of

cancer and little irregularity of the gastric outline (fig. 734). It is the third and least common variety of cancer of the stomach.

#### DIRECT SIGNS OF CANCER

The "filling-defect," when associated with a palpable tumour, is the direct sign of cancer. This is imitated only by the large pyloric ulcer, with adhesions and induration causing a palpable tumour—which is uncommon.

Filling-defects can be caused by tumours outside and pressing upon the stomach, but the extra-gastric nature of these filling-defects is recognised at the screen examination.

#### INDIRECT SIGNS OF CANCER

(1) *Obstruction of either Orifice.* Cancer at the cardiac end of the stomach may lead to obstruction below the cardiac orifice, but actually



Fig. 735.—CARCINOMA OF THE PYLORIC END OF THE STOMACH. INFILTRATION OF THE WALLS CAUSES RIGIDITY AND FREE OUTFLOW THROUGH A GIVING PYLORUS. THIS PATIENT HAD MANY MISFORTUNES: THERE IS A STONE IN THE LEFT KIDNEY, GALL-STONES WERE FOUND AT OPERATION, SHE ALSO HAD A SUBSTERNAL THYROID.

this is uncommon, cancer of the œsophagus and cardiospasm being the more frequent causes of obstruction.

Obstruction of the pylorus is more common since the greater number of cancers of the stomach—over 50 per cent—occur in this area. Even



*Fig. 736.*—PARTIAL FILLING OF THE SAME STOMACH AS FIG. 737, SHOWING INTERRUPTION OF THE LINEAR FOLDS OF MUCOUS MEMBRANE BY THE GROWTH.



*Fig. 737.*—CARCINOMA OF THE FUNDUS OF THE STOMACH CAUSING A SLIGHT IRREGULARITY OF THE SMOOTH LINE OF THE LESSER CURVATURE.

so, only a small percentage of cancers at the pyloric end of the stomach give rise to obstruction ; more frequently carcinomatous infiltration of the walls of the pylorus leads to a rigid gaping pylorus with free outflow.

(2) *Rapid Emptying.* As noted above, cancer which infiltrates the wall of the stomach leads to rigidity, and as this spreads into the pylorus—and 50-60 per cent of cancers of the stomach are found at the pyloric end—the pylorus becomes a thickened and rigid tube, loses its power of muscular contraction, and remains a gaping orifice, through which barium pours freely into the duodenum (fig. 735). The stomach in cancer of this type is therefore a relatively small one and empties rapidly.

(3) *Deformity of the Mucous Membrane.* The rugæ of the mucous membrane of the stomach run normally in wavy parallel lines, and the growth of cancer interrupts and obliterates these lines. Interruption and destruction of the rugæ is a valuable accessory sign of cancer when it can be determined with certainty that there is no spasm causing the deformity. It is another expression of the “filling-defect,” and is seen in all cases of cancer which cause a filling-defect (figs. 736 and 737).

#### DIFFERENTIAL DIAGNOSIS

Although the “filling-defect” is a cardinal sign of cancer, there are other causes of filling-defects which must be recognised.

(1) *Foreign Body.* Foreign bodies in the stomach, e.g. “hair-ball,” cause filling-defects in the gastric lumen. Large particles of food similarly cause filling-defects. All of these are mobile and can be shifted from the gastric wall by palpation.

(2) *Benign Tumour.* A benign tumour is most likely to be mistaken for cancer, particularly because the filling-defect is similar and because of its rarity. In outline a benign tumour is smoother than the filling-defect of cancer, it is usually a somewhat polypoid growth, and therefore causes a greater filling-defect of the gastric lumen than in the gastric outline (fig. 739).

(3) *Pressure from Without.* Pressure from the spine or from contraction of the abdominal muscles causes distortion of the gastric outline which is not difficult to recognise (see fig. 719). Similarly, an enlarged liver or gall-bladder, growth of the pancreas, enlarged spleen,

and any tumour occupying the upper middle abdomen, presses upon and to some extent causes filling-defect or distortion of the gastric outline. In films—as for instance in cancer of the pancreas—the filling-defect may appear like that of cancer of the stomach, but it can generally be seen by screen examination that the stomach and the tumour are independent.

(4) *Spasm*. The types of spasm to which the stomach is liable have already been discussed (see page 1486). The spasm which most closely imitates the filling-defect of cancer is the concentric spasm of the pyloric end of the stomach, which occurs with a gastric ulcer in this position. It is the most difficult condition we have to meet in differentiating between cancer and a non-malignant state. A filling-defect which is constant at all times, which cannot be relaxed or altered by massage and which remains the same at repeated examination and after medical treatment for ulcer, is almost definitely cancer. If, in addition to this, there is a palpable tumour corresponding to the filling-defect, the diagnosis becomes certain.

An ulcer on the anterior wall of the stomach, adherent to the abdominal wall or liver, may cause a filling-defect and tumour which cannot be distinguished from cancer. In such cases the true nature of the condition cannot even be determined macroscopically when removed from the body, and the diagnosis can only be made certain by microscopic section. Both ulcer and cancer may cause partial obstruction to the pylorus; with cancer the filling-defect is usually a large one and the tumour palpable, but more often cancer leads to a free outflow through the pylorus from infiltration of the gastric walls. Spasm is not an associated sign of cancer. On the other hand, an inflammatory condition, such as ulcer, gives rise to intense spasm which invariably causes some degree of pyloric obstruction and consequent delay in emptying of the stomach.

#### 4. SYPHILIS

*Syphilis of the stomach is rare. Pathologically it is a gummatous infiltration of the submucosa, and the X-ray appearances depend upon whether the infiltration is diffuse—causing a small contracted stomach—or localised with ulceration of the mucosa—causing the spastic contractions of an inflammatory lesion. At times, therefore, the radiological signs are like those of cancer and at times like ulcer. It follows that a lesion showing as an extensive filling-defect or deformity of the*

gastric outline, but in which no tumour can be felt, should not be diagnosed hastily as carcinoma without considering syphilis; in the same way the totally contracted, rigid stomach known as "leather-bottle," or "limitis plastica," is either cancer or syphilis. Also, an hour-glass contraction of the stomach in which no ulcer crater can be demonstrated, and which does not relax at repeated examinations or following ulcer treatment, may be due to syphilis of the stomach.

As elsewhere in the body, so in the stomach, syphilis may assume many forms, but the characteristic X-ray signs are :

- (1) *General contraction of the stomach ;*
- (2) *Rigidity of the stomach wall ;*
- (3) *Filling-defects without palpable tumour ;*
- (4) *Thickening of the mucous membrane folds ;*
- (5) *Hour-glass deformity with no ulcer crater.*

## 5. TUBERCULOSIS

Tuberculosis of the stomach is a very rare lesion which is only found when the alimentary tract is extensively diseased. Radiologically it is indistinguishable from syphilis.

## 6. BENIGN TUMOUR

Non-malignant tumour of the stomach is uncommon. The X-ray sign is a filling-defect in the barium-filled stomach, which is likely to be mistaken for cancer owing to the far greater frequency of malignant tumour.

The points which help to distinguish a benign from a malignant tumour are :

- (1) The *outline* is smooth and the tumour single, except in polyposis. Cancer shows many irregular projections.
- (2) The *gastric outline* is normal up to the base of the tumour, so that the filling-defect is greater in the lumen than in the wall. Cancer is primarily an infiltration of the gastric wall.

A variety of benign tumour called gastric polyposis shows multiple circular filling-defects in the gastric outline of the pyloric end of the stomach, or long smooth filling-defects running along the mucous



membrane rugæ in the fundus of the stomach. These multiple polypi are sometimes adenomata and sometimes inflammatory hypertrophy such as is seen in mucous membranes in other parts of the body.

## 7. GASTRO-JEJUNAL ULCER

The stomach after gastro-jejunostomy appears smaller than normal because barium passes out freely through the stoma, and the stomach never becomes distended.

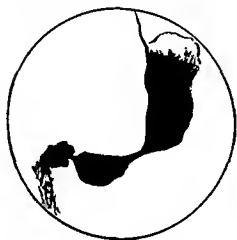


Fig. 732.—SYPHILIS OF THE STOMACH, SHOWING THE "DUMB-BELL" TYPE OF ORGANIC CONTRACTION.

(From Bull's "X-ray Interpretation.")

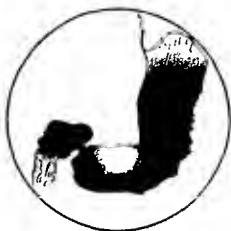


Fig. 733.—BENIGN TUMOUR IN THE PYLORIC END OF THE STOMACH. THE COMPARATIVELY SMOOTH OUTLINE OF THIS FILLING-DEFECT SUGGESTS ITS NON-MALIGNANT NATURE.

(From Bull's "X-ray Interpretation.")

The signs of gastro-jejunal ulcer are :

- (1) *Spastic closure of the stoma ;*
- (2) *Spastic contraction of the stomach round the stoma and of the jejunum below it ;*
- (3) *Ulcer crater (see fig. 201).*

The positive demonstration of an ulcer crater is direct proof of a gastro-jejunal ulcer, but the technical difficulties are such that no crater can be visualised or reproduced on a film in some cases. The indirect evidence of gastro-jejunal ulcer is, however, almost as definite as direct evidence ; closure of a gastro-jejunostomy stoma and spastic contraction of the stomach and jejunum are always secondary to inflammation in the stoma, and this inflammation is invariably gastro-jejunal ulceration, although the ulcer may still be an erosion without visible crater.

Extreme inflammation leads to adhesions between the jejunum and colon, and a jejunal ulcer may erode and perforate into the colon forming a jejuno-colic fistula.

### 8. RECURRENT CARCINOMA

Surgical removal of carcinoma of the stomach is hopeful when the tumour is situated in the mobile pyloric end of the stomach and when the patient is fortunate enough to come for examination early while the growth is small and confined to the stomach. At operation the



Fig. 740—RECURRENT OF GROWTH IN THE STOMACH FOLLOWING GASTRECTOMY FOR CARCINOMA. THERE ARE FILLING DEFECTS BOTH IN THE STOMACH AND JEJUNUM (From Bell's "X ray Interpretation.")

greater part of the stomach is removed, and X-ray examination subsequently shows a small vertical portion below the cardia passing directly into the jejunum.

Recurrence of growth occurs in glands or in the suture line of the gastrectomy. X-ray examination shows pressure upon the lesser curvature from a mass of growth outside the stomach, and irregularity both of the stomach and jejunum from invasion of the walls (fig. 740). The outlet may be narrowed, but rarely becomes obstructed; growth tends to spread diffusely along the mesentery.

Recurrent growth is the only complication of the operation for partial gastrectomy.

### 9. DIAPHRAGMATIC HERNIA

A small part of the cardiac end of the stomach may be found above the diaphragm; this is the congenital form of diaphragmatic hernia

and is not uncommon ; there may be no symptoms, or there may be attacks of colicky pain which—from its position—is regarded as cardiac. There are usually no symptoms until after middle life.

X-ray examination shows a small gas-bubble above the diaphragm, while the herniated portion of the stomach may or may not retain barium, and is easily mistaken for the lower end of the oesophagus.



Fig. 741.—DIAPHRAGMATIC HERNIA. A PORTION OF THE CARDIAC END OF THE STOMACH IS SEEN ABOVE THE DIAPHRAGM—CONGENITAL TYPE IN AN ELDERLY PATIENT. THERE ARE ALSO MULTIPLE DIVERTICULA IN THE COLON.

Acquired diaphragmatic hernia is often a chance discovery some years after a severe accident.

The stomach and colon are the abdominal viscera most often found in the thorax in acquired hernia. The intra-thoracic portion of the stomach is large—at least half of the stomach—unlike that of congenital hernia in which the greater part remains in its place below the diaphragm.

Acquired diaphragmatic hernia is immediately recognised with a

barium meal or barium enema, but sometimes the chest symptoms are more marked than the digestive, and a film of the chest shows faint shadows in curved lines or little fluid levels which are puzzling in diagnosis.

#### 10. CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS

This is pyloric obstruction, and the X-ray evidence is that of gross delay in emptying of the stomach in an infant in the first few weeks of life. When barium in milk is given to a normal infant it begins to pass out of the stomach almost immediately, and is evacuated in

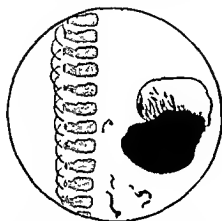


Fig. 742.—CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS. RIGHT ANTERIOR OBLIQUE VIEW TAKEN ONE HOUR AFTER A SMALL DRINK OF BARIUM. THERE IS A TRACE OF BARIUM IN THE DUODENUM AND A LITTLE HAS PASSED INTO THE SMALL INTESTINES.

(From Bull's "X-ray Interpretation.")

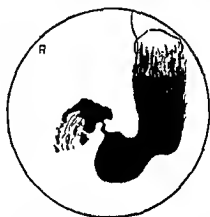


Fig. 743.—PYLORIC HYPERTROPHY IN AN ADULT. THE PYLORUS IS LONG AND ANGULATED AND PROJECTS INTO THE BASE OF THE DUODENAL CAP CAUSING IT TO BE CONCAVE. (From Bull's "X-ray Interpretation.")

less than three hours. The characteristic X-ray finding in congenital hypertrophic stenosis is that little or no barium passes out through the pylorus in the first hour, and the greater part remains in the stomach—or has been vomited—after three hours. Visualisation of the pylorus or of the duodenal cap is not possible in an infant, and the deep peristalsis—although observed clinically—is not the constant feature in an X-ray film of these cases, as is hyperperistalsis in duodenal obstruction of the adult.

#### 11. PYLORIC HYPERTROPHY

Pyloric hypertrophy may occur in adults, but without stenosis or any symptoms of obstruction. It is an X-ray finding, and does not

present a definite clinical picture. Kirklin and Harris (*Proc. Staff Meet. Mayo Clinic*, p. 644, Vol. vii, 1932), who first described it, stated that it was mostly found in the presence of disease of the stomach, duodenum, or gall-bladder.

On the X-ray film the pyloric canal appears longer than normal—due to the fact that it projects farther into the base of the duodenum. This projection causes the sides to overhang so that the duodenal cap resembles a spearhead. In addition, the pylorus may be angulated instead of a straight canal (fig. 743).

## II. THE DUODENUM

### THE NORMAL DUODENUM

THE appearance of the duodenum on the X-ray film is the same as in textbooks of anatomy. The first inch of the duodenum—which is histologically more like the stomach—has a particular radiological interest, since it holds barium for a few seconds and shows an outline like a rounded cone, referred to as the duodenal cap. The property of retaining barium stationary—even for a short period—is foreign to the small intestines, and is seen only in the first part of the duodenum and in the terminal ileum.

The duodenal cap has a further interest, both radiologically and pathologically, since it is the seat of 90 per cent of duodenal ulcers. Constant in its general outline it varies a little in size and position with the habitus of the patient. Like the stomach it is large and toneless in the hyposthenic, but relatively small and business-like in the hypersthenic. In the patient of average build and in the hyposthenic it is vertical, in the hypersthenic it lies horizontally or pointing downwards, and is situated behind the pyloric end of the stomach (see figs. 715, 716 and 717).

The second part of the duodenum passes vertically downwards and the third part crosses the spine obliquely behind the lesser curvature to the duodeno-jejunal angle. Barium passes rapidly through the duodenum and jejunum, showing only the feathery outline as it is caught up in the valvulae conniventes. Occasionally there is a momentary pause and occasionally there is retroperistalsis, but these points seem to have none but minor significance. True delay is, however, pathological.

## DISEASES OF THE DUODENUM

## 1. DUODENITIS

It may sometimes be seen at a screen examination that barium does not pause for the customary few moments, but is shot out by rapid contraction of the duodenal cap as soon as—or even before—it is quite filled. This irritability is sometimes due to irritation from outside, e.g. the inflammatory adhesions of cholecystitis; sometimes it is seen in conjunction with true gastric inflammation from a bout of

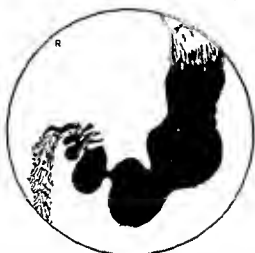


Fig. 744.—DUODENAL ULCER. MULTIPLE INCISURE CAUSING FIXED DEFORMITY OF THE DUODENAL CAP.

alcoholism; often it is a remote effect associated with a tender appendix. At operation congestion and actual erosion of the mucous membrane of the duodenum has been found, and this state, which we regard as duodenitis, may indeed be a forerunner of duodenal ulcer.

## 2. DUODENAL ULCER

Ninety per cent of duodenal ulcers occur in the cap, and over 99 per cent in the first two inches of the duodenum.

(a) *The cardinal sign of duodenal ulcer* is constant deformity of outline of the cap, a deformity which—once established—is permanent even in the periods of inactivity of the ulcer. The deformity may

assume many forms, but any departure from the normal outline (fig. 745) indicates disease of the duodenum, which in the great majority of cases is duodenal ulcer. The other causes of duodenal deformity are adhesions to adjacent viscera—usually the gall-bladder—post-operative adhesions, tumours pressing from outside, and primary new growth of the duodenum.

The common type of deformity is the spastic incisura cutting into the duodenal cap from one or both sides; the least common is the niche projecting from the outline like a gastric ulcer; chronic ulcers, with much induration and signs of obstruction, may show almost total obliteration of the cap (fig. 745).

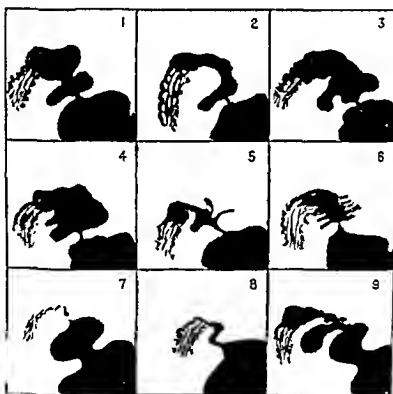


Fig. 745—Types of Deformity of the Duodenal Cap. 1. Spasm dividing the cap in the middle. 2. Deformity of the outer aspect. 3. Concave deformity of the base. 4. Irregularity of the outer aspect. 5. Gross deformity. 6. "Fir tree" deformity. 7. Obliteration of the cap with obstruction and gastric hyperperistalsis. 8. Total deformity with partial obstruction. 9. Gross deformity, part of the cap being almost separated from the rest as a pseudo-diverticulum.

(From Bull's "X-ray Interpretation.")

(b) Besides the direct signs of duodenal deformity there are certain accessory signs :

- (i) *Duodenal irritability ;*
- (ii) *Excess secretion ;*

- (iii) *Delayed emptying* ;
- (iv) *Enlargement of the stomach* ;
- (v) *Hyperperistalsis*.

(i) *Duodenal irritability* has already been mentioned ; in the absence of constant deformity it is not a sign of ulcer ; it is frequently seen in the presence of ulcer and may indicate activity. It is a sign which belongs to the screen examination, the only evidence on films being that a large series may fail to show a filled cap.

(ii) *Excess secretion in the stomach* is a sign only appreciated at the screen examination. It is seen in duodenal ulcers in conjunction with the other signs of partial obstruction, but is not pathognomonic of obstruction since it may occur from any general cause leading to gastric inertia, e.g. migraine.

(iii) *Delay in emptying of the stomach* of over six hours is a sign which—like excess secretion—is accessory to duodenal ulcer with partial obstruction, but as a sign is governed by the same limitations.

(iv) *Enlargement of the stomach* is a sequel to slow progressive obstruction to the outflow, and as such is an accessory in the diagnosis of duodenal ulcer. In the same way—

(v) *Hyperperistalsis* occurs from any progressive obstruction, and is most characteristically seen in duodenal ulcer. The waves are deeper and more frequent, so that as many as four waves can be seen passing through the stomach at the same time (fig. 746).

### 3. NEW GROWTHS OF THE DUODENUM

*Benign tumours* of the duodenum—leiomyomas, fibro-adenomas, hæmangiomas—may be found in any part of the duodenum, but they are rare. They are likely to be overlooked unless they occur in the first part of the duodenum, or unless they become large enough to cause obstruction. The smooth filling-defect in the lumen of the duodenum is similar to that of the benign tumour of the stomach.

*Primary cancer* of the duodenum is also rare : it arises more often from the ampulla of Vater than from the mucous membrane of the duodenum, and jaundice is an early symptom. When occurring in the first part of the duodenum cancer causes deformity and later obstruction, so that it is likely to be mistaken for duodenal ulcer—the much commoner lesion giving rise to similar X-ray signs.



## 4. DIVERTICULA

Diverticula are found in each part of the duodenum, but they differ in character and appearance in the three parts :

- (a) *From the first part*—the duodenal cap—true diverticula are uncommon ; false or apparent diverticula are found when the duodenal cap is grossly distorted by the scarring of a duodenal ulcer cutting off a corner of the cap of rounded outline and

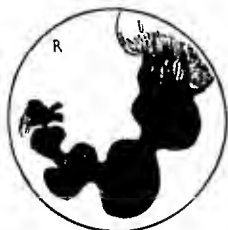


Fig. 746.—DUODENAL ULCER WITH PARTIAL OBSTRUCTION AND GASTRIC HYPERPERISTALSIS. THE STOMACH IS ENLARGED, THE WAVES OF PERISTALSIS ARE DEEP, AND FOUR ARE SEEN IN THE STOMACH AT THE SAME TIME.

(From Bull's "X ray Interpretation.")

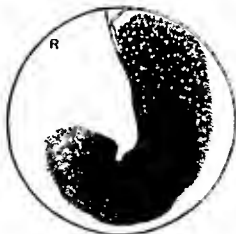


Fig. 747.—THE STOMACH OF OBSTRUCTION FROM CIRCUMSCRIBED DUODENAL ULCER. BARIUM IS SEEN MIXED WITH RETAINED FOOD AND SECRETION, THE STOMACH IS DILATED AND ALMOST INACTIVE.

(From Bull's "X ray Interpretation.")

narrow neck (see fig. 68). These are distinguished histologically by the fact that there is muscle in the walls, and radiologically because they do not retain barium after the meal has passed by.

True diverticula do, however, occur and are seen in the absence of duodenal ulcer. Such a diverticulum projects from the duodenal cap with rounded outline similar to that of diverticula elsewhere ; it is incapable of contraction, and retains barium for some hours after the stomach is empty of the meal (see fig. 69).

- (b) *From the second part.* This is the commonest situation for duodenal diverticula, and they mostly project from the medial aspect in the region of the ampulla of Vater (see fig. 70). They

are true diverticula and retain barium for several hours. Clinically they give rise to a flatulent dyspepsia, with occasional painful attacks, probably due to fermentation and inflammation within the diverticulum. Therefore patients are often sent for X-ray examination with the provisional diagnosis of cholecystitis.

- (c) *From the third part.* Diverticula occur also from the third part; they are usually small, often multiple, and are found not only in the third part of the duodenum but scattered throughout the length of the small intestines.

## 5. OBSTRUCTION

(a) *Obstruction in the first part of the duodenum* is most commonly due to the scarring and contraction of a chronic duodenal ulcer; very rarely it may be due to a primary new growth.

The screen examination shows an enlarged stomach full of secretion and food, and this should be emptied with a stomach tube and the stomach washed out daily for a week before it is X-rayed again. At the re-examination it may be possible to force barium through the pylorus and duodenum, and to show both on screen and films that the gastric side of the pyloric canal is normal and the duodenal cap totally deformed with only a small channel running through it.

In films taken at intervals the large stomach is seen, at first with deep waves of peristalsis, or hyperperistalsis, trying to force the stomach contents past the obstruction, and later as a toneless sac in a state of inertia—having given up the hopeless struggle (fig. 747).

(b) *Obstruction in the second part of the duodenum* is rare, and is due either to primary new growth of the duodenum, or is secondary to growth in the head of the pancreas. Since this is a more rapidly progressive form of obstruction the stomach is less dilated, hyperperistalsis is marked, inertia is not a feature, and the duodenum is dilated up to the point of obstruction.

(c) *Obstruction in the third part of the duodenum.* This is almost always due to the pressure of enlarged glands in the root of the mesentery, the cause of the enlargement being tuberculosis, carcinoma, or lymphadenoma. Obstruction is never complete, but the appearance of the duodenum filled out with barium—like the stomach—is a

striking one, which contrasts with its normal feathery outline; the passage of barium through the small intestines is normally so rapid that little more is seen than the barium which is caught up in the folds of the valvulæ conniventes.

## 6. DUODENAL DELAY

Apart from mechanical obstruction there is delay which occurs in the duodenum, and is a reflex inhibition from peritoneal irritation in some portion of the alimentary tract below the duodenum.

It is found in tuberculous peritonitis, and in small intestine obstruction with peritoneal irritation or peritonitis.

X-ray examination may show excess secretion in the stomach—but a normal stomach and duodenum; barium does not, however, clear readily from the duodenum, and although the duodenum does not become outlined as in partial obstruction, a little pool is apt to collect in the third part, showing that the normal peristalsis which allows no such thing is partially or temporarily inhibited.

Retroperistalsis is also seen in the screen examination, a column of barium being carried up the second and into the first part of the duodenum, from which it falls back into the stomach. This act of retroperistalsis, which may be seen in the presence of mechanical or reflex duodenal obstruction, is not of itself a sign of importance, since it is quite often encountered in routine examinations of the stomach and duodenum, and has no pathological significance.

## III. ILEUM AND JEJUNUM

THE passage of barium is so rapid along the small intestines that they are not outlined—only indicated by the temporary delay of barium in the valvulæ conniventes. It is, therefore, extremely hard to diagnose any lesion of the small intestines, and it is perhaps fortunate that those lesions are few in number and low in frequency.

The most important as well as the commonest lesion is acute obstruction, and as this is a surgical emergency it is rarely X-rayed. Chronic intussusception can be X-rayed and the point of obstruction determined: when the small intestine is intussuscepted into the colon, the papilla-like head of the advancing small intestine can be outlined by a barium enema, which also shows the length to which it has reached.

In the diagnosis of disease of the small intestines we are guided almost entirely by alteration in the normal rapid rate of travel.

As soon as barium has left the stomach it pursues a steady course through the twenty-three feet of small intestines and does not pause until it reaches the last twelve inches or so of the ileum. Within an hour of taking barium it may be seen passing into the cæcum. The rate of travel varies a little in different individuals and under different conditions ; there is a compensatory neuro-muscular balance between the stomach and the small intestines ; the stomach empties readily when the small intestines are in a state to receive the contents, and the introduction of food into the stomach accelerates the passage out of the terminal ileum into the colon.

As in duodenal delay the causes of delay in the passage of barium through the small intestines and into the cæcum are peritoneal irritation causing reflex inhibition of peristalsis, and obstruction due to intussusception, new growth, or foreign body.

Acute peritonitis is not often X-rayed. The causes of chronic peritonitis, which give rise to reflex inhibition of peristalsis and consequent delay in the passage of barium through the small intestines, are tuberculosis and diffuse secondary carcinoma, such as is seen from the rupture of a malignant ovarian cyst.

In these cases barium may be found in the small intestines twelve hours after it has left the stomach ; in tuberculosis it is usually in the terminal part of the ileum, and this with the cæcum is tender to palpation.

#### OBSTRUCTION

Partial obstruction may occur in some cases from intussusception, from primary and secondary new growth, from compression by a band of adhesions, and rarely from a large gall-stone which has ulcerated into the duodenum and cannot get past the ileo-cæcal opening.

The X-ray evidence of obstruction is :

The coils of small intestine

- (1) become and remain filled with barium ;
- (2) are dilated ; and
- (3) are distended with gas and show pools of secretion with fluid levels. Further
- (4) there is delay in the passage of barium into the colon.

In the normal subject a large film of the abdomen taken an hour or two after a barium meal shows barium scattered through the small

intestines : in the jejunum the appearance is feathery, and in the lower parts of the ileum this feathery look becomes lost as the valvulæ conniventes become smaller. But the appearance is never the same in films taken at ten-minute intervals ; barium is continually moving forwards, and it is mere chance if any loop of jejunum or upper ileum appears the same in successive films ; in the last few feet of the ileum—situated in the pelvis and right iliac fossa—there is physiological delay as the barium passes slowly into the cæcum, and this part of the intestine is continuously filled and shows a narrow lumen of diameter not greater than three-quarters of an inch with no valvulæ conniventes.

In obstruction, coils of small intestine become greatly dilated and filled with barium so that the valvulæ conniventes are obscured. As a rule one or two coils become thus filled and remain filled at successive examinations in a manner which is foreign to normal intestines. Such distended coils do not necessarily indicate the point of obstruction, but barium may be prevented by gas distension or by inhibition of peristalsis from proceeding further. Other coils of intestine may show no barium but pools of secretion in coils of gas-distended intestine.

In these cases of partial obstruction the further progress of the barium meal is often interrupted by vomiting ; barium which is not vomited is still found in the small intestines after twelve and twenty-four hours.

#### IV. THE APPENDIX

IN X-ray work as in every other branch of medical diagnosis, we have to learn and recognise the normal before we can know that any appearance or aggregate of signs denotes pathology.

For the appendix this is not possible. Histologically, every appendix removed shows greater or lesser evidence of disease, and therefore every conceivable X-ray appearance of the appendix has at some time been adduced as evidence of disease, and confirmed histologically. The diagnosis of "chronic appendix" on any grounds whatever can never be wrong.

In addition to this there are peculiar difficulties in the radiology of the appendix, difficulties attributable to its physiology, and which render the distinction between physiology and pathology impossible to make on X-ray evidence in certain cases.

(a) *Filling and emptying.* It is a physical platitude that a blind diverticulum cannot be filled unless it is first cleared of its existing

contents, and we do not yet know what controls the filling and emptying of the appendix. About 50 per cent of appendices show some degree of filling with harium at one examination—which points to even chances.

Further, we know that the appendix has a certain penchant for metals and insoluble bodies, and if it is fortunate enough to catch them as they pass it will retain them for days or even weeks. Pieces of metal ingested with the food and bristles of tooth brushes were, indeed, once thought to be the cause of appendicitis. In the shooting season lead shot in the appendix are frequently seen in patients X-rayed for some other condition; sometimes they remain for weeks, sometimes they are turned out in a few days. Repeated purgatives tend to empty the appendix, but a single purgative is not necessarily effective.

Inflammation is the most powerful and constant factor in emptying the appendix. An appendix which fills with harium and empties again in a few hours is invariably tender and is almost certainly inflamed.

From the above it will be seen that the filling of the appendix with harium is probably governed more by chance than anything, and that failure to fill is more often obedience to a physical law than to obliteration of the lumen. Some observers use three successive harium meals preceded by purgatives; this undoubtedly increases the chances of filling, but failure after an arbitrary number of attempts cannot be regarded as proof of organic stenosis.

Over-distension of the cæcum by harium enema will often result in filling of the appendix within the succeeding twenty-four hours; it is interesting to note that the appendix does not usually fill at once with a barium enema, but some process of emptying occurs after the enema is evacuated and so allows entry of the barium.

The emptying of harium from the appendix is another uncertain action; sometimes it passes out within a few days, but often the appendix remains partly filled for many days. It seems to be the normal function of the appendix to retain heavy metals, and there is no good evidence to show that slow emptying is pathological.

Like the rest of the colon the appendix has the power of fluid absorption, and barium shows concentration and segmentation after twenty-four or forty-eight hours.

(b) *Outline.* Occasionally an appendix shows a smooth unbroken outline of barium from base to tip, but more often it is broken up and

partly irregular, due no doubt to faeces. Sometimes circular filling-defects in the lumen—constant at repeated examinations—indicate faecoliths. Some appendices are curled and some are straight, or they may be sharply angulated or fixed at the tip; angulations and points of fixation indicate adhesions from past inflammation.

(c) *Position.* The appendix is most often dependent from the caecum or pointing inwards, but it may be retro-colic or ante-colic; occasionally the caecum also is ante-colic—flexed in front of the ascending colon—and the appendix lies under the liver.

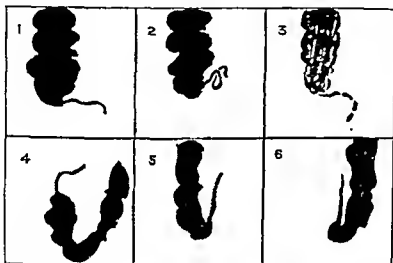


Fig. 748—VARIABLE POSITIONS AND APPEARANCES OF THE APPENDIX. 1. THE COMMON POSITION. 2. APPENDIX CURLED UPON ITSELF. 3. SEGMENTED APPEARANCE. 4. THE ANTE COLIC CAECUM WITH THE SUB HEPATIC POSITION OF APPENDIX. 5. RETRO CAECAL APPENDIX. 6. ANTE CAECAL APPENDIX.  
(From Bull's "X-ray Interpretation.")

(d) *Mobility.* The long straight appendix hanging down towards the pelvis is freely mobile. It is usually hard to demonstrate mobility in the curly appendix—which is also a difficult one to fill. The retro-colic and ante-colic appendices cannot be shifted from their positions.

(e) *Tenderness.* This is perhaps the most important—almost the only certain—point in diagnosis of the pathological appendix. An appendix which can be seen, rolled under the finger and shown to be locally tender to pressure, is an inflamed appendix. Whether such inflammation is a primary appendicitis, or whether it is due to a spreading infection such as tubo-ovarian inflammation or tuberculosis involving the mesentery and caecum, cannot easily be determined.

## SUMMARY

There are no certain signs by which we can differentiate between the normal and the abnormal appendix, but tenderness localised to the appendix, supported perhaps by filling-defects due to faecoliths or by evidence of adhesions, justifies a diagnosis of appendicitis.

Further, an appendix which empties rapidly and is locally tender to pressure is an inflamed appendix.

## V. THE COLON

The colon can be examined with a follow-through meal, or by barium enema or both. The only information obtained from the follow-through meal is that of motility, and even this is unreliable since movement along the large intestine is almost entirely dependent on defaecation; defaecation may fail to happen at 24 hours even in those of regular habits, and barium retained in the colon up to 48 hours is likely to pack hard and become constipating—requiring a considerable purgative or an enema to remove it. Thus a follow-through examination may show a different motility in the same patient on different occasions.

For the determination of organic disease examination should be by barium enema; a thin suspension of barium sulphate is run into the colon by gravity through a self-retaining enema nozzle, and the flow is watched under the fluorescent screen and the colon palpated as it fills—with the patient in different positions. This method reduces the error in diagnosis of disease of the colon to the lowest possible minimum.

## NORMAL COLON

The normal colon is filled with  $1\frac{1}{2}$  to 2 pints of fluid. The diameter of the colon is greatest at the caecum and diminishes progressively to the rectum where it is again larger. The normal flexures are those described in anatomy, but there are often small secondary flexures; these are all examined in turn by rotating the patient into different positions as the colon fills. All these flexures are exaggerated in patients of the hyposthenic habitus (visceroptosis), and are least marked—the angles being widest—in patients of hypersthenic build.



In films these flexures show overlap, and if disease is found at screen examination films are taken in such oblique positions as are necessary to demonstrate it; otherwise it is not necessary to record each of the many flexures.

Haustrations are most marked in the transverse colon, less in the cæcum, descending colon and sigmoid, and are absent in the rectum.

The ileo-cæcal valve is no sort of valve in the mechanical sense; in the majority of normal colons barium passes into the ileum—sometimes a little, sometimes freely—and this backflow seems to depend



Fig. 749.—NORMAL COLON OF SOMEWHAT HYPERSTHENIC TYPE; SMALL SIGMOID LOOP, HIGH STRAIGHT TRANSVERSE COLON.  
(From Bull's "X-ray Interpretation.")



Fig. 750.—CHRONIC ULCERATIVE COLITIS. RIBBON LIKE COLON WITHOUT HAUSTRATIONS.  
(From Bull's "X-ray Interpretation.")

more on the fluid contents of the ileum than on the state of the valve. If food has been taken five or six hours previously barium is less likely to pass back through the ileo-cæcal valve than if the patient is examined fasting.

*Emptying of the colon.* Little can be interpreted from the quantity of barium retained in the colon in a film taken after evacuation of the enema; emptying seems to depend a great deal on the psychology and temperament of the patient; really good physiological emptying with uniform contraction of the lumen of the bowel does not always occur, and many apparently normal colons retain a great deal of the enema after evacuation.

## PATHOLOGICAL CONDITIONS OF THE COLON

## (A) INFLAMMATORY DISEASES

1. *Ulcerative colitis.*
2. *Tuberculous colitis.*
3. *Actinomycosis.*
4. *Diverticulitis.*

## 1. ULCERATIVE COLITIS

The colon of ulcerative colitis has a thickened and comparatively rigid wall and a narrowed lumen. It outlines rapidly with a barium enema, is incapable of distension, and is filled with a small quantity of fluid—often less than a pint.

When filled the colon is distinctive because of its small size and narrow lumen free of haustrations; it has a ribbon-like appearance. There is a general contraction so that all the flexures are smaller and the total length of the colon appears less (fig. 750).

This is when the whole colon is affected and the disease has existed for some time. In earlier cases the distal parts of the colon show these changes and the proximal half appears radiologically normal; the rectum and sigmoid arc always the parts most affected in ulcerative colitis and bacillary dysentery, and the inflammation spreads progressively upwards; in amoebic dysentery and tuberculous colitis the caecum is most affected and the disease spreads downwards.

In chronic ulcerative colitis the mucous membrane becomes undermined, large pieces are shed, and islands of inflamed mucous membrane like polypi remain surrounded by submucosa. This polypoid appearance can be demonstrated on films by using a thin emulsion of barium, or by filling the colon with air after evacuation—the technique of Kirklin and Weber (*Staff Meet. Mayo Clinic*, Vol. v, No. 45, 1920). With healing, fibrosis and contractions may distort the lumen and produce irregularities of contour, but actual obstruction does not occur.

## 2. TUBERCULOUS COLITIS

This differs from ulcerative colitis in that it affects the caecum primarily, and only in severe cases spreads down to involve the greater

part of the colon. The other inflammatory diseases involving the cæcum are appendicitis, amoebic dysentery, and actinomycosis; the differential diagnosis can only be made by correlating all the available evidence—clinical, bacteriological, radiological, and finally surgical and microscopical.

From the X-ray examinations little more than circumstantial evidence can be obtained except in the very advanced cases. Barium meal shows no delay through the small intestines, but there is tenderness to palpation of the terminal ileum and cæcum, and the lumen may be irregular in outline suggesting spasticity. Barium enema also shows tenderness of the cæcum, and the outline may be normal under distension, or may show rigidity and palpable thickening and ultimately contraction. In cases not so far advanced, spasticity may be the only abnormal sign, and it is manifested by intermittent contractions of the cæcum, so that the barium performs a constant ebb and flow under screen observation. Passage of barium into the ileum may or may not occur, but in any case it has no significance. As a rule the appendix does not fill, and this combined with tenderness of the cæcum makes the diagnosis of appendicitis almost inevitable on the basis of probability.

### 3. ACTINOMYCOSIS

This uncommon specific inflammation gives an X-ray picture similar to tuberculosis, but rigidity of the cæcum is usually marked and the thickening palpable. It closely simulates carcinoma of the cæcum.

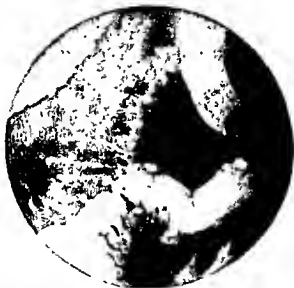
### 4. DIVERTICULITIS

Multiple diverticula of the colon are found quite often in patients over sixty, and occasionally as a surprise finding in patients considerably younger. They are projections of the bowel lining through the muscular coat, are always most marked in the sigmoid, but may occur through the whole length of the mobile parts of the colon. Diverticula are not necessarily associated with inflammation, and conversely there may be marked spastic contraction of the sigmoid with no demonstrable diverticula. Words and terms such as "diverticulosis" and the "pre-diverticular stage of diverticulitis" have been coined to express these phases.

Diverticula arise only from the mobile parts of the colon, not from the rectum; they occur mostly on the side of the colon away

from the mesentery and often project into the appendices epiploicæ. When the colon is filled with a barium enema, diverticula are seen as rounded projections from the lumen; sometimes they are small, sometimes large, and as noted above they may be associated with very marked spasm, while at other times there may be little or none (fig. 751).

The evidence of spasm of the colon is contraction of the circular muscle-fibres so that the affected area has an appearance something like the teeth of a saw; the lumen is narrowed, and the tooth-like projections radiate from the centre. At the point of some or all of these



*Fig. 751*—MULTIPLE LARGE DIVERTICULA, THE SIGMOID AND DESCENDING COLON OF AN ELDERLY PATIENT. THERE IS LITTLE ASSOCIATED SPASM.

projections, round diverticula may be seen (fig. 752). This spastic contraction in the presence of diverticula of the colon is evidence of inflammation, i.e. diverticulitis; the affected area is tender to palpation, and thickening of the howel wall can be appreciated in thin patients. Inflammation in the diverticula leads to oedema of the mucous membrane of the colon with narrowing of the lumen; often there is pericolitis, and occasionally diverticula of the sigmoid become adherent to the bladder. Perforation into the bladder has been reported.

The X-ray finding of a filling-defect with partial or even complete obstruction is similar to that of carcinoma of the colon. In the differential diagnosis the clinical evidence must be taken in conjunction with the radiological.

Diverticulitis is an inflammatory disease; the patient is ill with septic fever, the leucocytes are increased, but there is no loss of weight, and rarely is the passage of blood or mucus indicative of diverticulitis. On the X-ray side the finding of multiple diverticula in relation to a gross filling-defect suggests that the cause is inflammatory, but it must be added that a certain percentage of carcinomata begin in an area of diverticula. The filling-defect of carcinoma is that of an irregular constriction, and the saw-edge type of contraction is not that of carcinoma; besides, spasm is not an associated feature of new growth.



Fig. 752.—"DIVERTICULITIS" OF THE SIGMOID: THE "SAW EDGE" TYPE OF CONTRACTION DUE TO SPASM. DIVERTICULA ARE SEEN PROJECTING FROM THE POINTS OF CONTRACTION.

Whereas the finding of diverticula in relation to a filling-defect is suggestive of diverticulitis, the absence of diverticula is suggestive of new growth, provided that spasm and pressure from outside can be excluded.

In recording the screen findings of a barium enema films are taken with the patient in that position which best demonstrated the lesion; since the colon curves about the abdomen in three dimensions of space, the best position for radiography varies with the habitus of the patient and the nature of the disease.

### (B) NEW GROWTHS

1. *Benign tumours.* These are uncommon in the colon as elsewhere in the alimentary tract, but the incidence is higher in the colon than

in other parts because polypus of the rectum is not so infrequent, and because the mucous polypi residual from ulcerative colitis are often classed as new growth.

Polypus of the rectum is diagnosed by digital examination or by proctoscope. In any case it is very unlikely that the single small polypus will be seen by X-ray examination; it is not so difficult to recognise the diffuse inflammatory type of polyposis; the mucous membrane pattern becomes a honeycomb pattern which can be seen by using a dilute emulsion of barium, by compressing the bowel in those areas where compression can be suitably applied, or by filling the colon with air after evacuation of the barium enema.



Fig 753.—VILLOUS PAPILLOMA CAUSING A FILLING DEFECT IN THE TRANSVERSE COLON.

Papillomata are also found in the colon. When small they produce a somewhat inconstant filling-defect in the lumen of the bowel which is easily mistaken for faeces; the fixed position at re-examination after repeated lavage establishes the organic nature of the filling-defect, but the differential diagnosis from malignant disease is difficult; besides, these papillomata may become malignant.

2. *Malignant tumour.* Carcinoma may arise from the epithelium of the colon in any part, but does, in fact, arise most commonly in the rectum or sigmoid.

The cardinal sign of carcinoma is the constant filling-defect in the bowel lumen; this filling-defect is fixed in form, irregular in outline,

and has not the regular saw-edge outline of spasm (fig. 754). When a filling-defect of this nature can be identified at screen examination with a palpable tumour, the diagnosis is almost certainly carcinoma. In some cases there is partial obstruction with dilatation of the howel above and a palpable accumulation of fæces, at other times there is complete obstruction to the enema; the rectum and howel below the obstruction dilate until the patient is no longer able to bear the distension, hut no harium passes through the obstruction. The obstruction is, however, only apparent, the upward thrust of the enema



Fig. 754.—CARCINOMA OF THE DESCENDING COLON CAUSING A CONSTANT FILLING-DEFECT BUT LITTLE OR NO OBSTRUCTION TO THE FLOW OF THE ENEMA.

(From Bull's "X-ray Interpretation.")



Fig. 755.—MEGACOLON. THE SIGMOID LOOP REACHES UP TO THE SPLENIC FLEXURE AND THE ENTIRE ABDOMEN IS OCCUPIED BY THE BARIUM-FILLED COLON.

(From Bull's "X ray Interpretation.")

causing the projections of growth to act as a valve and to close up the lumen; these cases are not usually obstructed completely to the downward flow of fæces.

In differentiating between the filling-defect of carcinoma and other filling-defects the important points are the constant shape of the deformity and its identity with a palpable tumour. Fæces and gas cause filling-defects, hut they are inconstant in form and absent at re-examination after a purgative. Spasm and the irregularities of diverticula and diverticulitis have already been referred to. Inflammatory hypertrophy causing a recognisable filling-defect and even palpable thickening may occur in tuberculosis and actinomycosis; both these diseases are most frequently located in the cæcum and ascending colon, and the similarity to new growth may be such that

the diagnosis cannot be established on X-ray evidence alone. Both tuberculosis and actinomycosis are low febrile illnesses, and the symptoms in the colon are rather those of irritation and rapid passage of faeces than of obstruction. At the X-ray examination the filling-defect is more extensive and the palpable thickening less than a carcinoma of the same magnitude, and the caecum is tender to palpation. Healed dysentery may produce irregular contractions of the bowel lumen; there is no obstruction in these cases, the filling-defects are multiple, and the lumen of the colon throughout is narrower than normal.

Apparent filling-defects can also be produced by pressure on the colon from outside; sharp bends in the colon and the pressure from the ribs or the brim of the pelvis in thin patients express barium from an area of the colon; tumours of the spleen, stomach, or kidneys—and in women tumours of the uterus and ovary—may press upon and flatten the colon, but the extra-colic nature of these filling-defects is recognised by palpation under the screen.

### (C) CONGENITAL ABNORMALITIES

1. *Megacolon.* Various degrees of large colon are met with. The commonest is the large sigmoid loop, a loop capable of enormous distension reaching to the splenic flexure, but also retaining the power of contraction when the bowels are emptied. Or the whole colon may show an unusually large diameter, which distends readily, shows poor tone, and little disposition to contract at evacuation. Such cases are often diagnosed clinically in children who are pot-bellied and constipated, and are called Hirschsprung's "disease." Barium enema distends and fills the colon so that the abdominal cavity is almost entirely obscured by the barium shadow. Adult patients may retain three pints of fluid without discomfort. After evacuation a certain quantity of barium runs out by gravity, but re-examination shows that a large quantity is still retained and the colon shows little of that contraction which drives out the fluid contents in normal subjects.

2. *Left-sided colon.* The colon may lie entirely on the left side of the abdomen and the small intestines on the right side. It is a congenital anomaly and is usually a chance finding, since of itself it gives rise to no symptoms. The duodenum in these cases retains its mesentery and is mobile.



3. *Transposition of viscera.* This is another congenital anomaly which does not produce symptoms. It is recognised clinically by the

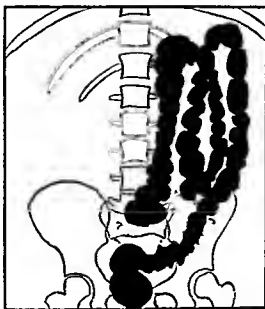


Fig. 750.—LEFT-SIDED COLON, DUE TO NON-ROTATION OF THE GUT IN FETAL LIFE.  
(From Bull's "X-ray Interpretation")

fact that the liver and spleen and the thoracic viscera are transposed. X-ray examination shows the abdominal viscera to be an exact mirror image of the normal.

## VI. THE GALL-BLADDER

THE density of the gall-bladder is such that it does not throw a recognisable shadow. When thickened by chronic cholecystitis or distended the rounded fundus can sometimes be seen below the liver, and when adherent to the duodenum the concave imprint of the fundus of the gall-bladder in the barium-filled duodenal cap is seen. Rarely the gall-bladder walls become calcified, or gall-stones contain calcium in such concentration that they can be recognised; these amount to less than 5 per cent of all gall-stones.

### CHOLECYSTOGRAPHY

The gall-bladder can be outlined by administering a halogen-containing chemical by mouth or intravenously. This chemical, which

was discovered by Graham and Cole (*Journ. Am. Med. Assn.*, p. 613, 1924), is excreted through the liver into the gall-bladder and throws a shadow of relative opacity.

The oral method is the method of choice, but when no shadow is obtained the examination must be repeated intravenously, since it is uncertain whether the dye has failed to enter the gall-bladder because of organic disease, or whether it has not been absorbed from the alimentary canal. Failure to obtain a shadow is often a technical one—the patient has not followed directions, or films have not been taken in such a way, or are not of such quality, as to reveal the gall-bladder shadow—but when the technical side is correct and no shadow is obtained, both from oral and intravenous administration, the indication is—in the absence of jaundice or severe liver disease—that the cystic duct is obstructed, or the gall-bladder so filled with stones that insufficient dye can enter to throw a shadow. Sometimes in patients with visceroptosis an atonic gall-bladder does not empty well before the dye is administered, and therefore a very faint and diluted shadow is obtained, or perhaps no shadow is seen at all by the oral method. This is overcome by repeating the test intravenously, when a shadow—although faint—is obtained.

Cholecystography tells the size, shape, position, and mobility with respiration of the gall-bladder, and its functional capacity indicated by filling and emptying—which also implies patency of the ducts. Uniform contraction after a meal shows that the walls are normal.

It is an accurate and valuable test of the functional efficiency of the gall-bladder and ducts; by cholecystography the pathological gall-bladder is distinguished from the normal and in most cases the nature of the pathology is determined.

But the accuracy and value are relative to the conditions under which the examination is made. They are highest where the examination is made by or under the supervision of a radiologist, because he will not be satisfied until the normal or pathological condition of the gall-bladder is clearly demonstrated. The accuracy becomes progressively less when radiography is carried out according to a fixed technique and time-table, as may happen in a hospital X-ray department; and if in addition the technical work is bad, cholecystography cannot be rated as high as clinical examination and is not worth the extra expense.

The details of administration of the dye are as important as the radiographic precision, since X-ray work, however good, cannot show a shadow when little or no dye has reached the gall-bladder. In order

to obtain good filling the gall-bladder should be empty and passively relaxed during the hours the dye is being excreted from the liver. We have learned from cholecystography that the way to empty the gall-bladder is by eating, and so the patient is directed to take a large mixed meal. After the meal the patient drinks the dye and goes to bed, taking nothing more before the X-ray examination the following morning.

To keep the gall-bladder relaxed vagal irritation must be avoided, and for this reason the patient is directed not to take a purgative or any medicine for two days before the examination; violent diarrhoea more than anything else reduces the chance of a good filling—possibly because the gall-bladder takes part in the general contraction of unstriated muscle; conversely, vomiting does not prevent gall-bladder filling provided that the dye is retained long enough for some to be absorbed; a good shadow can be obtained though the dye is vomited after ten minutes. Many patients experience nausea after drinking acid tetraiodophenolphthalein, and this is probably helpful in keeping the gall-bladder at rest; such patients mostly show good shadows.

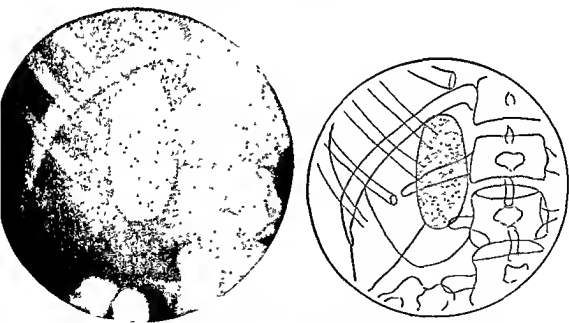


Fig. 757.—CHOLECYSTOGRAPHY. NORMAL GALL-BLADDER: THE OUTLINE IS EVEN AND THE DENSITY UNIFORM. NOTE THE RELATION TO THE LIVER AND KIDNEY SHADOWS.

#### THE NORMAL GALL-BLADDER SHADOW

In size, shape and position the normal gall-bladder varies, just as do the other abdominal viscera, according to the habitus of the patient.

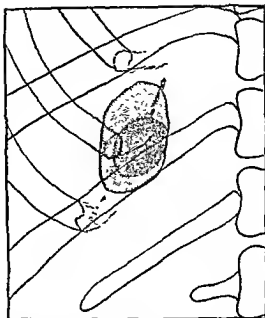


Fig. 758.—CHOLECYSTOGRAPHY OF A HYPERSTHENIC PATIENT. THE FUNDUS IS POINTING FORWARDS AND THE GALL-BLADDER IS SEEN END ON CAUSING A CIRCULAR DENSITY WITHIN THE MAIN SHADOW. NOTE THE HIGH POSITION OF THE GALL-BLADDER AND THE CALCIFICATION OF COSTAL CARTILAGE CROSSING THE GALL-BLADDER SHADOW.

(From Bull's "X-ray Interpretation.")

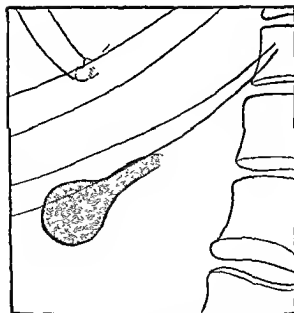


Fig. 759.—LEFT ANTERIOR OBLIQUE VIEW OF FIG. 758, SHOWING THE FORWARD POSITION OF THE GALL-BLADDER. TAKEN IN INSPIRATION.

(From Bull's "X-ray Interpretation.")

In the individual of hypersthenic habitus the gall-bladder is high, relatively small, and projecting little below the liver; owing to the upward pressure of the colon and abdominal fat the fundus of the gall-bladder points forwards and sometimes so directly forwards that the shadow is superimposed on itself and foreshortened (fig. 758).

In the hyposthenic patient the gall-bladder is long and pendulous, near the mid-line, with the fundus reaching perhaps into the pelvis (fig. 760). Like the other abdominal viscera containing muscle-fibres

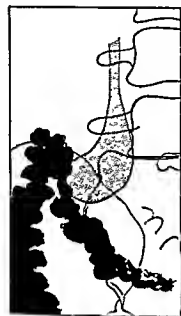


Fig. 760.—GALL-BLADDER OF A HYPOSTHENIC PATIENT: PENDULOUS TYPE WITH THE FUNDUS POINTING UPWARDS AND LYING BELOW THE ILIAC CREST. NOTE THE RELATION OF THE GALL-BLADDER TO THE BARIUM FILLED COLON.

(From Bull's "X-ray Interpretation.")

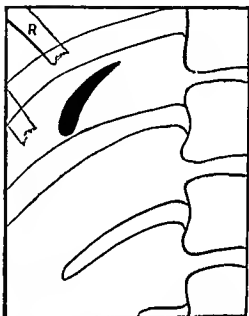


Fig. 761.—NORMAL GALL-BLADDER SHOWING UNIFORM CONTRACTION AFTER FOOD. HIGH GALL-BLADDER IN A PATIENT OF HYPERSTHENIC HABITUS.

(From Bull's "X-ray Interpretation.")

the gall-bladder in these patients is of poor tone and the dye pools in the fundus; after food it shows concentration and poor but uniform contraction, so that the sides and neck of the gall-bladder may be outlined.

In the average patient the gall-bladder is seen as a pear-shaped shadow projecting an inch or two below the liver margin, and lying two or three inches from the spine in the right mid-abdomen. Its respiratory excursion is that of the diaphragm and liver, and the fundus is the lowest point. A gall-bladder may be displaced so that the fundus points a little outwards, especially in fat patients, but when it points inwards it is drawn there by adhesions.

Filling and emptying indicates the health and functional activity

of the gall-bladder. *Filling* shows that the cystic duct is patent and that the gall-bladder has potential space and is not so filled with stones that no dye or hile can enter; it also implies that the common bile-duct is patent since no dye would pass into an over-distended organ. *Emptying* confirms that the ducts are patent, but—most important of all—shows the state of the gall-bladder walls. The healthy gall-bladder contracts uniformly and shows a clear outline surrounding a greatly diminished volume (fig. 761), whereas the rigid walls of chronic cholecystitis are unable to contract at all, a certain amount of hile passes out—perhaps by siphonage—and the shadow is fainter, but there is no contraction of the walls.

*Failure to fill.* Whereas filling shows patency of the ducts and potential space in the gall-bladder, failure to fill implies obstruction of the cystic duct or a gall-bladder full of stones, provided that absorption failure has been ruled out by intravenous injection, and that the functions of the liver are not seriously deranged. In jaundice obstructing the smaller bile-capillaries or the hepatic and cystic ducts, little or no dye can reach the gall-bladder, and in obstruction of the common duct no hile can leave it. Apart from this the liver must be seriously diseased and the patient gravely ill to prevent dye passing through the liver; multiple metastases of carcinoma do not necessarily preclude a filling of the gall-bladder.

*Concentration.* Often the dye appears more concentrated in the smaller volume of the contracted gall-bladder, whereas the shadow in the uncontracted gall-bladder of cholecystitis is always fainter after food. Although concentration is a property of the normal gall-bladder, any visible increase in density probably results from tonic contraction emphasising the shadow, since the amount of concentration which can occur in the few hours of observation must be small to appreciate in terms of radiographic density. As a test of the absorptive properties of the mucous membrane of the gall-bladder, therefore, we should not interpret too much from cholecystography.

*The outline* of the normal gall-bladder is clearly defined and describes an unbroken curve down the sides and round the fundus and fades away towards the neck; irregularities in this line are usually due to adhesions or to cholecystitis with thickening and deformity of the walls, but a certain amount of distortion can also be produced by pressure in thin patients, such distortion being easily recognised and not constant in films taken in different positions.

The density is uniform except where overlapped by the shadows of surrounding structures of unequal density, and it is the object of radiography to project the gall-bladder so clear of overlapping shadows that the integrity of form and density is beyond doubt. The density appears to be about that of ground glass, filling-defects due to non-opaque calculi are seen as dark circular areas, and gas in superimposed bowel is somewhat similar.

#### STRUCTURES WHICH CONFUSE THE GALL-BLADDER OUTLINE

The adjacent structures which have to be recognised and thrown clear of the gall-bladder shadow by suitable radiographic technique are:

(1) *Hepatic flexure of the colon.* This always contains some gas, and can be very troublesome in the hypersthenic patient in whom the gall-bladder is pressed upwards by a distended colon, or in the hyposthenic in whom the gall-bladder lies to the inner side of the hepatic flexure. However, the gall-bladder bears a constant anatomical relation to the colon, namely, that the fundus of the gall-bladder lies

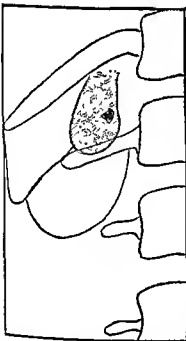


Fig. 762.—CALCULUS IN THE PELVIS OF THE RIGHT KIDNEY SUPERIMPOSED ON THE SHADOW OF THE DYE-FILLED GALL-BLADDER. THE RELATIVE POSITIONS OF THE SHADOWS ALTER IN THE OBLIQUE AND LATERAL POSITIONS.

(From Bull's "X-ray Interpretation.")

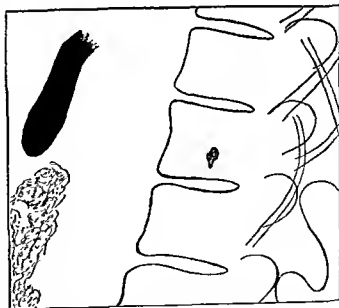


Fig. 763.—LATERAL VIEW OF FIG. 762. THE KIDNEY STONE IS SEEN IN THE SHADOW OF THE BODY OF THE LAST THORACIC VERTEBRA, THE GALL-BLADDER LIES IN FRONT AND IS SLIGHTLY ANTERIOR TO THE HEPATIC FLEXURE OF THE COLON.

somewhat anterior. These structures can be thrown clear of one another by a left anterior oblique view, or in extreme hyposthenic patients by a lateral.

(2) *Gas in the duodenum*, when it occurs, is often in the form of a small chain, and has a resemblance to the filling-defects of non-opaque calculi; confusion would only arise if a single film were taken, as films taken in different positions show the transient nature of these shadows.

(3) *Overlapping shadows of bones*. The vertebrae with their transverse processes, the lower ribs and calcified costal cartilage, and the ilium in extreme visceroptosis may overlap the gall-bladder shadow in the different positions in which it may lie. They can be separated from one another by films in the oblique position or in different phases of respiration; a thinly filled gall-bladder lying somewhat centrally over the spine may be almost invisible until an oblique position is used.

(4) *Calcifications in other organs, etc.* A single calculus in the pelvis of the right kidney, calcified mesenteric or lumbar glands, warts, plaster strapping, clothes, or other objects on the skin—all of these throw shadows which may interfere with interpretation if they happen to be projected into the gall-bladder shadow. As in the other examples of overlapping shadows, they can mostly be differentiated by oblique views or films in different phases of respiration, but in any case in which the shadows lie close together and cause real difficulty in diagnosis the same principle of technique applies as in all X-ray work, i.e. two films at right angles—a postero-anterior and a lateral.

#### PATHOLOGICAL CONDITIONS OF THE GALL-BLADDER

##### 1. CHOLECYSTITIS

(a) *Acute cholecystitis* with catarrhal swelling of the cystic duct does not allow filling of the gall-bladder. In the same way obstructive jaundice is a contra-indication to cholecystography, since there is obstruction to the flow of the bile and therefore no dye will reach the gall-bladder.

(b) *Chronic cholecystitis*. Chronic cholecystitis leads to thickening and rigidity of the gall-bladder walls, and the organic changes produced by chronic cholecystitis are recognised by cholecystography.

A gall-bladder which has been inflamed may become adherent to



surrounding structures and show a slightly altered position. Normally the fundus of the gall-bladder is in the axis of the body; in hypersthenic patients the fundus may be pushed forwards or a little outwards, but deflection inwards is due to adhesions; such adhesions are commonly to the duodenum, and the concave shadow where the fundus of the gall-bladder indents the duodenum can sometimes be shown by barium meal (fig. 764).

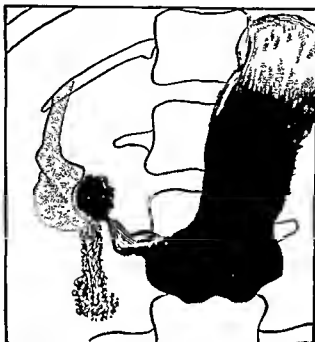


Fig. 764.—CHRONIC CHOLECYSTITIS WITH ADHESION TO THE DUODENUM. THE GALL-BLADDER HAS FILLED BUT THE OUTLINE IS DEFORMED AND THERE IS A CONCAVE DEPRESSION WHERE IT IS ADHERENT TO THE BARIUM FILLED DUODENAL CAP. NOTE THE REFLEX SPASM OF THE PYLORIC END OF THE STOMACH.

(From Ball's "X-ray Interpretation.")

Irregularity of outline due to scarring of the walls is another indirect sign of chronic cholecystitis; thickening of the mucosa tends to blur the normal sharp outline.

But the most significant sign of chronic cholecystitis is total failure of contraction after a full meal; the normal gall-bladder empties and shows uniform contraction, the gall-bladder of cholecystitis empties to some extent—indicated by a fainter shadow—but does not contract at all.

## 2. GALL-STONES

Gall-stones are classified pathologically according to their composition, but from the X-ray point of view the broad classification is: *gall-stones which throw a shadow and those which do not.*

Most gall-stones contain some calcium, but it is only when the calcium is concentrated in one layer or is high in percentage that a shadow is seen without the aid of cholecystography. Such gall-stones constitute less than 5 per cent of all cases.

Whether or not gall-stone shadows are recognisable on a plain X-ray film, cholecystography is also desirable because it gives information as to the functional activity of the gall-bladder, the patency of the cystic duct, and the condition of the walls.

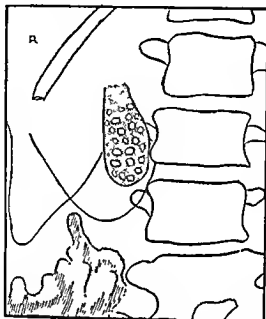


Fig 765.—MULTIPLE FACETED STONES IN A DYE FILLED GALL-BLADDER. EACH OF THE STONES HAS A THIN SURROUNDING RING OF CALCIUM.

(From Bull's "X-ray Interpretation.")

The gall-stones which contain calcium in high percentage show as shadows of increased density through the dye filling the gall-bladder. They may be large or small, single or multiple. Sometimes when the calcium is distributed evenly their density is uniform throughout, at other times calcium is concentrated in a layer round the gall-stones, forming ring shadows with relatively translucent centres; the ring shadow type is often multiple.

Radio-lucent gall-stones have a density less than that of the dye in the gall-bladder, and therefore appear as multiple faceted filling-defects in the gall-bladder shadow.

Another type of solid deposit in the gall-bladder is "gall sand"; this deposit may contain a high percentage of calcium, and is then

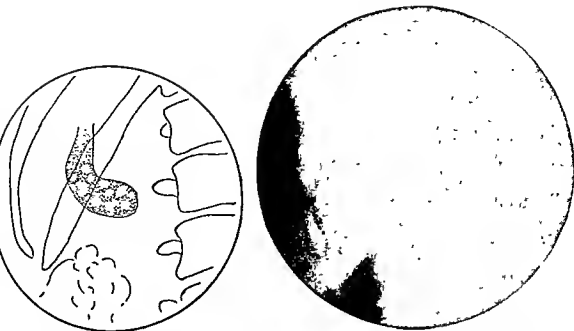


Fig. 766.—MULTIPLE "NEGATIVE" SHADOWS IN A DYE-FILLED GALL-BLADDER. THESE STONES ARE RADIO-  
LUCENT AND THEIR PRESENCE IS ONLY SHOWN BY THE FILLING-DEFECTS IN THE GALL-BLADDER SHADOW. NOTE  
THAT THE FUNDUS OF THE GALL-BLADDER IS DRAWN TO THE MID LINE INDICATING ADHESIONS.

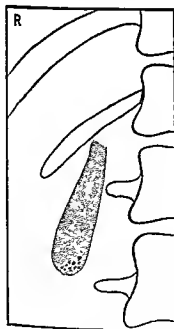


Fig. 767.—CHOLECYSTOGRAPHY. PIN-  
POINT OPACITIES IN THE FUNDUS OF  
THE GALL-BLADDER DUE TO SMALL  
STONES CONTAINING CALCIUM.  
(From Bull's "X-ray Interpretation.")



Fig. 768.—DEFORMITY OF THE DUODENAL CAP  
RESULTING FROM ULCERATION OF A GALL-  
STONE INTO THE DUODENUM. THERE IS A  
FISTULOUS CONNECTION BETWEEN THE DUO-  
DENUM AND THE GALL-BLADDER, AND BARIUM  
IS SEEN TRACKING ALONG THE HEPATIC AND  
COMMON BILE DUCTS.

(From Bull's "X-ray Interpretation.")

seen as multiple pin-point radio-opaque spots in the fundus of the gall-bladder (fig. 767).

The gall-bladder which contains stones does not always fill with dye, or at least with enough dye to show a shadow outline; this failure to fill may be due to the gall-bladder being so packed with stones that insufficient dye can enter, or to one stone rolling into the orifice of the cystic duct and causing temporary obstruction, or to swelling or fibrosis blocking the cystic duct.

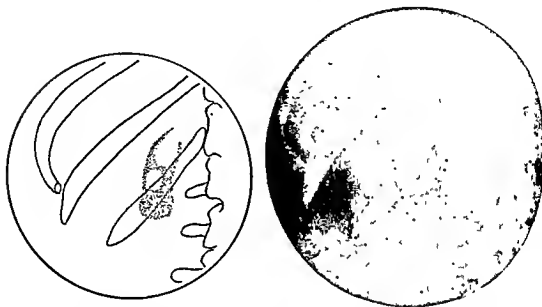


Fig 768.—BILOCULAR GALL-BLADDER WITH TWO RADIO-LEUCENT CALCULI IN THE UPPER LOCULE.

Failure of a gall-bladder to fill after intravenous injection indicates either a gall-bladder filled with calculi, or mechanical obstruction of the cystic duct—provided that jaundice or gross disease of the liver is excluded.

Gall-stones are frequently associated with chronic cholecystitis, but in some cases in which the stones are few or amounting only to gall sand there is no evidence of impairment of gall-bladder function; the gall-bladder fills well, showing a normal outline, and revealing the stones as filling-defects in its density, and when re-examined after food the walls have contracted uniformly and surround the gall-stones more closely. In these cases a good deal of mobility can be seen in the gall-stones if the patient is examined in the erect and prone positions.

Sometimes an inflamed gall-bladder adherent to the duodenum actually perforates and discharges its contents into the alimentary

tract. In these cases no shadow is obtained by cholecystography, but barium meal shows a deformed duodenum and thin branching shadows extend upwards from the duodenum into the liver outlining the hepatic and bile ducts (fig. 768).

A variation in the shape of the gall-bladder which may be congenital or a sequel to inflammation is an hour-glass contraction dividing it into two loculi. The gall-bladder may contract well after food or may contain stones (fig. 769).

Besides being found in the gall-bladder, stones may be seen in the hepatic ducts or in the common bile-duct; the latter is uncommon. Stones in the hepatic ducts are more often seen as a recurrence following cholecystectomy. Cholecystography is now of no service, and plain films are taken of the liver in the postero-anterior and lateral positions. It is fortunate for diagnosis that these stones more often than not contain a high percentage of calcium, and they are seen as opaque shadows lying well above the lower edge of the liver in the postero-anterior film and lying anterior to the vertebral bodies in the lateral position.

### 3. NEW GROWTH

In chronic cholecystitis a thickened mucous membrane may develop polypoid overgrowths which may be recognised as rounded projections from the gall-bladder walls into the density of the shadow. Papillomata can also occur.

Small papillomata and adenomata also occur and are not uncommon. They are characterised by single small circular filling-defects which are usually seen best after the gall-bladder has contracted and emptied partially after a meal. These filling-defects are constant in all positions, and are often seen high in the fundus shadow—an unlikely position for a single small calculus.

Carcinoma can take origin from the mucous membrane of the gall-bladder; jaundice is an early sign, and these cases do not often come to X-ray examination.

## SECTION 2

### RADIOLOGY OF THE URINARY TRACT

#### NORMAL APPEARANCES

*Kidneys.* In the patient of average build, the kidney shadows extend from the last thoracic to the third lumbar vertebra and the vertical axes incline at an angle of about  $30^{\circ}$  to the spine, being more or less parallel to the ilio-psoas muscle. The right kidney is usually half an inch lower than the left, but this is not constant. The left kidney can be lower than the right without being abnormal—the common causes being scoliosis, enlargement of the spleen, and a kidney which is anatomically larger than its fellow. It is always well to determine the cause when one finds the left kidney lower than the right since it may be pathological and the first indication of a renal tumour.

*Ureters.* The ureters are non-opaque and cannot be seen unless filled with a contrast fluid. They have a small field of mobility, but the line of each ureter can be described with fair accuracy as a vertical line from the pelvis of the kidney to the brim of the bony pelvis, crossing the transverse processes of the lower lumbar vertebrae in its descent. At the brim of the bony pelvis the line curves slightly outwards and finally inwards to reach the bladder at about the level of the ischial spine.

*Bladder.* The dome of the bladder varies in height according to distension, and in shape according to pressure from the pelvic viscera, but the normally contracted bladder which is not obscured by shadows in the rectum shows a slightly convex dome above the shadows of the pubic rami, the sides curving down to the base which lies just behind the symphysis.

#### NORMAL PYELOGRAM

The kidney pelvis, ureters, and bladder are outlined with sodium iodide injected from below (ascending pyelography), or with an intra-

venous injection of a pyridine derivative combined with iodine (descending pyelography).

The outlines of the normal ureters and bladder in pyelography are as described above. The outline of the kidney pelvis requires more description since there are so many variations of the normal that it is important to know the different forms to avoid confusion with the pathological.

The upper end of the ureter expands rapidly in wide curves to form the kidney pelvis; in the kidney of average position the pelvis is set



*Fig. 770.*—ASCENDING PYELOGRAM OF NORMAL KIDNEY SHOWING THE URETER, PELVIS, MAJOR AND MINOR CALICES.

almost at right angles to the ureter; in the high kidney the angle may be greater than a right angle, and in the kidney of visceroptosis the angle is acute, often with a secondary flexure near the uretero-pelvic junction. The lower boundary of the pelvis curves downwards and outwards, the upper boundary upwards and outwards. Laterally from the pelvis project the major calices—tubular branches with more or less parallel sides; the major calices are commonly three in number, the upper one being longer and more independent than the lower two; but the number can vary from two to five; among the larger numbers one is often quite small and appears rudimentary.

From the major calices project the minor calices—bell-shaped like

small kidney pelves and with horseshoe terminations when seen in profile, the horseshoe depressions being due to the imprints of the renal papillæ. Several minor calices project from each major calyx, and they point outwards (when they are seen in silhouette), backwards, and forwards.

The relative sizes of kidney pelves and calices are variable; sometimes the pelves are long and narrow—being little more than dilated ureters leading into long major calices (fig. 771), or conversely a pelvis may be fully rounded with major calices negligible, and the minor calices



*Fig. 771 — INTRAVENOUS PYELOGRAM SHOWING A NORMAL KIDNEY WITH SMALL PELVIS.*

opening directly into the pelvis (fig. 772). Any intermediate varieties may occur, but the form and outline of the kidney pelvis and calices on one side usually follow the same general pattern of those on the other side, so that a pyelogram which shows two very dissimilar kidney pelves suggests that one of the two is pathological.

Besides this point of dissimilarity, the signs of pathology in a pyelogram are :

- (1) Dilatation of the pelvis and calices—from partial obstruction.
- (2) Deformity of the pelvis and calices—from new growth and chronic infection.
- (3) Communication between the calices and parenchyma indicating tissue destruction—tuberculosis.



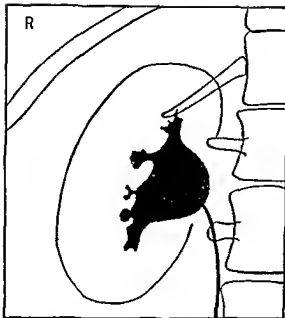


Fig. 772.—PYELOGRAM OF NORMAL KIDNEY OF THE TYPE WITH LARGE PELVIS AND SHORT CALICES. THERE ARE FIVE CALICES, ONE OF WHICH IS RUDIMENTARY.  
(From Bull's "X-ray Interpretation.")

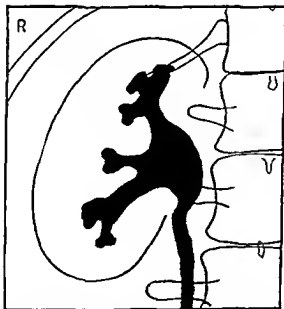


Fig. 773.—HYDRONEPHROSIS. THERE IS DILATATION OF THE MAJOR AND MINOR CALICES: THE MINOR CALICES HAVE LOST THEIR HORSESHOE OUTLINE AND BECOME ROUNDED: THE URETER IS DILATED BUT THERE IS LITTLE DILATATION OF THE PELVIS.

(From Bull's "X-ray Interpretation.")

- (4) Forward rotation on the vertical axis—horseshoe kidney, dystopic kidney.
- (5) Displacements—congenital dystopia, extrarenal tumour, and spinal curvature.

## I. THE KIDNEYS

### PATHOLOGICAL CONDITIONS

(1) *Hydronephrosis* is distension by fluid within the kidney pelvis, and the X-ray signs in the pyelogram are dilatation of the pelvis and calices with obliteration by distension of the fine horseshoe points of the minor calices. An intravenous pyelogram further shows a prolonged excretion time through the affected kidney.

The amount of dilatation varies with the nature of the obstruction and the length of time it has been operating; the relative dilatation of pelvis and calices is also variable—long-standing chronic infections of the kidney pelvis with narrowing and partial obstruction of the lower ureter produce the largest dilatations of calices, pelvis and ureter, whereas high obstruction of the ureter causes relatively more dilatation of the kidney pelvis. (Braasch & Hoyer, *Urography*, 1927. Phila. and London.)

(2) *Calculus*. The majority of renal calculi which are too large to pass down the ureters are radio-opaque, and also they are of uniform density and rounded outline; very large calculi are moulded to the shape of the pelvis and calices they fill.

In antero-posterior view of the kidney the calculus is lying in the part of the kidney shadow occupied by the pelvis and calices; in lateral view the shadow of a renal calculus lies within that of the vertebral bodies, and is thus distinguished from gall-stones and calcified mesenteric glands which lie anteriorly (see fig. 763).

A pyelogram shows the exact position of a calculus within the kidney and at the same time reveals kidney damage—evidenced by hydronephrosis or local dilatation of an affected calyx (fig. 774).

(3) *Tuberculosis*. Tuberculosis is a destructive inflammatory disease, and the changes from normal which we see in the pyelogram are the results of tissue destruction, fibrotic reaction, and calcification.

The characteristic of the pyelogram of active tuberculous pyelonephritis is an extension of the opaque medium into the kidney substance indicating that a tuberculous abscess in the parenchyma has

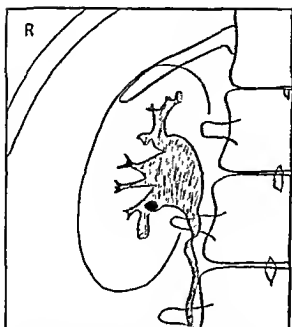


Fig. 774.—PYELOGRAM WITH A WEAK SOLUTION TO SHOW A RADIO-OPAQUE CALCULE THROUGH THE IODIDE SHADOW. EXCEPT FOR SLIGHT BROADENING OF THE LOWER MAJOR CALYX THE KIDNEY APPEARS NORMAL.  
(From Bull's "X-ray Interpretation.")

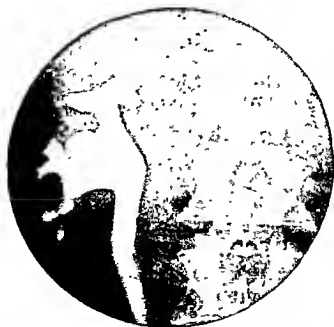


Fig. 775.—TUBERCULOSIS OF THE KIDNEY. THE AFFECTED CALICES IN THE UPPER AND LOWER POLES SHOW IRREGULAR DILATATION AND CONTRACTIONS, AND THE OPAQUE MEDIUM HAS PASSED OUT INTO THE KIDNEY SUBSTANCE.

discharged itself into the pelvis leaving an abscess cavity (fig. 775). This is rarely seen in pyelonephritis of any origin other than tuberculosis. The extension of the shadow beyond the calices is ragged and blurred, and the affected calices may show local dilatation, but the outlines of the unaffected calices and the pelvis remain normal.

In long-standing cases the calices may show irregular dilatation and contractions, and the pelvis may also show an irregular contracted outline leading out to a gaping dilated ureter. This is the natural sequel to chronic inflammatory thickening followed by fibrosis and contraction.

Calcium is deposited when a tuberculous abscess has been successfully walled off by fibrosis. Fine dots and streaks of calcium can often be seen in the kidney shadow if sought for in a film of good detail, and if this is compared with the subsequent pyelogram they will be found to correspond to diseased areas in the calices. Extensive chronic tuberculous pyelonephritis with secondary infection may show large calcifications filling the pelvis and calices. Occasionally the ureter becomes sealed off, causing the kidney to die and become calcified.

#### CONGENITAL ANOMALIES

The developmental anomalies which are found in the kidneys and revealed by pyelogram are :

- (1) Fusion of the kidneys—horseshoe kidney.
- (2) Arrested migration—renal dystopia.
- (3) Double pelvis and ureters.
- (4) Congenital polycystic kidney.

(1) *Horseshoe kidney.* The two kidneys may be lying in their normal position but joined together by a bridge of tissue between their lower poles, or they may be lying above one another on the same side of the abdomen. The kidneys are rotated forward on their vertical axes so that a pyelogram shows most of the calices superimposed on the shadows of the pelvis, with some minor calices projecting from the medial sides of the pelves and a few projecting in a normal manner from the lateral aspects—according to the degree of rotation (fig. 776). The ureters therefore descend from the middle or lateral aspects of the pelvic shadows in the antero-posterior view and curve inwards to reach their normal course over the transverse processes of the lower

lumbar vertebrae. When the two kidneys are fused and lying on the same side, the ureter of the lower kidney crosses the spine obliquely to reach its normal position.

(2) *Dystopic kidney*. In this anomaly one kidney—more frequently the left—has become arrested in its migration out of the bony pelvis and is found in the sacro-iliac region.

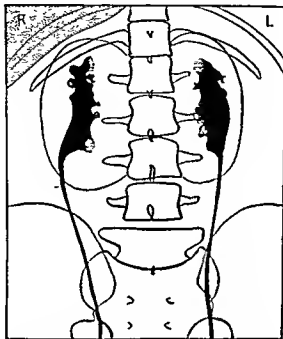


Fig. 776.—DOUBLE PYELOGRAM OF A HORSESHOE KIDNEY. MOST OF THE CALICES ARE ON THE MEDIAL SIDES AND THE URETERS LEAVE FROM THE LATERAL ASPECTS OF THE KIDNEYS.

(From Bull's "X ray Interpretation.")

A pyelogram shows that the ureter is short, and that the kidney is rotated forwards and widely separated from its fellow, thus distinguishing it from horseshoe kidney—in which the kidneys are necessarily close together—and visceroptosis, in which both kidneys are relatively low and the ureters redundant. Like all the congenitally abnormal kidneys the dystopic kidney frequently becomes pathological.

(3) *Double pelvis*. It was noted under "Normal Appearances" that the major calyx draining the upper pole was relatively long and almost independent. Sometimes this independence is complete and the kidney pelvis is actually divided into a small upper pelvis draining the upper pole and a larger lower pelvis draining the lower two-thirds. Beyond the pelvis the ureter may be duplicated and either joins into a single one lower in its course, or two ureters may run the whole

length together and open separately above one another into the bladder (see fig. 780).

(4) *Congenital polycystic kidneys.* An X-ray film of the abdomen shows that both kidneys are enlarged. A pyelogram shows that the pelves and calices in both kidneys are deformed. These are the points in the X-ray examination which—taken with the clinical evidence—point to a diagnosis of polycystic kidneys. It is better in these difficult cases to use an ascending pyelogram which ensures filling of the pelvis and calices with good definition.



Fig. 777.—CONGENITAL POLYCYSTIC KIDNEY: THE PELVIS IS NARROWED, THE MAJOR CALICES SHOW THE EFFECTS OF COMPRESSION, AND SOME OF THE MINOR CALICES ARE ENLARGED AND BELL-SHAPED.

The deformity of the pelvis and calices is deformity of compression—resulting from the growth of the cysts; there is narrowing and a certain irregularity in contour of the pelvis, and narrowing and elongation of the calices, which are often drawn out into narrow channels. The normal form of the minor calices is lost, they may be obliterated or may be enlarged and show an exaggerated bell formation (fig. 777); but the kidney pelvis—although narrowed—does not become obliterated.

#### TUMOURS

The list of tumours of the kidney is a short one, and except for small fibromata and adenomata which do not cause symptoms and are

not diagnosed during life all tumours of the kidney are malignant. They are :

- (1) The Grawitz tumour or hypernephroma.
- (2) The embryoma or Wilms tumour—occurring in infants.
- (3) Papilloma of the renal pelvis.

Tumours of the adrenal cortex cause apparent enlargement of the kidney by displacement, but they do not often invade the kidney substance, and even then cause little deformity of the pyelogram considering the size of the tumour mass.



Fig. 778.—HYPERNEPHROMA. THE PELVIS IS COMPRESSED AND THE CALICES MORE OR LESS OBLITERATED. THERE WAS A SECONDARY GROWTH IN THE ISCHIUM.

(1) *Hypernephroma*. X-ray examination shows that the kidney is relatively enlarged, and since the tumour often occupies one pole, the swollen outline may be apparent.

A pyelogram shows deformity of the calices and pelvis where they are pressed upon by the growing tumour. The appearance is somewhat similar to that of polycystic kidney—the calices being distorted and drawn out into slender channels with bulbous ends. In the affected area of the kidney the calices are ultimately obliterated (fig. 778); later the increasing pressure of growth may obliterate the pelvis, and an ascending pyelogram shows that no iodide passes beyond the upper end of the ureter. This and the unilateral deformity with a normal

kidney on the other side are two points which distinguish hypernephroma from congenital polycystic disease.

Hypernephroma is very malignant, and metastases occur early to the lungs and bones through the blood stream. It follows that the chest and any suspected bones should be X-rayed before radical surgery is undertaken.

(2) *Embryoma*. These tumours occur only in infants—sometimes even before birth—they are often bilateral, and the diagnosis is made on clinical evidence.

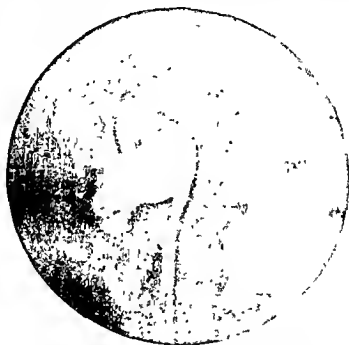


Fig. 779.—PAPILLOMA OF THE RENAL PELVIS CAUSING LOCALISED PRESSURE DISTORTION OF THE PELVIS AND UPPER CALICES.

(3) *Papilloma of the renal pelvis*. This tumour may exist for a considerable time without showing evidence of malignancy. X-ray examination of the kidneys shows no enlargement, but a pyelogram may show a constant filling-defect in the pelvic shadow and a degree of hydronephrosis—depending on the size and position of the papilloma.

The conjunction of clinical and X-ray evidence is important in the diagnosis of this tumour. Painless hæmaturia which the cystoscope shows to be coming from one kidney, and a small filling-defect in the opaque shadow of the renal pelvis—with or without hydronephrosis—are corroborative and make an almost certain diagnosis where the X-ray evidence alone is slender.



## II. THE URETERS

### PATHOLOGICAL CONDITIONS

- (1) Dilatation. ~.
- (2) Congenital anomalies.

(1) *Dilatation.* The common cause of dilatation of a ureter is the partial or intermittent obstruction of a stone; another cause is chronic inflammation, pyogenic or tuberculous, and a third is the pressure of an abdominal or pelvic tumour.

X-ray examination is usually made after an attack of renal colic; the majority of calculi are radio-opaque, and the rounded or oval shadow is seen in the line of the ureter—often in the ampulla at the level of the ischial spine. However, small calculi lying over the shadow of the sacrum may be invisible and a negative X-ray of the urinary tract should not be accepted where there is clinical evidence of a calculus passing down the ureter. In all these cases it is desirable to make an intravenous pyelogram to show the dilatation of the ureter and distension of the kidney pelvis and calices above the obstruction and the relative delay in excretion through the kidney; and if the calculus is radio-lucent the outlining of the dilated ureter above indicates the point of obstruction.

Tuberculous pyelonephritis early leads to infection around the ureteric orifice in the bladder, and in the later stages to thickening and dilatation of the ureter with a rigid gaping orifice into the bladder through which the iodide—introduced for a cystogram—flows. This is also seen in chronic cystitis from pyogenic organisms. It is inflammatory dilatation without obstruction, and the X-ray characteristic is the passive regurgitation from the bladder.

Abdominal and pelvic tumours which cause ureteric obstruction are usually of such size and nature as to direct attention to themselves before they produce the secondary signs of ureteric obstruction. Of the benign tumours the commonest is the pregnant uterus, and of malignant tumours carcinoma of the cervix.

(2) *Congenital anomalies.* The renal pelvis may be double and drained by twin ureters; these may join in any part of their course—often they join soon after leaving the kidney—or may run independent

courses down to the bladder which they enter close to one another. The full relationship of double ureters to one another—and how they cross in their descent and finally open into the bladder with the ureter

from the lower kidney section opening behind and laterally to that from the upper—is best seen by passing opaque catheters up the two ureters and taking stereoscopic films (fig. 780). The anomaly of double ureters may or may not be bilateral. Like all congenital anomalies of the urinary tract they are especially liable to pathological changes.



Fig. 780.—ASCENDING PYELOGRAM OF KIDNEY WITH DOUBLE URETERS.

### III. THE BLADDER

#### NORMAL APPEARANCES

The faint shadow of the bladder is seen when the rectum is cleared of gas and feces; the superior surface is convex or flattened, and the lateral walls parallel to the pubic rami. In the soft tissues on either side of the bladder are seen a number of small circular opacities—phlebs—of unexplained origin and no known significance. In elderly male patients multiple pin-point opacities behind the symphysis pubis are calcifications in the prostate.

To outline the bladder, 2 per cent sodium iodide is injected through the urethra; or the natural filling and progressive distension of the bladder can be followed with a series of films after intravenous injection with one of the pyelogram solutions—pyridine derivatives combined with iodide.

## PATHOLOGICAL CONDITIONS

(1) *Calculi* lying free in the bladder are seen on the floor of the bladder near the mid-line; they are more often single than multiple, rounded in outline, radio-opaque, and uniform in density. When found in other parts of the bladder they are either in diverticula or they have formed round a foreign body such as a suture which has perforated the bladder wall.

(2) *Inflammation*. Simple cystitis causes no change in the bladder outline. Almost the only inflammatory disease which leads to thickening, rigidity, and contraction of the walls is tuberculosis. A cystogram shows a total contraction in volume and an irregularity of outline due to the thickening and fibrosis which occur in this disease. The ureteric orifices are affected early, and they also become rigid and patulous, allowing the opaque medium to flow up from the bladder and outline the dilated lower ureters.

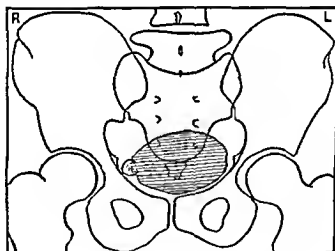


Fig. 781.—CYSTOGRAM SHOWING DIVERTICULUM FROM THE RIGHT LATERAL ASPECT OF THE BLADDER.  
(From Bull's "X-ray Interpretation.")

(3) *Diverticula* occur most often from the lateral aspects of the bladder, and are seen as rounded buds projecting from the cystogram shadow. A form of pseudo-diverticulum is the sacculation which occurs behind an enlarged prostate and retains urine after the bladder has contracted. In these cases the shadow of the prostate shows as a filling-defect in the base of the cystogram, and after emptying the bladder the iodide retained behind the prostate shows a semilunar shadow.

(4) *New Growth.* The bladder is lined by transitional epithelium and the only primary new growth which occurs with any frequency is a primary tumour from the epithelium. It begins as a papilloma which may be villous or sessile and which is ultimately malignant (fig. 782).

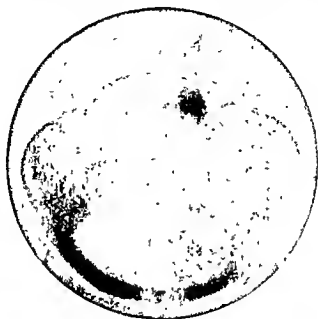


Fig. 782.—INTRAVEINOUS UROGRAM SHOWING FILLING DEFECT IN THE BLADDER DUE TO A LARGE PAPILOMA.

Small papillomata are not often recognised through the cystogram, but when carcinoma spreads and involves a considerable area of the bladder wall the filling-defect is readily identified, and by taking stereoscopic films the position of the tumour in the bladder can be determined.

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PART V  
RADIUM TREATMENT

SECTION 1  
RADIUM TREATMENT OF MALIGNANT DISEASE  
by  
STANFORD CADE

SECTION 2  
RADIO-THERAPY IN DISEASES OF WOMEN  
by  
MALCOLM DONALDSON

## SECTION 1

# RADIUM TREATMENT OF MALIGNANT DISEASE

by

STANFORD CADE

THE spontaneous emission of energy which is one of the chief properties of radio-active bodies has been used in the treatment of malignant disease since the beginning of the present century. In the clinical use of radium some knowledge of the physical factors governing the production of this energy is essential.

The complexity of the problems of radiation therapy necessitates a brief account of the most important physical facts bearing on therapy.

### TYPES OF RADIATION

The importance of radium and other radio-active bodies is due to their property of emitting rays and their consequent transformation into other elements. Radio-active elements are composed of heavy atoms, and emission of radiation is accompanied by an atomic transformation. The radiations are of three kinds possessing characteristic properties. Rutherford distinguished the three types as :

Alpha rays ( $\alpha$ ).

Beta rays ( $\beta$ ).

Gamma rays ( $\gamma$ ).

The alpha ray is a material particle ; it is the nucleus of a helium atom possessing a double positive charge, and is deviated in an electromagnetic field. Alpha particles are ejected at a speed of 9,000 to 20,000 miles per second ; in their passage through matter they quickly slow down and by annexing two electrons they become indistinguishable from helium atoms. Their range through air varies from 2.7 cm. to 8.6 cm., and they are arrested by a sheet of paper. They cannot be used therapeutically owing to their poor power of penetration.

The beta rays are also material particles; they are of the same type as cathode rays, namely, negative electrons. Like alpha particles they are ejected from the nucleus of the atom, but at a much greater velocity (186,000 miles per second). Their penetrating power is much greater than that of  $\alpha$  particles. The fastest beta particles penetrate 1 cm. of tissue; they are completely stopped by 0.5 mm. of gold; according to their power of penetration three groups are recognised: the "soft," "medium," and "hard" beta rays. The importance of this fact appears in connection with the question of filtration.

Gamma rays are true rays, comparable with Röntgen rays or visible light. They are of very short wave-length and great penetrating power: the hardest gamma rays are one hundred times more penetrating than the beta rays, and can pass through 25 cm. of lead. Gamma rays represent 4.8 per cent of the total energy of radium. They are never emitted alone by any radio-active substance, but together with beta rays. Their speed is that of light, 186,000 miles per second. One distinguishes "hard" and "soft" gamma rays; the soft rays are absorbed by 2 mm. of lead. When passing through tissues, gamma rays give rise to secondary  $\beta$  rays of therapeutic value.

#### PRODUCTS OF RADIUM

To understand the disintegration of radio-active elements it must be remembered that radio-active elements consist of a very large number of atoms of which only a small fraction explode at any one time. Although it is impossible to know how long any single atom will exist as radium, it is possible to determine the average life of the atoms. The average life of a radium atom is 2,900 years. In a radium preparation free from preceding members of the series, the amount of radium diminishes gradually, at such a rate that in 1,690 years one half of it will have disappeared. The remaining half behaves in a similar manner, and loses half its value in another 1,690 years. Each radio-active element has a definite *half value* period; these differ from a fraction of a second to several million years.

*Radon* or *radium emanation* is one of the products of disintegration of radium, and has a half value period of 3.85 days. One atom of radium, having an atomic weight of 226, expels an alpha particle and is transformed into an atom of radon with an atomic weight of 222. If we remember that an alpha particle is a helium nucleus, and that helium is an inert gas, we realise that the spontaneous disintegration of an active chemical element, a metal, has given rise to two inert

gases, one of heavy atomic weight (radon, 222) and the other very light.

Radon is chemically inert and transforms itself into radium A and helium by the expulsion of one alpha particle from each atom of radon. Radium A is a solid metal, with an atomic weight of 218. In its turn, radium A expels helium and becomes radium B. The latter undergoes a further change of a different type, accompanied by the production of beta and gamma rays. Further transformations occur, and in the end lead is obtained.

Radon was first identified as a gas by Rutherford in 1900. As it is an inert gas it can be separated from its parent substance, radium, by simple physical processes. For medicinal purposes the preparation of radon involves the following operations: (1) Separation of radon from the parent solution of a radium salt by means of suitable extraction apparatus. (2) Purification of radon to obtain the maximum concentration. (3) Introduction of the purified radon into glass capillary tubes. (4) Measurement of the containers. (5) Screenage by suitable metal, gold or platinum.

#### CHOICE BETWEEN RADON AND RADIUM

Whether radon gas or radium salts in needles are used the biological effects are due in each case to the gamma rays and secondary beta rays obtained. There are, however, certain advantages and disadvantages peculiar to each, and these may be briefly stated.

From the qualitative point of view there is no difference, but as radon's half life period is 3.85 days only, it is impossible to give a prolonged treatment of uniform intensity with radon.

The advantages of radon are as follows:

- (1) Possibility of varying the charge in the tubes or needles at will, without limit.
- (2) Distribution of the radon day by day according to needs.
- (3) No risk of loss.
- (4) Possibility of ambulatory treatment.
- (5) Loss of container within a patient is of no great importance.

The advantages of radium salts are as follows:

- (1) Absence of manipulation.
- (2) Avoidance of measurements.
- (3) No necessity for calculation on account of falling intensity.
- (4) Uniformity of intensity throughout the period of treatment.



## FILTRATION

The advantages of filtration have been known since the work of Dominici. The practical importance of filtration is now universally recognised and the tendency to increase filtration is more generally adopted. In treatment of malignant disease the object of filtration is to obtain gamma rays only. The elimination of primary beta rays is obtained by enclosing the radium salt or radon in a sheath of metal of high atomic weight capable of absorbing beta rays. Table I shows the screening power of various metallic substances. The best possible filter is platinum; 0.6 mm. of platinum absorbs 99.9 per cent of beta

TABLE I

Substance.	Density.	For Absorption of:	
		50% $\beta$ Rays.	99.9% $\beta$ Rays.
Aluminium. . . .	2.7	0.52 mm.	5.2 mm.
Brass . . . .	8.5	0.10 "	1.6 "
Copper . . . .	8.9	0.15 "	1.5 "
Silver . . . .	10.0	0.13 "	1.3 "
Lead . . . .	11.3	0.12 "	1.2 "
Gold . . . .	19.3	0.07 "	0.7 "
Platinum . . . .	21.5	0.06 "	0.6 "

rays. In the case of gamma rays filtration decreases the intensity of the long wave-length components to a greater extent than it does that of short wave-length radiation. Filtration, therefore, removes all primary beta rays, and a proportion of the softer gamma rays. It therefore raises the average of the penetrating power, at the expense of quantity. A filtered ray is, therefore, less intense but more penetrating. By increasing filtration to 0.8 mm. of platinum in interstitial radiation, risk of necrosis is diminished, the time factor can be prolonged, and hence the total tissue dose is raised. In their passage through matter gamma rays give rise to secondary radiation, spoken of as *secondary beta rays*. In external radium application these secondary beta rays produce effects which may influence the dose given and

should be screened. This secondary filter is obtained by the use of materials of low atomic weight; for this purpose rubber, celluloid, or bakelite are used with advantage and are interposed between the source of irradiation and the tissues treated.

#### NOTATION OF DOSAGE

There is so far no accepted unit of dosage in radium therapy. In England the term *milligram-hour* has been used by most radio-therapentists. It has been criticised as meaningless, but the alternatives suggested from time to time have failed to find general approval. Although by no means perfect, the term milligram-hour has definite use in clinical medicine provided the details of treatment—filtration, distance, method, time, size of field, number of units, strength of units, etc.—are specified. In all reference to dosage, calculations are made on the dose delivered; it should really be the dose received, or tissue dose which should be noted. The term “millicurie destroyed” used on the Continent is equally unsatisfactory. The “threshold skin erythema” has been adopted in the Memorial Hospital; an energy unit was put forward by Murdoch; both have their values, but bring us no nearer to a universally accepted unit expressing the energy absorbed by the tissues. Under the circumstances we will make use of the term *Mg. hour*, giving full details of the technique of treatment. To the trained mind this method of notation gives an accurate indication of what is being done and repetition of treatment is possible.

#### BIOLOGICAL EFFECTS OF RADIATION

It is beyond question that the most important quality of radium in medicine is the proved fact that the filtered gamma rays have a selective action on cancer cells; by that is meant that malignant growths are more readily affected, and by smaller doses than are normal adult tissues. It is quite another problem how this action is obtained. Is it a direct or an indirect effect? Is it a direct effect on the tumour cells and an indirect effect on the stroma? Is it a local or a general action? For the detailed discussion on the subject the reader is referred to the now numerous text-books on radiology and radio-therapy. The fact remains that the virtue of radium which makes it a useful weapon in the treatment of cancer is the discriminating effect on malignant cells.

## PHYSIOLOGICAL EFFECTS

When energy from radium irradiation penetrates living tissues a series of changes occur leading to injury of the cells and in certain cases to permanent inhibition of all vital phenomena and ultimate death of the cell. The atoms composing the protoplasm of the living cell, when submitted to gamma rays, are displaced from their normal position or removed altogether; this leads to a chemical change of the proteins of the cell, which break down into simpler compounds; thus the normal physiological processes of the cells are profoundly altered. The time which elapses between the bombardment by gamma rays and the occurrence of the changes is spoken of as *latent period*. In tissues undergoing cell division the rate of mitosis is altered, and after abnormal divisions mitosis is completely arrested; the arrest, however, may be only temporary. Histological studies and tissue culture studies show clearly the various changes which occur in irradiated tissue, and Cinti's films show this very graphically and convincingly. If the radiation given is of moderate intensity, recovery takes place, and cell division recommences after a time; in some cases complete recovery takes place, in other cases after dividing a few times the cell dies. Other important effects produced by radiation are changes in the hydrogen-ion concentration of the cell protoplasm, increase in the permeability of the cell wall and changes in the respiratory rate.

## SENSITIVITY

Cells identical in type show wide variation in their sensitivity to gamma rays, and this applies equally to cells of different type. Some cells die after a small dose, others remain alive after a dose many times as large. The law of Bergonié and Tribondeau that sensitivity to irradiation is in direct proportion to the reproductive activity is true only in a very general sense; a notable exception is the lymphocyte, which is one of the most radio-sensitive cells, although its power of reproduction is not great. There is a definite relation between the metabolic rate and sensitivity, and this applies to the increased metabolism during mitosis. At what actual stage of mitosis the cell is most susceptible is not yet definitely determined, but Strangeways and Hopwood have shown that the prophase appears to be the most sensitive stage.

The state of hydration of the tissues also affects sensitivity; blood

supply is another important factor, a fact that has been shown experimentally by Mottram and has been observed clinically; œdema and sepsis increase radio-resistance and the condition of the stroma influences the response to radiation. Normal tissues vary in their sensitivity to radiation, the spleen and bone-marrow, testis and ovary being sensitive, peripheral nerves, muscle and fat being resistant.

Tumours vary in their response to irradiation. Generally speaking, sarcoma and lymphadenoma are the most sensitive; of epithelial tumours, squamous-celled cancer is more sensitive than adeno-carcinoma, especially the mucus producing variety. Anatomical situation influences sensitivity, typical instances of this being provided by the radio-sensitive squamous-celled carcinoma of the cervix and vagina and the radio-resistant vulval epithelioma. The degree of differentiation bears a close relationship to sensitivity, as instanced in the sensitive lympho-epithelioma of the back of the tongue, and the more resistant squamous-celled carcinoma with cell-nests found in the rest of the mouth. The gradation of malignancy on histological grounds is to a certain extent useful in the estimation of radio-sensitivity, but in careful study of wide sections of tumours it is found that the same tumour varies both in its microscopical appearance and in its sensitivity; mixed tumours offer good examples of these variations. As a general rule, the more malignant the tumour the more radio-sensitive it is; this, however, must not be confused with the ultimate prognosis which depends solely on the subsequent development of metastases and not on the rate and degree of disappearance of a tumour by radiation. Natural radio-sensitivity can be lost by repeated radiation. This so-called acquired resistance is sometimes compared to immunity; it is due to latent changes produced by previous irradiation. Latent or acquired resistance is of very great practical importance, and should be considered when repeated radium treatments are necessary.

#### THE TIME FACTOR

To ensure permanent arrest of growth in malignant tumours, it is necessary to submit the cells of the tumour to efficient irradiation during a certain minimum period of time. The radiation time should be sufficiently long to affect all stages of the mitotic changes and all periods of cell activity. Whether the cells recover or die after radiation depends upon the intensity of the beam and the time of exposure.

The advantages of prolonged treatment were first pointed out by Régaud. He obtained sterilisation of a ram's testis by prolonged

irradiation with feeble doses of gamma rays, although he failed to do so by using three times the dose for a shorter period. This has led to the modern method of prolonged irradiation for periods of 7 to 10 days in interstitial methods, 2 to 4 weeks in surface methods, and up to 6 or even 8 weeks in mass or distance radiation. The time factor depends, therefore, on the method employed. The effect obtained can be varied by fractioning the dose or splitting the treatment into short periods; this is impossible in interstitial irradiation, desirable in surface treatments, and essential in distance irradiation by large quantities of radium. Experimentation with the time factor has shown that there is a "threshold value of time" which must be exceeded if any effect is to be obtained. Further, in considering the time factor, it must be remembered that a period of time always elapses before any noticeable change occurs. This latent period varies with the tissues irradiated, the dose absorbed, and the method of treatment. An instance of the latent period is the time which elapses between treatment and the shedding of hair, or the peeling of the skin.

#### EFFECTS OF RADIATION

*Stimulation.* It has often been asked by clinicians if it is possible to stimulate growth by weak irradiation. Experimental evidence is still very conflicting. Some physiological processes are accelerated by gamma radiation, but this quickening continues only for a short period, and is followed by cessation of activity. Experiments on the protoplasm of plant cells, on eggs, protozoa, and seedlings have given contradictory results. The work of Canti and his collaborators has shown that in tissue culture, where rate of growth can be observed accurately, there is no evidence of any stimulating effect.

*Inhibition.* This has been definitely proved both by experimental methods and by histological studies. There is also abundant clinical proof that inhibition of all activity follows irradiation. If irradiation is of such intensity as to cause inhibition only, abnormal cell division occurs when activity is resumed. Bohn irradiated frogs' eggs and did not observe changes till the stage of "blastula" was reached, when the whole structure degenerated. Lacassagne and Coutard observed changes in mammalian ovaries months and years after irradiation with long apparently normal periods in between. Inhibition of activity occurs after certain latent periods, and can be transmitted to a second generation. Régaud has shown that changes can be

obtained in rabbits' embryos when normal ova are fertilised by irradiated spermatozoa. Monstrosities and deformities transmitted to the second and even third generations have occurred in mice issued from irradiated parents.

*Destruction.* This is the essential quality of radiation and is the base of radium therapy. The degree of destruction depends upon the technique of irradiation, and can be varied both in degree and extent at the will of the therapist. It can be regulated with great accuracy and the greater the clinical experience the more accurate the degree of destruction. Régaud sub-divides the destructive effect of radiation into two types: (1) *Diffuse cytocaustic action*; this is really a radium burn and is obtained by the action of feebly penetrating rays acting for a sufficiently prolonged time. There is no selectivity in the type of destruction, all tissues undergoing necrosis. (2) *Selective cytolethal action*; this is a controllable effect; it can be obtained from identical quantities of radium provided that by suitable filtration primary beta radiation is eliminated and the time of irradiation controlled. It affects certain cells with greater speed than others, and apparently leaves normal structures unaffected, or causes only such injury as can be repaired.

*Radium Burns.* The term "Radium Burn" should not be applied to those reactions, erythema, pigmentation, and peeling, which are the inevitable concomitant of radium therapy. These reactions are as a rule painless and heal rapidly, leaving no scar or a pale thin cicatrix, which later may develop telangiectasis. The term *radium burn* denotes severe effects with massive necrosis of tissues, it is neither inevitable nor is it of any value. It denotes, as a rule, errors of technique or errors of judgment, and is due to either over-dosage or under-screenage. Burns may be due to  $\beta$  rays or to  $\gamma$  rays. The  $\beta$  rays are the most caustic; except in selected cases for special purposes they are not used in the treatment of malignant disease. Primary  $\beta$  rays are completely eliminated by 0.7 mm. of platinum. If the screenage of needles is brought up to 0.8 mm. of platinum all primary  $\beta$  rays and the softer  $\gamma$  rays are eliminated. The secondary  $\beta$  rays due to the effect of  $\gamma$  rays on the metal filter can to a certain extent be eliminated by a covering of rubber or secondary filter of a metal of medium atomic weight. This is not practicable in interstitial irradiation, but is possible in distance or surface treatment.

Burns due to gamma rays may be produced by small quantities of

radium applied for excessively long periods or by large quantities of radium applied for a shorter time: clinically two classes of burns are recognised:

(1) *The immediate burn*—this occurs within a few months from the treatment. It is due to an overdose of radiation by insufficiently screened radium. It may develop within two weeks from the treatment; the skin becomes red and œdematous, blisters and peels off, leaving a greyish-green slough (fig. 783). Similar changes occur in mucous membranes. The condition may be exceedingly painful, and it is very slow in healing, the slough taking eight to ten months to separate; around the burn the tissues are œdematous and indurated. Such a



Fig 783.—RADIUM BURN 6 MONTHS AFTER TREATMENT.

burn sometimes occurs with good screenage and moderate intensity; this is the case if the tissues are abnormal in their response to radiation. Predisposing factors are a poor blood supply, scarring, injury, previous irradiation with radium or X-rays, or treatment by diathermy or carbonic acid snow. An avascular structure such as cartilage is readily burned.

(2) *Delayed burns*—these may occur several years after treatment. Chevalier Jackson has described burns eight years after treatment. They are due to necrosis consequent on a progressive *endarteritis obliterans*; this is probably the cause of rectal burns following treatment of carcinoma of the cervix uteri. The lesion, whether immediate or delayed, must be distinguished from a recurrence; if in doubt biopsy is indicated; repetition of treatment should not be undertaken without

histological evidence of recurrence. The separation of the slough and healing can be accelerated by excision of the burn or diathermy destruction, sequestrectomy, and drainage; local application of ultra-violet light is also beneficial.

#### METHODS OF RADIUM THERAPY

Radium therapy of malignant disease is in practice *gamma-therapy*. The aim in view is to distribute in the tissues treated an adequate and homogeneous radiation, avoiding injury to normal structures. Homogeneity of radiation is essential if the whole diseased area is to be influenced by the gamma rays to the desired degree. To obtain this homogeneous irradiation various methods are used differing with the size and site of the lesion. Application of sources of radiation to a lesion are governed by two fundamental laws: (1) the intensity of radiation is inversely proportional to the square of the distance; (2) the diminution of intensity is due to the absorption by the irradiated tissues.

There are four methods of applying radium:

- (1) Cavity method.
- (2) Interstitial method.
- (3) Surface method.
- (4) Distance method.

*Cavity Irradiation.* Radium in suitable containers (needles or tubes) is introduced through the natural or artificial openings into the cavity of hollow organs. The method is of great simplicity, but its general use has been abandoned in most situations owing to the difficulty of obtaining homogeneous irradiation throughout the tumour. It is widely used in gynaecology, occasionally in the rectum and oesophagus, also in the bronchus, and, after suitable methods of approach, in the maxillary antrum. Apart from these situations, the use of this method has few indications. As a palliative in advanced cases, unsuitable for more accurate and efficient, but more complicated methods, cavity irradiation is to be recommended. In such cases, very high filtration (1.5 mm. or even 2 mm. of platinum, and a secondary filter of rubber) is essential, and slow treatment is indicated. The quantities of radium used average 40 mg.; the time of application is 48 to 90 hours. Arrest of hæmorrhage, diminution of discharge, and temporary inhibition of growth are obtainable.



*Interstitial Irradiation.* By interstitial irradiation, or "radium puncture," is meant the insertion of radium in suitable containers into the tissues. Dominici was the first to insert tubes into the centre of the tumour, and Stevenson of Dublin went one step further and introduced the radium needle, which, however, was made of steel. Régaud developed the method and used platinum needles. The interstitial method does not give pure gamma radiation, as the tissues in the immediate vicinity of the needles receive secondary beta rays from the metallic filter. Tubes are no longer used in this type of treatment, being entirely superseded by needles.

The idea of "needling" is to surround the tumour with a palisade of needles of small radium content left in position for 7 to 10 days. The needles used contain 0.5 mg., 1 mg., 2 mg., 3 mg. of radium, and should all be of the same linear intensity, the active length being 1 mg. of radium per cm. In order to facilitate calculations, needles of 0.6 mg., 1.33 mg., and 6.0 mg. have been used, these giving multiples of 5 microcuries per hour. All needles have suitable eyelets and sharp points

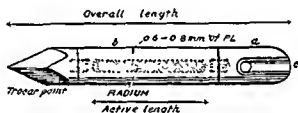


Fig. 784.—DIAGRAM OF RADIUM NEEDLE.

- a. Head with eyelet.
- b. Platinum container.
- c. Groove for thread.

(preferably trocar points) (fig. 784). They are threaded with linen thread, silk or silkworm-gut, securely tied and inserted into the tissue. They are kept in position either by suturing each needle to normal tissue or by inserting them in couples in opposite directions, tied in such a way

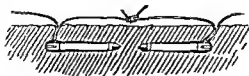


Fig. 785.—METHOD OF ANCHORING NEEDLES.

that each needle anchors its fellow (fig. 785). Needles are made of platinum or gold; in most clinics platinum needles of a minimum of 0.6 mm. of platinum screenage are used.

Interstitial irradiation is suitable for tumours of small or moderate dimensions, accessible on all sides. It is used especially in the treatment of carcinoma of the tongue, lips, tonsil, buccal mucosa, skin (including epitbelioma of the penis), the female urethra and as part treatment in breast cancer. The tissues in immediate contact with the needles are destroyed by the secondary beta rays; the action of the gamma rays on the tissues around the needles, for about 1 cm., produces lysis of the malignant cells. The possibility of secondary infection must be kept in mind, as radiation as a rule lowers tissue resistance to infection; bony and cartilaginous necrosis must be guarded against by careful screenage. The histological nature of the tumour influences the choice of method of irradiation. Generally speaking, rodent ulcers and epitbeliomata are suitable for needling, while lympho-epitbeliomata and sarcomata do not require it, if other forms of irradiation (surface and distance methods) are available. Vascular tumours such as the thyroid or bone sarcomata are not suitable for needling. Bone sarcomata, if needled, necrose rapidly, and spongy infected sequestra filled with pus, surrounded by a wall of fibrous tissue, lead to a condition much worse than the original disease, even if the malignant process is temporarily arrested.

*Surface Method.* By surface irradiation is meant the application of moderate quantities of radium at comparatively short distances from the skin. The distance between the skin and radium varies between 1.5 cm. to 4 cm. according to the quantities of radium used and the methods employed. The apparatus carrying the radium units is known as a "plaque," "collar," or "pack." The material used in their construction must be light, flexible, and free from metallic substances. The object of the plaque is to keep the radium at a given distance from the skin. The applicators are made of sorbo rubber, columbia paste (bees-wax, paraffin and sawdust), wood, piano felt, or any substance such as nidrose, stent, or plastic wood, which can be moulded to the part and used as a foundation for the distribution of radium. The greater the quantity of radium available, the thicker the plaque. 15 mm. was a standard distance at Westminster Hospital as long as quantities of radium varying between 40-80 mg. were used. With increased quantities (150-250 mg.) the distance was increased to 4 cm. or more. By this method continuous or intermittent irradiation varying from 12 to 20 hours daily is given; irradiation is continued 14 to 31 days, and leads to erythema and peeling of the skin. The method is employed in the treatment of breast cancer, cervical

metastases, malignant inguinal glands, intracranial tumours, and, if distance radiation is not available, for the treatment of thyroid carcinoma, sarcoma of the mediastinum, and bone sarcoma.

*Distance Radiation.* The most promising method of radium therapy is that known as "Distance Radiation," "Teleradium," or "Mass Radiation," employing specially constructed containers known in this country under the name of "bomb." One of the essential points in the use of distance radium is the improvement in the rate of transmission by the increase of the radium-skin distance (focal distance). The increased rate of transmission can be illustrated by the following example (fig. 786).

If a source of radiation is placed 1 cm. from the skin, the percentage dose received by the skin and by a tumour 5 cm. deep to it is :

$$\frac{(1 \text{ cm.})^2}{(1+5) \text{ cm.}^2} = \frac{1}{36} = 3\%$$

If the radium is placed at 2 cm. from the skin, the tumour remaining at the same depth of 5 cm. the percentage dose is :

$$\frac{(2 \text{ cm.})^2}{(2+5) \text{ cm.}^2} = \frac{4}{49} = 8\%$$

At 3 cm. from the skin under the same conditions the figure is :

$$\frac{(3 \text{ cm.})^2}{(3+5) \text{ cm.}^2} = \frac{9}{64} = 16\%$$

At 5 cm. from the skin the percentage dose is :

$$\frac{(5 \text{ cm.})^2}{(5+5) \text{ cm.}^2} = \frac{25}{100} = 25\%$$

At 10 cm. from the skin the percentage dose is :

$$\frac{(10 \text{ cm.})^2}{(10+5) \text{ cm.}^2} = \frac{100}{225} = 44\%$$

It is evident that by increasing the focal distance from 1 cm. to 5 cm. the transmission rate is increased from 3 per cent to 25 per cent.

The increase is made at the expense of the total energy, and therefore the quantity of radium used must be greatly increased. The object of mass radiation is to deliver a depth dose of high intensity uniformly throughout a tumour otherwise inaccessible. The efficiency of the method is in direct proportion to the ratio between the amount of radium and the focal distance. The price of radium, the construction of the container, the essential accessories, the space, the personnel needed for its proper application, continue to make mass radiation an expensive method of treatment, but all these considerations are only

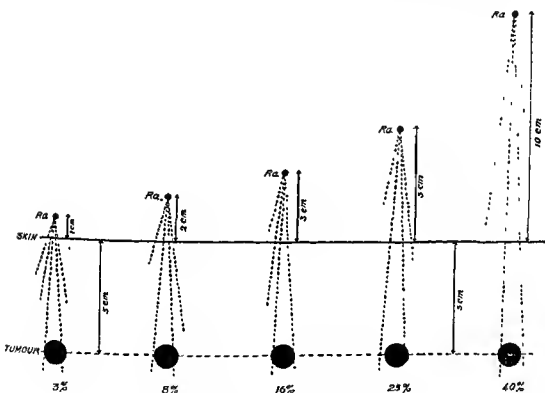


Fig. 786.—DIAGRAM TO ILLUSTRATE THE ' INVERSE SQUARE ' LAW.

of importance if a simpler but equally efficient method of radium therapy is applicable. The "bomb," therefore, should not be used if simpler methods with smaller quantities of radium are equally efficacious. When, however, lesions such as cancer of the post-cricoid area or pyriform fossa are to be treated, questions of expense and complexity of treatment are of no importance if the results obtained are good.

Bombs containing quantities of radium varying from 1 gm. to 8 gm. have been used in numerous clinics for about ten years. The best known models are those of Ferroux, at the Paris Radium Institute (2 gm. and 8 gm.); of Failla, at the Memorial Hospital, New York (4 gm.); and of Sievert, at the Radiumhemmet of Stockholm (3 gm.

and 5 gm.). A duplicate of the latter is in operation at the London Radium Institute under a Committee known as the "Beam Committee." At Westminster Hospital units of 1 gm., 2 gm., and 4 gm. have been used.

Five models have been used since the first "bomb" was installed in 1928. The present model has the advantage of being of small dimensions, 38 lbs. in weight. It is counterpoised over pulleys, and rotates on an axis through its centre of gravity. The radiation channel is 3.5 cm. internal diameter, and has a gold internal screen 1 cm. thick, 3 cm. long. It has all the criteria considered essential in a distance radiation apparatus: it is small, mobile, easily manipulated; the radiation

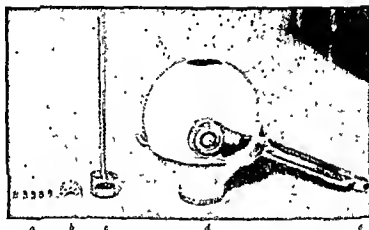


Fig. 587.—THE WESTMINSTER HOSPITAL BOMB.

- a. Radium units.
- b. Horseshoe container for a.
- c. Carrier for b.
- d. The bomb—made of lead and gold which receives c and which is suspended from a.

channel is narrow, so that multiple ports of entry can be used; the focal distance is variable at will up to 5 cm.; it is comfortable to the patient and perfectly safe for the attendant (fig. 787).

The method of application is as follows: the various ports of entry are mapped out on the skin with Finzi's ink (fig. 788). All the ports are treated in rotation; a metal holder in the form of a truncated cone of the correct angle is applied to each port of entry and fixed in position by elastic bands (fig. 789); the patient is brought to the "bomb" room with the holder in position and the bomb is lowered into the metal cup in a few seconds. Absolute accuracy of application is thus obtained, with perfect safety to the staff (fig. 790).

The quantity of radium used at present is 2 gm. The cases treated are chiefly pharyngeal neoplasms and bone sarcoma, but selected cases



*Fig. 788.*—PORTS OF ENTRY MAPPED OUT IN FINZI'S  
INK. CASE OF PYRIFORM FORSA CARCINOMA.



*Fig. 789.*—METAL HOLDER APPLIED TO EACH PORT OF ENTRY  
OUTSIDE THE "BOMB" ROOM.



*Fig. 790.*—BOMB ATTACHED TO METAL HOLDER IN POSITION.

of cancer of the breast, thyroid, and other situations are also considered suitable. The results obtained are unparalleled by any other method of treatment, and with increased quantities of radium, mass radiation will undoubtedly cover a larger field of usefulness.

#### VALUE OF RADIUM TREATMENT

The clinical use of radium is increasing year by year, and the number of patients treated by radiation is growing in proportion with the facilities available. This increase in numbers is confirmed by reliable statistics from all official sources. The Tenth Annual Report of the British Empire Cancer Campaign draws attention to this increase; although the figures of the report are only a mere fraction of the whole, they are of great significance. Whereas in 1930 the number of recorded cases of cancer treated by radium alone was 1129, in 1933 the number had risen to 4952. The three year "cures" based on complete freedom of disease after radium as the sole method of treatment increased from 68 in 1930 to 415 in 1933. The importance of these figures is the fact that, whereas the number of cases treated has increased four times, the three year "cures" have increased seven times. This improvement signifies the advances made in radio-therapy.

*Final Results of Radiological Treatment of Cancer.* In comparing the results obtained by radiation with those of surgical excision, it is essential to consider the *absolute percentage cure*, that is the percentage cures in proportion to the total number of cases examined. In surgical statistics operable cases only are considered; radiological statistics include borderline and inoperable cases. Forsell's statistics for Sweden show permanent healing percentages as follows:

TABLE II

	<i>Surgery.</i>	<i>Radio-therapy.</i>
Cancer of the skin . . . . .	65%	78%
Cancer of the lip (operable, without metastases) . . . . .	73%	86%
Cancer of the mouth (without metastases) . . . . .	41%	55%
Cancer of the cervix (absolute % cure)	18%	20.6%
Cancer of the cervix uteri (operable and borderline) . . . . .	35.6%	40%

## VALUE OF RADIO-THERAPY AS A PALLIATIVE MEASURE

The absolute value of radio-therapy cannot be assessed either by statistics of 5-year cures, or by comparison with other methods of treatment. The method must not be judged by the mortality statistics alone. The palliative value of radiation is of great importance, if considered from the *number* of cancer patients submitted to it. The Swedish Cancer Association statistics show that only one-third of those seeking advice are suitable for surgery; and of those who submit to operation at the most only one-third are definitely relieved. Of the total number of cancer patients only 10 per cent are permanently treated by excisional surgery. The rest (90 per cent) have only radio-therapy as a method of alleviation. The extent of palliation and prolongation of life obtained from radio-therapy (Radium and X-rays) can be gauged by the frequency of primary healing. In a period of six years, 1921-7, 4470 patients were examined at the Radiumhemmet, of whom two-thirds received radiological treatment only. Of these 38 per cent obtained primary healing and freedom from *symptoms*. Half the number of cases were patients with uterine, skin and lip cancer. Healing took place in 92 per cent of skin cancer, 78 per cent of lip cancer, and 58 per cent of uterine cancer. Even local recurrences should not detract from the value of palliation by radio-therapy, as the incidence of recurrences is diminished and the interval period of freedom from symptoms increased.

Successful irradiation, and hence its value as a therapeutic measure, depends upon certain cardinal principles.

(1) Success does not depend upon "*Radium*," but on *Radium efficiently applied*.

(2) Palliation, as distinct from possible cure, calls for a different technique.

(3) Combination of Surgery and Radium has improved end-results in certain localisations to such an extent that it should be recognised as the treatment of choice.

(4) The chief causes of failure are: (a) insufficient resources; (b) bad organisation; (c) inexperience; (d) lack of knowledge of the natural history of cancer.

(5) Inefficient irradiation is useless and should not be undertaken.



## RADIUM TREATMENT OF CANCER OF THE TONGUE

The treatment of epithelioma of the tongue with radium has now been practised for about fifteen years, and it cannot be said any longer that it is in the experimental stage. It is universally recognised that radium offers more than surgery in this anatomical situation, at least in connection with the primary growth. The actual value of radiotherapy in lingual cancer should not be assessed by comparing results from excision, e.g. those of Butlin, with the results of radiation, e.g. those of Régaud, although even in such a comparison the advantage is on the side of radiation. The problem should be looked at from a much wider angle—namely, that the barrier of operability as applied to excision is not applicable to radiation. If *all* cases of cancer of the tongue are taken into consideration the advantages of radium are as follows: (1) operability is of secondary importance—that is, cases unsuitable for excision, owing to (a) local extent of the disease; (b) inaccessibility (e.g. the posterior group); (c) age of the patient; (d) medical contra-indications to major operation, are still suitable for radiation. (2) In expert hands the treatment by radium is (a) safe; (b) painless; (c) efficient to arrest or eradicate the disease. (3) Functional results are undoubtedly better from radium than surgery. It is naturally possible to excise early epitheliomata of the tip of the tongue by small local operations, and in such cases there is little advantage in using radium, except that local recurrence is less likely after irradiation than after local excision. Permanency of results is also established; seven, nine, eleven and more years of survival with complete freedom from disease can be shown in most clinics. The percentage of survivals, however, varies with several factors of which the most important are extent of the disease and virulence of the neoplasm. It is noticed, however, that the heaviest loss is during the first two years; that after the third year the fall in the survival rate diminishes very appreciably and it can be said with confidence that after the fourth year the chances of permanent cure are very good indeed. If a patient survives five years, the probability of recurrence is very small, so that the figures for the sixth and tenth year are nearly the same.

*Method of Treatment.* The general principles of radium therapy of cancer of the tongue can be summarised as follows:

(1) *Oral toilet must precede all treatment.* Septic teeth or even healthy teeth which are mechanically in the way should be extracted.

The importance of a clean mouth cannot be exaggerated, and additional sepsis from the teeth and gums renders the treatment more difficult, more unpleasant to the patient, and less efficacious. The gravity of the disease should outweigh considerations of vanity, comfort, or any other reason which may be brought forward against clearance of the mouth. Some workers have stated that extraction of teeth is not only unnecessary but even dangerous—this has not been the author's experience. In the presence of metal fillings, gold bridges, or even with healthy teeth, should these not be sacrificed, protection by rubber or vulcanite plates will diminish the degree of reaction. The criticism levelled against lead protection plates does not hold good for the up-to-date *small coverings accurately fitting over the teeth, light in weight, and of which the patient is quite oblivious* (fig. 791).



Fig. 791.—VULCANITE OR RUBBER DENTAL "PROTECTION PLATES."

(2) *Treatment of the Primary Growth should always precede treatment to the lymphatic area.* Disturbance of the lymphatics and lymphatic glands by surgery or irradiation prior to the treatment of the primary growth is highly undesirable, as for the success of irradiation it is necessary to have the parts irradiated under the best possible conditions as regards blood supply and venous and lymphatic drainage. Cell metabolism, state of hydration and local natural drainage have a fundamental influence on the effect of radiation on the tumour cells, and the less the interference the better the chance of success.

(3) *Treatment of the lymphatic area, either by surgery or irradiation or a combination of both, must be carried out in all cases of lingual cancer.* It equals in importance the treatment of the tongue itself, and should never be omitted, as experience shows that the risk of leaving the neck untreated is very great.

The choice of the treatment to the cervical lymphatic area depends upon several factors, and will be analysed later (see page 1584).

*Technique of treatment of the Primary Growth.* Cancer of the tongue is sub-divided into two anatomical groups: (1) anterior—in front of the V of circumvallate papillæ; (2) posterior—in the vallecula and epiglottis (fig. 792). A secondary sub-division is into lateral groups

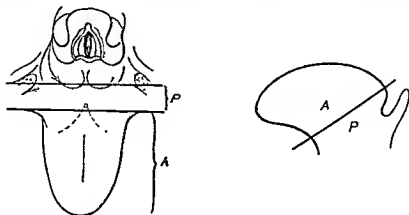


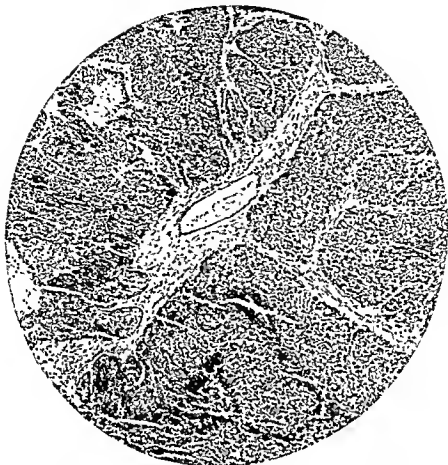
Fig 792.—ANATOMICAL SUB-DIVISION OF LESIONS IN THE TONGUE. A, ANTERIOR. P, POSTERIOR.

and inferior group, the latter often encroaching on the floor of the mouth. Histologically, cancer of the tongue is sub-divided into three groups: (1) squamous-celled carcinoma with cell-nests (fig. 793); (2) transitional-celled carcinoma without cell-nests (fig. 794); (3) lympho-epithelioma (fig. 795). All three histological types occur in the anterior



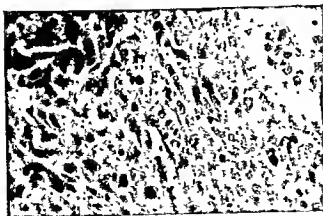
Fig. 793.—SQUAMOUS CELLED CARCINOMA WITH CELL-NESTS.

group. The lympho-epithelioma occurs in the posterior group, its frequency being about 10 per cent of the total cases. This anatomical and histological classification is of essential practical importance. The posterior group and the lympho-epitheliomata are extremely radio-sensitive, they are also of a high order of malignancy, of rapid



*Fig. 794.*—TRANSITIONAL-CELLED CARCINOMA (WITHOUT CELL-NESTS).

dissemination, and of grave prognosis. The knowledge of this differentiation dictates the selection of treatment. Cancer of the tongue can also be classified according to its clinical appearance into three main groups: (1) papillary or cauliflower, (2) nodular, (3) ulcerative. The first group is the most radio-sensitive, the last the most resistant to radiation, whilst the second group occupies a midway position.



*Fig. 795.*—LYMPHO EPITHELIOMA.

Successful radium treatment of lingual cancer requires experience in radium therapy and adequate equipment. In the best circumstances the choice of treatment depends upon three main factors: site of lesion, type of lesion, radio-sensitivity.

*Anterior and Antero-lateral Group.* In this situation the method of choice is needling. A harrage is placed deep to the lesion so as to irradiate the tumour bed, and the lesion is surrounded by a palisade of needles regularly spaced at the periphery (fig. 796). The needles used in the tongue vary according to the volume and size of the growth. The following table gives the specification of the needles (table III):

TABLE III

<i>Amount of radium.</i>	<i>Overall length.</i>	<i>Screenage in platinum.</i>	<i>External diameter.</i>
0.6 mg.	16 mm.	0.5 mm.	1.6 mm.
1.33 "	27 "	0.6 "	1.8 "
2 "	33 "	0.65 "	1.9 "

Additional screenage up to 0.8 mm. of platinum is desirable in the proximity of the angle of the jaw.

*Technique.* A number of suitable needles are selected and threaded with thick linen thread or carbolised silk. A knot is tied in juxtaposition with the eyelet; the needles are counted, placed in a suitable container, or threaded on a piece of linen which is folded and tied. The needles are boiled for five minutes and then placed in a weak solution of flavine. For small lesions a local anæsthetic with suitable premedication is sufficient; in most cases a general anæsthetic is desirable; gas and oxygen by the intra-tracheal method with a nasal catheter is the most suitable anæsthesia, as this leaves a free mouth and the pharynx can be lightly plugged. The mouth is kept open with a suitable gag; the tongue is pulled forward and the lesion examined by inspection and palpation. If the lesion is small, 0.6 mg. needles are inserted vertically around it and sutured in position; in more extensive lesions, 1.33 mg. or 2 mg. needles are placed horizontally in pairs from opposite directions and tied to each other (see figs. 785 and 786). All the threads are collected together, counted, tied and cut short with the exception of one or two threads which are brought out loosely at the angle of the mouth and attached to the skin with plaster or mastisol.

The quantity of radium varies with the extent of the growth from 6 mg. to 15 mg. or even more. The needles are left in position 7 days in most cases ; very extensive growths may require 10 days treatment or re-needling at the end of the first week. The total dose given this way varies from 1000 to 3000 mg. hours. The needles are removed under evipan anæsthesia. Whilst the needles are in position, the patient is kept in bed ; he is instructed not to talk but to write down his requirements. The mouth is irrigated three or four times a day with a mild solution of Condy's fluid. Diet is of necessity restricted to fluids, and the patient is encouraged to drink as much as possible. The mouth is carefully examined every day and the local reaction and

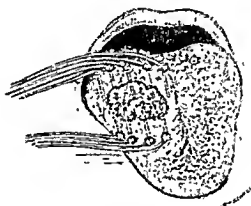


Fig. 796.—NEEDLING OF TONGUE.

position of needles are watched. Pain may be entirely absent, but is slight as a rule in the majority of patients. Oedema is common, occurring at the end of the first 24 hours and subsiding in most cases in 4 or 5 days.

*Posterior Group.* The ideal treatment of this type of lesion is external irradiation by a bomb containing 1 gm., 2 gm., or 3 gm. of radium, and this method should be used in the posterior group of lingual cancer whenever practicable. It presents great advantages over needling. The vallecula, epiglottis and back part of the tongue are difficult of access through the mouth, and external needling, even with a guiding finger in the mouth, presents great technical difficulties, and consequently it is difficult to irradiate the *whole* lesion. By distance mass irradiation with a "bomb," a wide area in the posterior part of tongue and adjoining tissue can be treated with great accuracy and

the desired reaction obtained with the least discomfort to the patient. Four ports of entry are made use of, two on each side of the neck. Treatment is given to each port alternatively, two ports being treated daily. Duration of treatment: 1 hour per port with 2 gm. of radium, 2 hours with 1 gm. of radium. Distance: varying from 4 to 6 cm. Duration of treatment: 21 days; total time per port: 7 to 10 hours. The average total dose delivered is 30,000 mg. hours. With the correct technique it is possible to give an efficient depth dose with only an erythema of the skin or epilation. In resistant cases peeling of the

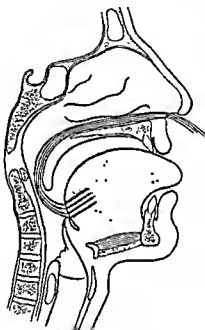
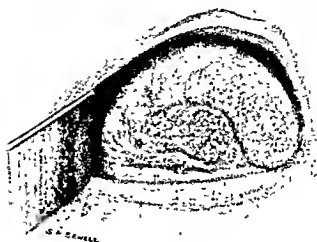


Fig 797.—NEEDLING OF THE LESION IN POSTERIOR PART OF TONGUE. THREADS BROUGHT OUT THROUGH THE NOSE.

skin is at times unavoidable. If distance irradiation is not available, surface treatment with plaques containing 100 mg. of radium is the best alternative; these are applied to each side of the upper part of the neck at a distance of 4 or 5 cm. continuously 20 hours daily for three weeks (fig. 800). The results are good, but the skin reaction is more marked and extensive peeling is unavoidable. If needling of the posterior part of the tongue cannot be avoided, the needles can be introduced through the mouth from behind forwards with an angular needle holder, but they cannot be sutured in; the threads are collected in a bunch and brought out through one or both nostrils and tied; by this method there is less likelihood of the needles becoming displaced than

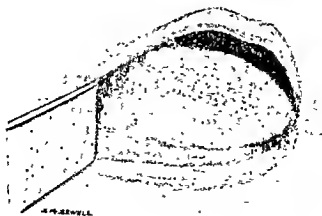
when the threads are brought out through the mouth (fig. 797). An alternative method to needling through the mouth is the introduction of the needles from outside either by puncturing the skin or through an open incision, when a finger in the mouth guides the direction of the needle. In the posterior group of cases, interstitial treatment is not the best method of irradiation, and should be used only when surface or distance irradiation cannot be carried out from lack of facilities.

*Changes produced by treatment.* As early as the third day from the beginning of irradiation visible changes may be noticed. In most cases no change is observed until the fifth or sixth day. The earlier the reaction the better will be the result. The first change noticed is



*Fig. 798*—CARCINOMA OF THE TONGUE BEFORE RADIUM TREATMENT.

a flattening out of the neoplasm. This is most noticeable in the cauliflower or papillary type; in the ulcerative type the crater becomes shallower and the edges less well defined. In a few days the area treated becomes paler, and gradually a layer of fibrin spreads over the lesion; between the seventh and tenth day a thick white film covers the lesion, which is sharply demarcated from the surrounding tissues; the colour of the fibrin changes from white to yellow in the following week, and in successful cases it gradually diminishes in size, healing taking place from the periphery. In about one month the lesion is healed and no induration is felt on palpation (figs. 798 and 799). This is



*Fig. 799*.—THE SAME PATIENT AS IN *Fig. 798*—AFTER TREATMENT.



the normal reaction. Inflammation, œdema, acute sepsis, necrosis, or hæmorrhage are complications which can be guarded against by careful attention to technique. In cases previously treated by radiation, there is a risk of radium necrosis both of soft tissues and of adjoining bones. The reaction in such cases is atypical, severe and painful, and lasts several weeks.

#### TREATMENT OF THE LYMPHATIC AREA

The selection of treatment of the cervical areas depends upon several factors. For practical purposes it is useful to separate the cases into three groups: (1) No palpable cervical glands; (2) Palpable cervical glands (operable); (3) Inoperable or fixed masses of glands. The local condition of the tongue influences the choice of treatment. Treatment is imperative in all groups of cases, but the selection of treatment requires careful consideration. A block dissection of the cervical glands is indicated in the following circumstances: (1) The primary growth must be healed with a reasonable prospect of permanency; (2) The glands in the neck must be palpable but strictly operable; (3) The general condition and age of the patient must permit a major surgical procedure. If the above conditions are not present, block dissection should not be undertaken and irradiation should take its place.

*Group 1. Unpalpable Glands.* Irradiation by X-rays or by means of a radium collar is the method of choice. The treatment should be



Fig. 800.—RADIUM COLLAR MADE OF SORBO RUBBER.  
RADIUM SKIN DISTANCE, 4 CM

given not earlier than two weeks after the irradiation of the primary growth. Technique of irradiation by a collar is by no means simple. A wide area must be treated, extending from the mastoid process and lower jaw above to the clavicle below and transgressing the mid-line

in front. The greater the quantity of radium and the greater the radium-skin distance the better the result. The collar must fit accurately and its thickness should be 3 or 4 cm. 100 mg. is required, and this is distributed on the outer aspect of the collar. Daily treatments of an average of 14 hours are given for 10 to 14 days and sometimes longer (fig. 800). Sterilisation of the lymphatic area can be thus obtained. If a homh is available, the collar can be dispensed with. If time is of great importance, needling of the neck is a good substitute for external treatment.

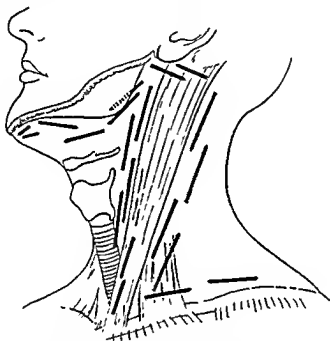


Fig. 801.—NEEDLING OF CERVICAL LYMPHATIC AREA.

*Group 2. Palpable Glands.* In this group the palpable glands may be clinically malignant, or suspicion only of malignancy may be entertained. It is, however, impossible on clinical examination to distinguish with certainty the true nature of glands. This group should be submitted to a block dissection of the neck if the operation is otherwise not contra-indicated. Irradiation is undertaken in all cases where block dissection is contra-indicated. When the glands are enlarged the insertion of needles through the skin (closed method) presents risks of puncturing the glands and spreading the disease to the cellular tissues of the neck. The method known as "open needling" is here indicated. Under general or local anaesthesia an incision is made along the anterior border of the sterno-mastoid from the mastoid to the clavicle; the muscle is exposed, freed and retracted outwards. The common facial vein may have to be divided to allow a better exposure

of the upper part of the anterior triangle. The vascular sheath and glands are exposed and under vision the lymphatic area is accurately needled without damage to the vessels or glands; the supraclavicular fossa, the posterior triangle, and submaxillary area are then needled. The total quantity of radium needed is about 40 to 50 mg. in 20 to 30 needles (fig. 801). All the threads are tied together and cut short except one, which is brought out through the wound and acts as a guide for the subsequent removal of the needles. The wound is closed without drainage. At the end of a week the wound is partially reopened, the needles withdrawn and the wound closed, a drain being inserted. The operation can be done on both sides of the neck at the same sitting if bilateral treatment is indicated.

*Group 3. Inoperable Glands.* In this group of cases the mass of glands is fixed to the deep structures, or there is a diffuse swelling in the neck. Such cases should be submitted to a preliminary course of X-ray therapy, followed by needling of the mass without incision of the skin. The needles are placed transversely into the mass, from behind forwards. The treatment is of a palliative nature, but sometimes complete disappearance of the glands follows. Needles can be left in position up to ten days. Erythema of the skin invariably follows, and sometimes even peeling. In this type of case interstitial irradiation is essential, as a large mass of tissue must be irradiated and the external treatment alone is not adequate.

## RESULTS

The results of radium treatment of cancer of the tongue show the true value of the method if a sufficiently large number of cases in various clinics are analysed. Individual results of small numbers in small centres during their first few years, which could be called the years of apprenticeship, are useless; they are no more a true indication of the value of radium treatment than the mortality of a Wertheim's operation in the hands of a novice is the true indication of its operative risk. The following list of cases represents a fair view of the results:

*Roy Ward and Durden Smith (Radium Institute, London).*

*Five Years: 1925-1929.*

Total number of cases treated	.	.	.	154
Enlarged glands	.	.	.	111
Initial disappearance of growth	.	.	.	116
Recurrence of primary growth	.	.	.	78

In these 154 cases 4 survived five years, 6 for three years, 13 for two years, 47 for one year, 38 for six months, 46 less than six months.

*Douglas Quick (Memorial Hospital, New York).*

*Ten Years : 1917-1927.*

Total number of cases treated . . . .	473
Alive (unselected) (periods not stated) . .	87 or 18%

*Régaud (Radium Institute, Paris).*

*Five Years : 1920-1925.*

Total number of cases treated . . . .	311
36 cases excluded from statistics for various reasons . . . .	Total: 275
Healing of primary lesion . . . .	133=48.3%
Of these inoperable . . . .	89
Died of regional metastases . . . .	57
Survived 1-6 years . . . .	77=28%

*Berven (Radiumhemmet, Stockholm).*

Three year cures in 15 out of 20 cases =	75%
Five year cures in 7 out of 12 cases =	58%

*Stanford Cade (Westminster Hospital, London).*

*Eight Years : 1925-1932.*

Total number : 255.

Seven year survival . . . .	2 cases of 18 treated = 11%
Six year survival . . . .	3 " 16 " = 18.7%
Five year survival . . . .	3 " 16 " = 18.7%
Four year survival . . . .	6 " 21 " = 28.5%
Three year survival . . . .	14 " 51 " = 28.2%
Two year survival . . . .	12 " 46 " = 26%
One year survival . . . .	15 " 34 " = 44.9%
Less than one year survival	20 " 23 " = 86.9%

An analysis of the cases shows that a great proportion of them had glandular metastases when treated, and that in a number of cases extent of the disease precluded any real hope of permanent cure. The conclusions from the above figures are :

(1) Total disappearance of neoplasms, including glandular metastases, can be achieved by means of radiation.

- (2) Disappearance of lesions is not always permanent.
- (3) Radium has reached at least equality of status with surgery in the treatment of operable cases.
- (4) In inoperable cases radiation is the only method of treatment available.
- (5) Palliation given by radium is worth while from the patient's point of view, although it vitiates the value of statistics if compared with surgical statistics.

#### RADIUM TREATMENT OF CANCER OF THE BUCCAL MUCOSA

Squamous-celled carcinoma of the buccal mucous membrane occurs in three clinical types, similar to those met in the tongue. (1) Papillary or cauliflower type, occurring often in patches of leucoplakia; (2) Nodular type, which clinically is a tumour in the substance of the cheek, of characteristic hardness and infiltration; (3) Ulcerative type, with the hard edge and a base of granulomatous appearance. Nearly all cases are histologically squamous-celled carcinomata with cell-nests. Glandular involvement is as frequent as in cases of lingual cancer. The general principles of radium treatment both of the primary growth and of the lymphatic field are identical with those of lesions of the tongue.

*Technique.* External radium application or needling are both used according to the method favoured by the particular clinic. In all cases, excision with the knife or diathermy is contra-indicated, as it adds nothing to the immediate or remote results of radiation. In small growths, not involving the muscles of the face or the gingival mucosa, an *intra-oral surface application* is indicated. A plaster cast is taken of the area to be treated, a suitable stent or shellac applicator is made from the cast and small radium needles inserted into the applicator. For a lesion of 2 cm. diameter, about 15 mg. of radium are required. Treatment is intermittent; according to the patient's tolerance 12 to 18 hours' treatment is given daily. The lesion is inspected every day and treatment stopped on the first appearance of the fibrinous deposit; as a rule 7 to 10 days' treatment is required.

If the lesion is of the ulcerative type, or involves the muscular layer of the cheek, *interstitial irradiation* is indicated. A series of needles are inserted in the submucosal layer under the lesion, and in the healthy tissue above and below; 1 mg., 1.33 mg., or 2 mg. needles

are used, according to the length of the lesion ; 7 days' treatment is given. The dosage varies from 1000-3000 mg. hours.

In extensive lesions involving the skin, both needling and external surface application by means of rubber or columbia paste plaque are indicated. Needling should precede surface irradiation. The plaque need not exceed 2 cm. in thickness, should cover a wide area beyond the growth, and carries between 20-40 mg. of radium. By the combined treatment even extensive growths perforating the cheek can be made to heal entirely. Involvement of the gingival mucosa or bone makes the treatment more complicated, but is no contra-indication to irradiation.

As an illustration we can quote a case of extensive squamous-celled carcinoma of the cheek which had involved the jaw, perforated through the skin, and formed numerous sinuses ; it was treated by needling first through the mouth, followed immediately by needling through the skin. By this method continuous treatment for 14 days was given by the interstitial method and a total of 6384 mg. hours was delivered. At the end of the fifth month after treatment, the ulceration both inside the mouth and outside on the face had entirely healed. There is no recurrence so far, four years after treatment.

#### RADIUM TREATMENT OF CANCER OF THE LIP

All cases of cancer of the lip are eminently suitable for radium therapy, malignant growths of the lip being, next to skin lesions, the



Fig. 802.—EPITHELIOMA OF LIP BEFORE RADIUM TREATMENT.



Fig. 803.—EPITHELIOMA OF LIP AFTER RADIUM TREATMENT.

most susceptible to radium therapy; only a small number of very virulent cases prove failures. There is no doubt that in the case of the lip, radium offers the best possible means of eradicating the disease. Lesions of the lip are either superficial and hypertrophic or infiltrating and ulcerative; the former are the more sensitive to radiation. Glandular involvement is much rarer than in cases of lingual or buccal cancer, and treatment of the cervical lymphatic area is by no means as essential as in the latter group. With small lesions, and in the absence of palpable cervical glands, treatment to the neck can legitimately be omitted. Needling is the best and quickest method of treatment, but surface application by external or combined external and intra-buccal plaques is widely used both in England and in France. The technique of the surface and interstitial method is identical with that applicable to the mouth and tongue. Extraction of septic teeth is of great importance; and the severity of the reaction depends a good deal upon the presence of sepsis. The lesion takes about one month to epithelialise (figs. 802 and 803).

### RESULTS

The author's cases consist of a series of 50 patients, of whom nine died of the disease. As all cases seen were treated irrespective of the stage of the disease, the absolute cure rate is therefore 82 per cent, extending up to ten years. Forsell, in operable cases without glandular metastasis, obtained 86 per cent of cures, as compared to 73 per cent from surgical treatment. At the Paris Radium Institute of 112 cases treated in a period of six years, healing of the primary lesion was obtained in 76 cases. The Radium Institute, London, in the period 1925-1929 treated 45 cases, of which 45 per cent remained free from disease for more than three years.

### RADIUM TREATMENT OF EPITHELIOMA OF THE PALATE

Epithelioma of the palate is differentiated according to its position into lesions of the *soft* palate and those of the *hard* palate. The prognosis varies with the localisation; in the soft palate there is in the majority of cases early involvement of the cervical glands, and in a number of cases this is bilateral. The ultimate prognosis, therefore, is very grave, although the primary lesion is sensitive to radiation and disappears with adequate treatment. Carcinoma of the hard palate must be distinguished from new growth of the maxillary antrum

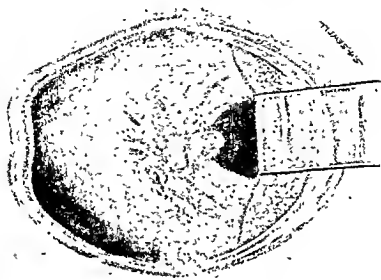


Fig. 805.—EPITHELIOMA OF SOFT PALATE AFTER RADIUM TREATMENT.

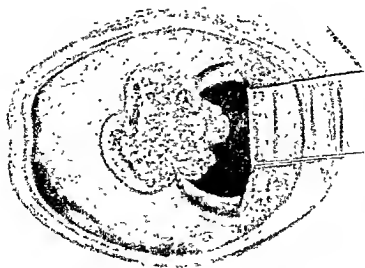


Fig. 804.—EPITHELIOMA OF SOFT PALATE AND UVULA BEFORE RADIUM TREATMENT.



involving the palate. In cancer of the mucosa of the hard palate, occurrence of lymphatic involvement is not so frequent as in the posterior group. The technique of irradiation varies with the situation of the growth. In the anterior group *surface radiation* by an intra-oral dental appliance is the treatment of choice; the plaque is made of vulcanite, shellac, or stent from a plaster cast model; the lingual aspect of the applicator is covered with a layer of lead 1 mm. thick for the protection of the tongue. 15-20 mg. of radium are required; the proximity of the bony palate necessitates full screenage. The plaque is worn 18 to 20 hours daily for 7-10 days. In the posterior group (soft palate) *interstitial irradiation* is the method of choice; this can be done either with needles or with seeds; the introduction of seeds is simpler and quicker; the threads are brought out through one or both nostrils and tied; with attention to technique the needles or seeds can be kept in place seven days. The reaction and healing are similar to those in other parts of buccal mucosa (figs. 804 and 805).

#### RADIUM TREATMENT OF CANCER OF THE MAXILLA

Neoplasms of the maxillary antrum are either sarcomata or carcinomata. The latter are squamous-celled, transitional-celled, or adenocarcinomata. *In selection of treatment the choice lies between radiation or excision.* Excision necessitates very wide removal of the whole upper jaw and at times removal of malar bone, ethmoid cells, and exenteration of the orbit; the results of surgical removal give only a small percentage of permanent cures, and this with grave mutilation, both in appearance and function. Although radiation cannot claim a large percentage of permanent cures, the results obtained are very encouraging; the treatment although tedious is safe, and functional disturbance is small and can be remedied; external deformity can be entirely obviated (fig. 806).

Histological examination must precede treatment. If the tumour is a sarcoma, X-radiation is the method of choice. No operative intervention of any kind is necessary; excision adds nothing to the patient's prospects of freedom from disease and may conceivably hasten dissemination.

Sarcoma of the upper jaw is extremely radio-sensitive, and external radiation is all that is required. High voltage X-rays have given such gratifying results that it should be the first method of treatment; it is safer and simpler than radium, and the results obtained are good if the technique is up-to-date and the dose adequate.

Carcinoma of the antrum is much more resistant, and here radium is definitely superior to X-rays. Treatment by radium requires an accurate estimate of the extent of the neoplasm; it is therefore essential to expose the growth surgically. This can be done through the canine fossa, through the nose, or through the palate. The palatal approach is the method of choice. Douglas Harmer and Douglas Quick prefer this route after ten years' experience of the method. The object of the operation is threefold: (1) access to the neoplasm for partial removal and for irradiation; (2) drainage; (3) provision of an inspection window, so that remnants of growth or recurrences can be easily detected at the earliest possible stage. Fenestration of the palate (fig. 806) fulfils the three conditions without external mutilation. The operation is done under gas and oxygen anaesthesia through a nasal catheter passed into the trachea; this facilitates plugging of the pharynx. A window is cut in the hard palate with the diathermy needle; the actual extent and position of the window depends upon the stage of the disease; removal of the alveolar margin is necessary in about half the cases.

The soft palate, or the posterior part of it, and uvula are preserved, as this assists functional restoration of speech by a suitable dental appliance. Access having been obtained to the lesion, as much of the growth as possible is removed by means of a diathermy loop, extensions into the ethmoidal and sphenoidal areas being looked for. Irradiation is carried out in two stages. The first stage consists in the immediate insertion of a relatively large quantity of radium into the cavity of the antrum, 30 or 40 mg. in tubes of 5 or 10 mg. each, screened by 1 mm. of platinum, being carefully placed in position and the cavity plugged. The plugging and radium are removed 24-48 hours later. A week or ten days after operation the second stage of irradiation is proceeded with; a plaster cast of the cavity is taken, and a hollow model is made consisting of two separate layers of shellac. This applicator is sometimes made in two sections, to facilitate introduction. Radium needles



Fig. 806.—FENESTRATION OF PALATE (BILATERAL).  
ABSENCE OF DEFORMITY. NO RECURRENCE SIX  
YEARS AFTER TREATMENT.

of small dimensions (0.5 mg., 0.6 mg., or 1 mg.) are placed between the two layers of shellac, which are sealed; a plate containing 1 mm. of lead for the protection of the tongue completes the apparatus. The applicator is worn 10-12 hours daily until the cavity is covered with a thin white film (10-12 days). The cavity epithelialises in four to eight weeks. A permanent denture with suitable obturator is then made.

## RESULTS

Douglas Harner in the Semon Lecture for 1931 gives a full analysis of the results of radiation treatment of malignant disease of the upper jaw and this can be summarised as follows:

TABLE IV

NEW (*Mayo Clinic*).

Total 97 cases.

Growths starting in the antrum . . . 70 (a)

Growths starting outside the antrum . . . 27 (b)

<i>Total.</i>	<i>Dead, lost, or recurred.</i>	<i>Alive free from disease (years).</i>			<i>Per cent.</i>
		1-3	3-5	5-8	
(a) 70	49	5	5	11	30
(b) 27	13	1	5	8	51

TABLE V

QUICK (*Memorial Hospital, New York*).

	<i>Total.</i>	<i>Dead.</i>	<i>Lost.</i>	<i>Current file.</i>	<i>Free from disease (years).</i>				
					<i>Total</i>	5-10	3-5	1-3	-1
Maxilla . . .	122	48	35	39	29	9	4	8	8
Antrum . . .	103	42	31	24	16	4	8	2	2

TABLE VI

HARMER'S AND CADE'S (Cases up till 1931)

	Total.	Dead.	Lost.	Alive.	Alive without disease (years).							Alive with disease (years).							Dead (after years).						
					-1	1	2	3	4	5	+5	-1	1	2	3	4	5	+5	-1	1	2	3	4	5	+5
Harmer	53	40	3	9	2	1	1	1	-	-	1	1	1	1	-	-	-	-	23	8	3	1	2	-	3
Cade	15	6	-	9	2	1	1	2	-	-	-	1	2	-	-	-	-	-	3	2	1	-	-	-	-

The total number of cases collected by Harmer is as follows :

TABLE VII

NOSE : SINUSES : (Carcinoma)

Total.	Dead.	Lost.	Alive.	Alive without disease (years).								Alive with disease (years).							
				-1	1	2	3	4	5	+5		-1	1	2	3	4	5	+5	
819	427	136	256	29	13	54	8	40	3	60		-	-		-	49	-	-	

Harmer's conclusions are that, although the results are poor, this is due chiefly to the fact that most of the cases were quite inoperable, and treatment was given solely for palliation. In early cases good results can be expected in at least one third of the cases. Prognosis in sarcoma is better than in carcinoma.

## RADIUM TREATMENT OF CANCER OF THE TONSIL

Malignant disease of the tonsil is now recognised to be of very grave prognosis, and most surgeons are of the opinion that surgical treatment by excision with the scalpel is hardly ever successful in producing a permanent cure. Radiation, with or without additional diathermy excision, is the treatment of choice, and whether X-rays or radium be employed it should be given a trial as the treatment of choice.

Clinically three different types of tonsillar neoplasms are recognised,

the ulcerative type, the papillomatous type, and the massive non-ulcerating tumour. Histologically they are either squamous-celled carcinoma or lympho-epithelioma. Metastases occur rapidly and not infrequently are bilateral.

The technique of irradiation has passed through several phases: interstitial peroral treatment, external treatment, and a combination of both with or without additional diathermy excision have all been tried. There is, however, no doubt that Berven has proved that the method of choice is mass radiation (telerradium) with a bomb containing 2, 3, or more grams of radium. Results both immediate and remote have improved dramatically by the adoption of this method of treatment. A combination of X-rays and bomb treatment has given encouraging results, and the method adopted at Westminster Hospital for the past three years is such a combined simultaneous irradiation; with the 2 gram bomb, three ports of entry are used on the affected side of the neck, at 6 or 7 cm. radium-skin distance; 30 to 36 hours' treatment spread over 21 days are required. The primary tumour disappears in most cases. Permanency of results cannot as yet be assessed. If no bomb is available, treatment can be given either by peroral needling or by means of a collar carrying 100 mg. of radium at a distance of 4 cm. applied 12 to 16 hours daily for several weeks.

#### RADIUM TREATMENT OF CANCER OF THE LARYNX AND PHARYNX

In the treatment of laryngeal and pharyngeal cancer, radiation by X-rays and radium has made rapid strides both in technique and in results obtained. Each case of cancer of the upper air-passages should be carefully considered as to possible treatment by radium prior to any operative intervention.

The gravity of the disease cannot be exaggerated; the mortality is so great, early diagnosis so rare, and surgical excision such a formidable undertaking that the possibilities of radiation should be very fully explored.

*Larynx.* In malignant disease of the larynx, the term endolaryngeal or intrinsic cancer should be restricted to growths of the vocal cords, ventricular bands, and anterior commissure. These growths cause early symptoms and manifest their presence by hoarseness; they grow slowly, have no tendency to invade the laryngeal skeleton till late in the disease, and do not produce glandular metastases as long

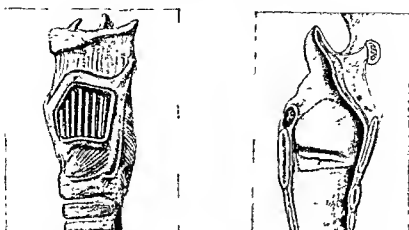


Fig. 807.—FENESTRATION OF THYROID CARTILAGE ONLY AND CORRESPONDING AREA IN THE LARYNX WHICH IS IRRADIATED. FOR EARLY CASES

as they remain truly intrinsic. They are amenable to treatment by laryngofissure with a very good percentage of cures. Radium treatment is superior to laryngofissure in functional results, but should only be attempted by those who have the necessary experience and facilities to carry out the treatment.

*Technique.* The method of treatment consists in interstitial irradiation by open operation of access. It is known as Harmer's method of fenestration of the larynx, and consists in the removal of portions of the thyroid cartilage, cricoid cartilage and hyoid according to the extent and situation of the growth. The cartilages are removed to prevent subsequent necrosis and to permit close and accurate juxtaposition of the radium needles to the lesion, without opening the air-passages. In cases of a bilateral lesion or a growth involving the anterior

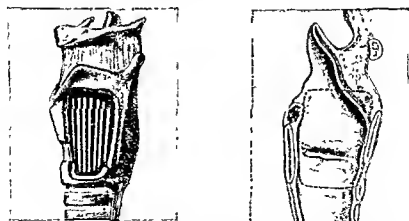


Fig. 808.—FENESTRATION OF THYROID CARTILAGE AND CRICOID CARTILAGE, SHOWING THE AREA INSIDE THE LARYNX WHICH IS IRRADIATED. IN CASES OF SUBGLOTTIC EXTENSION.

commissure, a bilateral fenestration should be done. Figures 808 and 809 illustrate the area of cartilage removed and the corresponding irradiated area in each case. The needles used should have 0.8 mm. of platinum; their length, and therefore radium content, varies from 1 mg. to 2 mg. of radium. The number of needles used varies from 7-10, and if left in position 7 days the dose varies from 1,000 mg. to 2,600 mg. hours. Following the treatment there are two distinct periods of reaction: (1) The *immediate reaction* which does not always occur, and is noticed at the end of the period of irradiation; it is mild in character, characterised by slight dysphagia, loss of voice, slight cough, and swelling of the upper aperture of the larynx. (2) The *delayed reaction*; this occurs



Fig. 803.—FENESTRATION OF THYROID AND CRICOID CARTILAGES WITH REMOVAL OF HYOID, SHOWING THE AREA WHICH IS IRRADIATED. FOR CASES WITH UPWARD AND DOWNWARD EXTENSION OF THE LESION.

3 to 4 weeks after treatment, and is more severe, the degree varying with the dose given. There is an increase of secretion of mucus, œdema of the pharynx and upper laryngeal aperture, and a good deal of discomfort; it lasts about 10 days. During the treatment the lesion can be observed, and flattening of the growth is noticed about the fifth day; filming of the lesion follows, and gradually the cords assume a normal appearance.

Fenestration of the larynx in suitable cases is the method of choice; it requires very little radium; the operative procedure is practically without risk, and the results compare very favourably with those of laryngofissure.

*Pharynx.* The growths in this region comprise: (1) The epilaryngeal group: epitheliomata of the epiglottis, pharyngeal aspect of the arytenoid and aryepiglottic folds. (2) The lateral pharyngeal group:

lateral wall of pharynx and sinus pyriformis. (3) The hypopharyngeal group: posterior wall and post-cricoid area. This classification is based not only on the anatomical origin of the lesion, but the disease in each group presents a different clinical picture with a great variation in symptoms and prognosis. There are peculiar difficulties inherent to this situation, due to the difficulty of access, interference with deglutition, respiration and speech, presence of pharyngeal mucus and proximity of the laryngeal cartilages.

*Technique of Treatment.* Radium treatment can be carried out by one of the three following methods, according to the facilities available:

(1) In the epilaryngeal group where the lesion is on the ary-epiglottic fold or epiglottis, subperichondrial resection of the ala of the thyroid cartilage with removal of the hyoid gives good access; great care should be taken to avoid opening the pharynx or larynx; needling is as described for the operation of "fenestration" (fig. 809). The needles can with advantage be sutured in position to prevent their accidental displacement and to ensure accurate and uniform irradiation. About 18 mg. in long needles of 2 mg. and 3 mg. are required. Period of irradiation, 7 days.

(2) *Surface Irradiation* by means of columbia paste or rubber collar; 100 mg. of radium placed at a distance of 4 cm. 14 hours daily for 3 weeks. This method is advocated if facilities for mass radiation are not available (see fig. 800).

(3) *Distance Mass Radiation.* This is undoubtedly the method of choice. A bomb containing 2, 3, or 5 gm. of radium is the ideal weapon for the treatment of pharyngeal cancer. Daily exposures of 1 or 2 hours, according to the quantity of radium used, are given. The distance varies from 5 cm. to 10 cm. Multiple ports of entry are made use of. Treatment is prolonged over a period of 4 to 6 weeks (see figs. 788, 789 and 790).

The results obtained are superior to all other methods, with the exception of high voltage X-radiation by Coutard's protracted fractional treatment. With this method about 15 per cent of 3-year cures are obtained.

#### RADIUM TREATMENT OF CANCER OF THE BREAST

Surgical treatment of cancer of the breast has been standardised since Halsted's operation was described in 1894 and its modification, by



the removal of the pectoralis major, two years later. Surgical statistics are in uniform agreement as regards results for a period nearly 40 years long. The best results are : 70 per cent of 5-year cures in early cases without involvement of axillary glands. This seems a most satisfactory state of affairs, but does not tally with the large numbers of recorded deaths from breast cancer. The desirability of improving the results is best seen when other factors than operability are studied. The most important factor is that only a very small proportion of cases are seen prior to the development of axillary metastases. Portman's figures show that 95 per cent of cases have glands involved when operated upon. Sampson Handley states that 70 per cent of all patients operated upon should be considered as having intra-thoracic involvement. Dean Lewis in a study of cases at Johns Hopkins Hospital from 1889 to 1931 states that although the results for the first five years are encouraging, ultimately nearly all patients die of the disease. Douglas Quick states that when all factors are analysed it is found that of all patients operated upon, 70 per cent die of the disease by the end of the fifth year, and that the majority of the remaining 30 per cent die of cancer ultimately. Under these circumstances it is important to see if radiation, alone or in combination with surgery, improves the patient's prospects.

In this respect Forsell's statistics for combined radiological and surgical treatment over a period of ten years are of great importance. In 75 cases where post-operative treatment was carried out, 29 per cent were symptomless for five years. In 45 cases of pre- and post-operative treatment, 40 per cent were symptomless for five years. The average five year result with surgery alone (in all operated cases, including those with axillary glands) is 20 to 23 per cent ; with combined treatment 39 per cent. Thus the end results are nearly 100 per cent better by the combined method. The results of pre- and post-operative irradiation are greatly superior to merely post-operative irradiation. Westermarck analyses cases that died from cancer of the breast as follows :

Untreated : Average life 31 months.

With operation : Average life 39 months.

With operation and post-operative irradiation : Average life 49 months.

With pre- and post-operative irradiation : Average life 61 months.

Local recurrence after surgery : 55·7 per cent.

Local recurrence after combined treatment : 34·6 per cent.

Portman's results with post-operative irradiation give a 5-year curability rate of 43 per cent, that is 10 per cent improvement on his own compilation of surgical results alone. Pfahler in an extensive analysis of 1000 private patients in various stages of the disease gives the following figures: At the end of five years with no treatment, 12 per cent will be alive; with surgery alone 35 per cent, with surgery combined with radiation 55 per cent will be alive. He concludes that:

"It seems, therefore, that it has been sufficiently demonstrated that even with our present imperfect knowledge and imperfect technique, irradiation is proved to be of definite value in the treatment of cancer of the breast, and that when irradiation is given skilfully and thoroughly the patient's chances are nearly doubled as compared with surgery alone."

Geoffrey Keynes, in an analysis of 171 cases of primary carcinoma of the breast, shows 46.1 per cent of three years survival in inoperable cases. Here the value of radiation is clearly superior to other methods of treatment. Geoffrey Keynes' conclusions are as follows:

"The results of radium compare favourably with those of pure surgery. No exaggerated claim is made for radium such as it should supplant surgery, but radium has a definite place in the treatment of cancer of the breast and should be used instead of, or combined with, surgery according to circumstances. The place of radium will vary with the stage of the disease and other conditions. In inoperable tumours it is the treatment of choice, and in hopeless cases remarkably good results can sometimes be obtained."

*Selection of Treatment.* The choice of treatment should be guided by the experience of the results by the various methods available. Under no circumstances should radiation be considered as a method opposed in any way to radical surgery. The sole consideration in planning treatment must be the ultimate result. It may be of practical value to tabulate the various factors which influence the choice of treatment, since from these the indication for radium can be deduced.

(1) *Importance of Age of Patient.* The gravity of the disease and the prognosis bear a definite relation to the age of onset. In patients under 40 years of age, surgery alone gives indifferent results. The fourth decade is a borderline group. After 50 years of age, surgery shows its best five year survival rate. In old patients conservative surgery equals radical measures.

(2) *Operability.* A removable growth is not always operable. Very extensive fungating tumours benefit enormously from surgical

treatment. The deciding factor is glandular involvement, fixation of the breast and type of growth.

(3) *A combination* of surgery and radium offers a better chance than surgery alone. Pre- and post-operative irradiation is of much greater value than post-operative irradiation. Of the two, pre-operative radiation is the more important. With adequate technique, it does not delay healing or complicate the operative procedure.

With these cardinal points in view, the following principles of treatment can be formulated :

(1) Early growths without axillary glands in women in the fifth decade should be submitted to radical operation.

(2) Borderline cases, from the point of view of operability, should be submitted to pre- and post-operative irradiation.

(3) Inoperable or advanced cases are better treated by radium than by surgery ; but fungating masses should in all cases be removed by a conservative operation, although such operation is quite inadequate if considered from the orthodox surgical conception of the problem.

(4) In young patients, in cases where operation is either impossible on general grounds, or refused, radium offers quite good prospects of five year survival with freedom from symptoms ; results vary from restoration to normal to the presence of a residual but quiescent mass.

(5) Treatment of cancer of a lactating breast is primarily by radium.

(6) Post-operative recurrences—in the skin, in the sternum, in the supraclavicular fossa are eminently suitable for radium treatment.

*Technique of Treatment.* The problem of radium treatment of breast cancer has been somewhat obscured by the general acceptance of " needling " or interstitial irradiation as the main method of treatment. Needling, however, is only a part of the treatment, and surface or external irradiation is at least of equal importance. The indications for each method are quite definite and the ideal radium treatment is external or surface radiation ; in general use a two-stage treatment offers many advantages.

*When Radium is the Sole Method of Treatment.* The following technique or " two-stage " treatment is adopted :

*First Stage : Interstitial Irradiation.* The needles employed vary in length, and therefore in radium content, but the linear intensity is the

same throughout. The total quantity of radium required varies with the size of the breast and the size of the tumour; 50 mg. may suffice for a small breast; in a large pendulous breast, as much as 120 mg. may be required. The object in view is not only to surround the neoplasm with radium, but to treat the whole breast and the lymphatic area. With skill, the area treated is greater than that

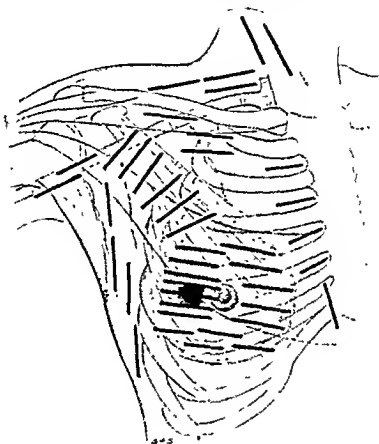


Fig. 810.—DIAGRAM SHOWING "RADICAL NEEDLING" OF BREAST.

included in the most extensive radical operation; it includes the lymphatics along the internal mammary vessels and always includes the supraclavicular fossa (figs. 810 and 811).

The *tumour* is first irradiated; long needles are placed deep to it in the substance of the breast or the pectoral muscles, as occasion demands; these form a deep barrage; a second layer is placed superficially to the tumour, at right angles to the first series. The *breast* is then irradiated; the glandular and fatty tissue is needled, the needles being placed in parallel rows in couples tied to each other, or in a circle radiating from the tumour. The *lymphatic area* is next dealt with.

(a) The *supraclavicular fossa*: needles are placed above and parallel to the clavicle and also along the line of the sterno-mastoid. (b) The *costo-coracoid area*: needles are inserted below the clavicle parallel to it if long needles are used, or vertically if shorter needles are used; the needles must be placed deep to the pectoralis minor. (c) The *anterior parts of the intercostal spaces*: the second, third, fourth and fifth spaces are needled near their sternal ends. (d) The *axilla*: needles are placed deep to the pectoral muscles from the external margin



Fig. 811.—DIAGRAM SHOWING NEEDLES IN POSITION IN BREAST.

inwards along the anterior axillary wall; the same is done along the posterior wall on the subscapular muscle; the apex of the axilla is reached through the pectoral muscles, and a few needles are placed along the brachial and axillary vessels (fig. 810). The mid-axillary line is needled from the apex of the axilla to the lower border of the costal margin. The skin punctures are sealed with mastisol, and the whole area covered with a thin layer of gauze kept in position by elastoplast.

*After-treatment.* The needles are left in position seven days; in a few exceptional cases ten days' irradiation is given. The dressings are

not disturbed. The needles are removed under evipan anæsthesia. Most needle punctures are inflamed when the needles are removed; some debris and pus can be squeezed out from each puncture hole. Hot fomentations are applied for two days; the area is then kept dry with zinc stearate powder.

*Second Stage: Surface Irradiation.* The object of the second stage is to provide an irradiation of uniform intensity throughout the area treated; it supplements the needling, which gives a very high intensity in the vicinity of the needles, but leaves spaces between the needles which must be considered as under-irradiated. External irradiation also provides the possibility of increasing the time factor up to three weeks or more, while it permits fractioning the time of treatment and allows the patients periods of rest and recuperation each day. The distance between the skin and radium varies with the quantity of radium available, from 15 mm. to 3 or even 4 cm. Various substances are employed to make plaques: sorbo ruhber and columbia paste are used extensively. The main object of the plaque is to ensure the distance between skin and radium.

A plaque or jacket is prepared for each individual case. If short radium-skin distances are used, the needles are distributed equidistantly on the outer surface of the plaque in parallel rows; the distance between two adjacent needles and two adjacent rows is such as to provide uniform distribution at a depth of 15 mm. or more. If larger quantities of radium are available (150–250 mg.) the distance is increased to 4 cm.; a jacket of thin sorbo ruhber or a thin plaster jacket is used as a foundation; wooden boxes containing at one end units of 10 or 15 mg. are placed on the jacket (fig. 812) and strapped on or sutured in position. Treatment is intermittent, 12 to 16 hours daily being given; the treatment is spread over a period of 10 to 15 days.



Fig. 812.—SORBO JACKET TO WHICH ARE ATTACHED WOODEN BOXES EACH CONTAINING 10 MG. OF RADIUM. RADIUM SKIN DISTANCE, 4 CM.

*Pre-operative Radium Treatment.* In pre-operative irradiation, the surface treatment is the method of choice: needling is contra-indicated as a preliminary treatment if operation is contemplated. The treatment is carried out by means of a jacket; 3 cm. as a minimum radium-skin distance is necessary; the treatment is prolonged and an adequate dose must be given (with 100 mg. of radium, 12 hours daily, a total of 15,000 mg. hours is given in 14 days). A period of three weeks should elapse between the end of radiation and the operation. Healing is not delayed, and there is no added difficulty in the operative procedure.

*Post-operative Treatment.* This is in every way similar to pre-operative treatment. It should commence as soon after operation as possible, even before the removal of the stitches (about the fifth day). The radium jacket is placed over the dressings. The dose varies according to the pre-operative treatment.

*Treatment of Post-operative Recurrences. Skin Nodules.* If few in number and discrete, interstitial irradiation with small needles (0.6 mg., 1 mg., 1.33 mg.) is the treatment of choice. Each nodule is undermined by one or two needles, and surrounded on all sides by a barrage of needles. Peeling of the skin at the end of the treatment is the rule. If the nodules are very numerous, and involve a large part of the flap, needling is contra-indicated; a slow treatment with a jacket will lead at the end of 4-5 weeks to peeling of the skin and disappearance of the nodules. In such cases an area of normal skin beyond the nodules should be treated, as otherwise a marginal spread often occurs.

*Supraclavicular Glands.* If a "bomb" is available this should be employed. The technique is similar to that used in the treatment of cervical glands in pharyngeal and oral lesions. An alternative is "closed" needling of the supraclavicular area (the insertion of needles without an incision of access). A "collar" in this situation is difficult to apply efficiently.

*Sternal Metastasis.* The recurrence of disease in the scar or adjoining tissues in the neighbourhood of the sternum is suitable for radium treatment and the prognosis is surprisingly good. Masses of tumour as big as an orange fungating through the skin can be made to disappear. The method of treatment is a two-stage irradiation. Needling of the sternum presents no difficulty or danger if the screenage is adequate.

## RESULTS

The first operable case of cancer of the breast treated at Westminster Hospital by radium alone is now in the seventh year free from any evidence of disease and in normal health. My personal series of cases from 1926 to 1932 show a survival rate as follows :

TABLE VIII

<i>Date.</i>	<i>Total.</i>	<i>Operable.</i>	<i>Inoperable.</i>	<i>Alive.</i>	<i>Dead.</i>	<i>Untraced.</i>
1926-7	6	—	6	1	4	1
1928	11	3	8	6	5	—
1929	43	10	33	12	28	3
1930	48	13	35	22	23	3
1931	49	18	31	26	19	4
1932	43	18	25	38	5	—
<i>Total</i>	200	62	138	105	84	11

There can be no doubt that the prognosis in cancer of the breast can be materially improved by the judicious, careful, and expert use of radium, either as the sole method of treatment or in combination with surgery. Early diagnosis remains the greatest single factor influencing the terminal results. The development of visceral or skeletal metastases depends chiefly on the stage of the disease when treatment is undertaken, but there is some indication that radiation if adequate adds a little to the length of period of freedom from disease.

## RADIUM TREATMENT OF SKIN NEOPLASMS

Cancer of the skin occurs in two main types: Epithelioma and Rodent Ulcer. Both are suitable for radium therapy, and both have given good results from the earliest days of treatment by radium. Since the publications of Wickham and Degrais more than twenty-five years ago, showing that it was possible to produce complete healing of skin cancer by radiation, methods of treatment have been developed in nearly all radium clinics on both sides of the Atlantic, and to-day these are very nearly standardised. In the case of rodent ulcers, Finzi maintains that more than 95 per cent of successful results can be legitimately anticipated. To those familiar with radium technique, the treatment by radium of skin lesions seems to be nearly as specific as that of syphilis by arsenic, mercury and iodides, and that of malaria



hy quinine. Methods of treatment vary, chiefly according to the facilities available and also with the training of the therapist. It is therefore not surprising to read certain accounts where radon emanation is given as the method of choice and others where surface application by  $\beta$  or  $\gamma$  rays is the sole method recommended. In France and Belgium external or surface irradiation is in vogue, whilst the interstitial method is considered superior in some clinics in England. All centres agree that the results are always better in lesions which have not been submitted previously to other forms of treatment. Local recurrences after surgical excision, diathermy, carbonic acid snow, cauterisation, ionisation, X-radiation, etc., do not give such good results as the non-treated lesion. Recurrence after an inefficient treatment by radium is always less susceptible to a second radium treatment and the risk of necrosis is greater.

#### RODENT ULCER

Histologically a rodent ulcer is a basal-celled carcinoma; it approaches closely the undifferentiated cutaneous epithelioma and is the most radio-sensitive cutaneous lesion. The natural rate of growth of a rodent ulcer is very slow and often spreads over many years; this is an important clinical point in differential diagnosis between an epithelioma and a rodent ulcer; it is, of course, a well-known phenomenon that very old-standing rodent ulcers, especially those irritated by ineffective treatment, undergo metaplasia and change into typical keratinising squamous-celled carcinomata. There are in connection with rodent ulcers two universally accepted statements which need correction: (1) that rodent ulcer never occurs on the hands or feet; (2) that rodent ulcer never gives rise to metastases in lymphatic glands. The occurrence of typical rodent ulcers on any part of the skin including hands and feet has been recorded, and the author has seen several such cases histologically proved. Glandular metastases are exceedingly rare but do occur, the secondary deposit in the gland showing a histological picture indistinguishable from the primary rodent ulcer in the skin.

*Clinical types.* There are three types of rodent ulcer:

(1) *Superficial type.* This is a sclerosing lesion, hardly ever reaching the underlying tissues for any depth; it spreads with a serpiginous margin and fibrosis leads to partial healing spontaneously. The lesion has a slightly raised edge, while scabbing is common, and rate of growth is slow.



Fig. 813.—RODENT ULCER BEFORE TREATMENT.

*Treatment* in this type depends upon the surface extent of the lesion ; in small ulcera not exceeding 2 cm. in diameter,  $\beta$  ray therapy presents advantages, as being a rapid inexpensive form of treatment. Monel metal plaques containing about 5 to 8 mg. of radium spread over a surface 1 cm. square screened by the thinnest layer of gold (0.05 mm.) are used. The plaque is applied directly on the lesion after removal of the scab. Applications of 40 minutes to one hour are sufficient. About 5-10 days after the treatment there is a definite reaction : erythema, increased secretion, and formation of a scab. Healing is rapid, and the scar left behind is nearly invisible. The reaction can be diminished in



Fig. 814.—RODENT ULCER AFTER TREATMENT.

severity if the treatment is fractioned in periods of 10 to 15 minutes given every day. If the lesion is more extensive in size, but still of the superficial type, gamma radiation is preferable. A small columbia paste plaque is made to cover the lesion and overlap it on all sides. 20 mg. of radium in needles are applied on the plaque, and treatment is continued for 10-12 hours daily for 5-7 days.

(2) *Proliferative type.* The lesion is of hypertrophic character and resembles a fleshy papilloma, but the skin has a semi-translucent appearance with newly formed capillary vessels at the edge. The lesion may be of considerable size before ulceration occurs, while clinically it is sometimes mistaken for a sebaceous adenoma; ulceration occurs in the centre of the growth.

*Treatment.* In this type of lesion implantation is the method of choice. The best results with the least reaction are obtained by needles screened with 0.5-0.6 mm. of platinum. Gold seeds are at times more convenient. If the latter, 0.5 mm. gold screenage should be used, and 2 cm. long seeds containing 2 millicuries of emanation are convenient in most cases. Near the canthus of the eye interstitial irradiation is definitely superior to surface treatment and safer as regards possible damage to the eye or subsequent development of cataract.

(3) *The ulcerative type.* This lesion closely resembles an epithelioma; it infiltrates and ulcerates, the base is covered with a scab, and the edge is raised. It is this type of lesion which involves underlying structures, erodes cartilage and bone, and causes great deformity (figs. 813 and 814).

*Treatment.* The periphery of the lesion is surrounded by a barrage of short needles containing 0.6 mg. of radium placed at a distance of 0.75 cm. from each other and about 3 mm. from the edge of the ulcer. The needles are left in position a week, and the treatment is supplemented by a surface plaque of columbia paste, carrying 20-30 mg. of radium applied intermittently over 10 days or more. With attention to details even old-standing and extensive lesions can be made to heal.

#### EPITHELIOMA OF THE SKIN

The commonest site of skin epithelioma is the face, but no part of the skin is immune; lesions on the hand are common; anal epithelioma, epithelioma of the vulva and of the penis belong to this group.



Fig. 310.—] PRINCELOMA OF FACE AFTER TREATMENT.



Fig. 315 — PRINCELOMA OF FACE BEFORE TREATMENT.

Multiple lesions are not uncommon, especially on the face. Industrial cancer in tar and gas workers, agricultural labourers, chimney sweeps, etc., is well known. The degree of malignancy varies on the face; with the exception of the lip, a lesion may exist for a long time, spread widely and destroy the nose, eye, or ear, without giving rise to metastases. Epithelioma developing on lupus, healed or active, malignant changes in scars, X-ray burns, and psoriasis treated by unfiltered X-rays, belong to this group. That radium is the first and best method of treatment has been established for many years. It is only exceptionally and for reasons of expediency that surgical excision is sometimes preferred to radiation.

*Treatment.* Interstitial irradiation gives excellent results, and in a shorter period of time than surface application. Needles of 0.6 mg., 1.33 mg., or 2 mg. are used; seven days' treatment is required. Radium is preferable to radon. Surface application is a second best, and is not economical in radium or in time. The resulting scar from interstitial irradiation is less visible. Treatment of the lymphatic areas by radium should only be undertaken in cases where the glands are palpable, but an expectant attitude is quite legitimate, as the glands appear very late and by no means in all cases (figs. 815 and 816).

#### EPITHELIOMA OF THE PENIS

This type of skin lesion requires special description. In competent hands with a knowledge of the disease in this situation amputation can be avoided in the vast majority of cases. Success of treatment is more common than failure, except in very advanced cases, which are beyond hope of cure, and in these amputation is permissible.

*Types of Lesion.* From the radiation point of view, carcinoma of the penis can be classified in two main groups: (1) The nodular infiltrating type—it involves the corpora cavernosa and the corpus spongiosum relatively early, and the skin is rarely ulcerated, although involved; sinuses leading to the urethra occur. This type is radio-resistant, and is not suitable for radium treatment; it occurs commonly in patients with old-standing urethral disease and strictures which require instrumentation. Such patients have little to gain from conservative treatment, but have much to gain from amputation of the penis, as even if the treatment by radiation is successful and the neoplasm is destroyed, the functional result is bad.

(2) The second type of carcinoma of the penis is a true skin lesion, which occurs on the epithelial covering of the penis, the commonest situations being the corona, prepuce and glans; clinically the lesion is either of the papillary and cauliflower type or of the ulcerative variety. It rarely involves the urethra except at the meatus, and seldom infiltrates the corpora cavernosa. Such lesions are radio-sensitive, amenable to successful radium treatment, and leave the patient with an organ which is normal in every way and functionally useful. The papilliferous and cauliflower type is more sensitive than

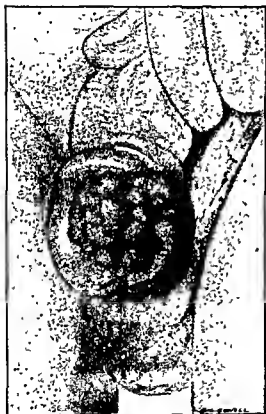


Fig. 817.—EPITHELIOMA OF PENIS BEFORE TREATMENT

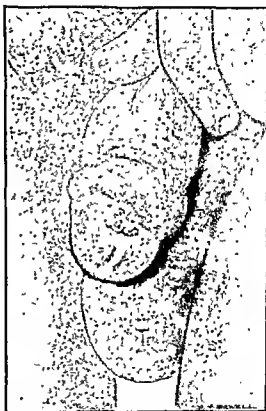


Fig. 818.—EPITHELIOMA OF PENIS AFTER TREATMENT.

the ulcerative variety, thus following the general rule of all neoplasms. Healing is rapid in the papilliferous type, but much slower in the ulcerative, especially if the glans is involved (figs. 817 and 818).

*Technique.* The treatment consists in interstitial irradiation with needles of suitable length. The total amount of radium used varies from 10 mg. to 35 mg. according to the extent of the growth; time: 7–10 days. Suprapubic cystostomy is not necessary; catheterisation is occasionally required, but as a rule micturition is normal throughout the period of treatment. Some œdema and mild infection is unavoidable,

but this causes no constitutional disturbance. It is desirable to protect the testis by covering the penis with a cylindrical shield of lead 2 mm. thick.

Treatment of the inguinal regions is imperative in all cases. The end results at the end of five years are definitely better if both inguinal regions are irradiated. Local recurrence is rare in lesions of moderate extent if the treatment is adequately given. Radiation of the glandular area by means of plaques or a "bomb" is simpler, safer, and more efficient than wide surgical excision of inguinal glands. Plaques of 75 mg. are applied to each groin, 12-14 hours daily for 14 days. Definitely enlarged and clinically malignant glands have been successfully treated by this method.

In the author's series of 34 cases of carcinoma of the penis, healing of primary growth occurred in all but 4 cases of very advanced disease (90 per cent), and freedom of disease up to periods of ten years has been reached.

#### RADIUM TREATMENT OF CANCER OF THE RECTUM

Surgery remains so far the first, the most important, and the most successful method of treatment of rectal cancer. The percentage of 5- and 10-year cures is relatively high, and the immediate operative mortality diminishes year by year. But although the operation itself is as satisfactory as any excisional procedure in the treatment of malignant disease, it is difficult to remain content with excision as the sole weapon against rectal cancer if it is remembered that only a small proportion of cases fall into the group suitable for surgical removal (40 to 60 per cent).

Radium, on the other hand, has given very few good results, and it is no exaggeration to say that with few exceptions most surgeons and most hospitals have practically abandoned radium as a method of treatment. It is therefore most important to review the position of radiation in this site, and to show what has been learned in technique and what is the true present position.

*Operability.* Of all the factors to be assessed in the study of cancer of the rectum, "operability" is perhaps the most difficult. The definition of operability depends upon many factors, such as the general condition of the patient, the local condition of the growth, the skill, courage, and judgment of the surgeon. Certain clinical findings are considered by most as definite signs of operability; of these the degree

of mobility of the rectum is the most important. Fixation of the bowel to surrounding structures is a limitation of operability generally recognised, but even this may be due to inflammatory changes and sometimes disappears after colostomy. The figures of operability vary in different countries. The highest figure is 61 per cent of a total of 1118 cases quoted by four American authors. The lowest figure is 35 per cent of a total of 337 cases quoted by four authors in Switzerland. In England the average percentage of operability is 38 per cent from a total of 1312 cases quoted by four authors. W. B. Gabriel, in a survey of the cases at St. Mark's Hospital, gives the operability rate as 54 per cent (in a total of 698 cases from 1921 to 1931). The percentage of operability is always higher in women than in men, owing to the greater roominess of the pelvis, the presence of the pouch of Douglas, and the possibility of the excision including the vagina if considered necessary.

In an analysis of 6000 cases of cancer of the rectum in which operations for radical cure were performed in ten different countries, the Ministry of Health report arrives at the following conclusions: (1) On the average a period of twelve months is allowed to pass between the occurrence of the first symptom of the disease and the operation. (2) Less than half of the total cases when seen by a surgeon are considered operable. (3) A sixth of all the operated cases died as the result of the operation. (4) The more advanced the stage of the disease the higher the operative mortality. (5) Two patients out of every five submitted to a radical operation are alive three years afterwards. In a very important contribution on the Radium Problem, Sir Charles Gordon-Watson and Cuthbert Dukes state that from their observations on the spread of cancer of the rectum they arrived at the following conclusions of interest in relation to radium treatment: (1) In three-quarters of the cases of cancer of the rectum accepted by surgeons as operable, the cancer has already spread by direct continuity into the peri-rectal tissues. They call these C cases. (2) In more than half of these C cases, the ano-rectal or retro-rectal lymphatic glands contain metastases. (3) Cancer of the rectum commences in the mucous membrane and extends slowly in direct extension through continuity of tissue on the surface and by infiltration of the rectal wall. As a rule, lymphatic dissemination does not play any part in the spread of cancer of the rectum until the growth has spread by direct continuity into the peri-rectal tissues.

*Radium Treatment.* The indifferent or even bad results obtained by radium in rectal cancer and the inevitable feeling of disappointment



which followed are due to several factors. These can be briefly summarised as follows: (1) Columnar-celled carcinoma is more radio-resistant than other forms of cancer and requires treatment which is highly specialised, as otherwise a burn results without arrest of the disease. (2) Access to the growth is very difficult, and uniform irradiation demands a technique of the highest order. (3) Sepsis is practically inevitable and limits the possibilities of radiation; it leads to local infection and peritonitis, and excludes certain routes of approach on account of the complications and high mortality. With adequate technique it is still our opinion that radium offers something to the patient with rectal cancer, and that the possibility of permanent relief can be given in a few selected cases. It should never take precedence over surgery, which must be urged in every operable case.

The indications for radium are therefore as follows:

- (1) Borderline cases in which operability is doubtful.
- (2) Inoperable cases.
- (3) Operable cases in patients in whom operation is contra-indicated or refused.
- (4) The refusal of colostomy.
- (5) Pre-operative irradiation—providing that the treatment is correctly carried out, in cases in which operation must be delayed.

#### CARDINAL PRINCIPLES OF IRRADIATION OF THE RECTUM

*Colostomy.* This should be carried out in all cases. To avoid colostomy is not the primary object of irradiation, and there is no doubt that the risks of radium treatment are definitely diminished by a preliminary colostomy. It is possible, but not always advantageous, to avoid colostomy.

*Irradiation.* It is essential in this group of cases to administer pure gamma-therapy. All primary beta rays should be screened; as much as possible of the secondary beta rays should be eliminated. The time factor is of equal importance to filtration, and it is the experience of most workers that rectal cancer requires prolonged irradiation if success is to be obtained.

*Methods of Irradiation.* Most methods of irradiation have now been abandoned, and the following is described as the only method in which the risk of radiation is small, post-radiation pains negligible, and results encouraging.

*Radium required.* The following radium is necessary to carry out the treatment:

- (a) 40 mg. of radium, preferably in tubes of 10 mg. each; overall length, 20 mm. Screenage, a minimum of 1 mm. of platinum.
- (b) Needles of 1 mg. per cm. active length, screened by at least 0.8 mm. of platinum, about 30 or 40 mg. are required.

*Technique.* Irradiation consists of a simultaneous intra-rectal and peri-rectal insertion of radium in such a manner that the tumour is submitted to the effects of radium from the periphery from a series of weak foci of irradiation and from the lumen of the bowel from a relatively high source of radiation.

*Peri-rectal Irradiation.* High lithotomy position is necessary. The coccyx is removed, but otherwise no surgical exposure is necessary. The needles are inserted by puncturing the skin and guiding their

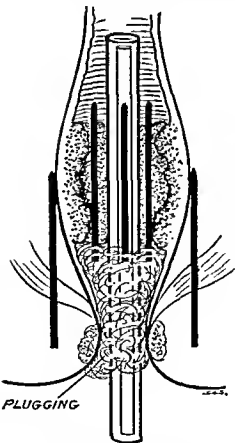


Fig. 819.—PERI-RECTAL NEEDLING AND INTRA-RECTAL CAVITARY IRRADIATION.

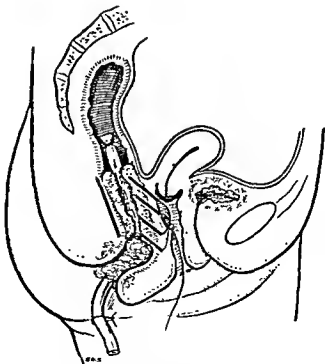


Fig. 820.—TRANSVAGINAL AND POSTERIOR NEEDLING. INTRA-RECTAL TUBE IN POSITION

position by a finger in the rectum. In the male a metal sound is introduced in the urethra and the prostate and urethra brought down as near the perineum as possible; in the female the anterior group of needles are placed through the vagina (figs. 819 and 820).

*Intra-rectal Radium.* Three or four tubes of 10 mg. each are used. They are inserted in a rubber tube of 1 mm. thickness and a knot tied in between each tube. The total length of the tube is about 10 inches. This is inserted with the aid of a proctoscope into the lumen of the growth (fig. 821). The rectum is tightly plugged so as to keep the tube in position; the end of the rubber tube is sutured to the anus in two places (fig. 822).

Both sets of radium are left *in situ* seven to ten days. On removing the radium the growth is seen to be flatter and the surface covered with a film of white fibrin. The immediate results are encouraging, and the above irradiation appears to be a step in the right direction. It is far too early to express an opinion on the ultimate results.

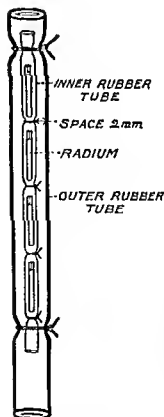


Fig 821.—DIAGRAM OF INTRA-RECTAL RADIUM APPLICATOR.

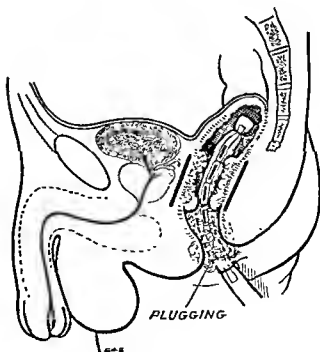


Fig 822.—PERI-RECTAL AND INTRA-RECTAL IRRADIATION.

*Seeds.* Some workers advocate the use of seeds instead of needles. They are much easier to introduce and sometimes lead to complete arrest of disease. The introduction of seeds direct into the growth through the sigmoidoscope or proctoscope is, however, not an ideal method, and presents definite risks of post-radiation hæmorrhage, which may prove fatal. Further study in this method is urgently needed.

#### CARCINOMA OF THE ANAL CANAL

This is a squamous-celled neoplasm, in no way different from a skin cancer in any other situation. It is eminently suitable for radium treatment. The ideal method of radiation is by the interstitial method, with needles in preference to seeds. Gabriel quotes 50 per cent success in this type of case, from the cases at St. Mark's Hospital. A greater percentage of success should be obtained if the lesions are completely surrounded by radium and treatment is prolonged for at least seven days.

Surface treatment of inguinal glands should be carried out in all cases of anal carcinoma. Plaques of spongy rubber, felt, or columbia paste are applied to each groin. If the radium skin distance is 4 cm., 100 mg. of radium to each groin are applied 12 hours daily for 10 to 14 days. If a distance mass radiation apparatus is available, this can very conveniently replace the plaques, and in such cases hospitalisation is not essential.

It should be noticed that neither at the Radium Institute in Paris, nor at the Radiumhemmet in Stockholm, is radium favoured for rectal cancer. It is, however, essential that further work in this direction be carried out, and the future remains more hopeful than at first seems to be the case on perusal of the literature on the subject. Sir Charles Gordon-Watson has several inoperable cases alive and free from disease for periods between 5 and 7 years.

#### RADIUM TREATMENT OF CANCER OF THE BLADDER

Carcinoma of the bladder presents problems of its own, and it is perhaps one of the anatomical sites of malignant disease where the opinion on radium therapy varies most. Barringer on one hand reports for the past ten years repeated successful treatments; on the other hand, urologists in this country are nearly unanimous in pointing out the futility of radium in bladder cancer. The position has been clearly

defined in a recent publication by Durden Smith, and the value of radium is illustrated by this author in the statistics of the Radium Institute (London), (table IX).

TABLE IX  
DURDEN SMITH, RADIUM INSTITUTE, LONDON

Carcinoma of bladder.	Number treated.	Cystoscopic method.	Suprapubic route.	Alive over		
				3 yrs.	4 yrs.	5 yrs.
Papillary . . .	23	20	3	3	2	5
Infiltrating . . .	15	10	5	1	1	1
Total . . .	38	30	8	4	3	6

Barringer reports 75 per cent of cures, the majority over five years in the papillary type, and 35 per cent of cures in the infiltrating variety. Burnam has obtained 13 cures among 24 operable cases, or 54.1 per cent. In inoperable cases, Burnam quotes 7.5 per cent of cures in 80 cases.

The value of radiation in cancer of the bladder, as elsewhere, entirely depends upon knowledge of the disease, knowledge of radiation, and the proper selection of cases and methods of treatment.

#### METHODS OF TREATMENT

*Endoscopy.* The introduction of radon seeds through an operating cystoscope has quite naturally attracted a great deal of attention from the urological surgeon. It presents many advantages, but is only suitable for a very selected group of cases, namely, when the neoplasm is small in extent, situated in a position where the whole lesion can be visualised, and each seed placed very accurately in position. Difficulties associated with cystitis, hæmorrhage, and extent of the growth restrict its range of usefulness. If seeds are used, non-removable platinum or gold seeds (0.5 mm. filtration), containing 1 mc. to 1.5 mc. per seed are used.

*Cystostomy.* In the large majority of cases this is the method of choice. Wide exposure, perfect illumination, and adequate suction are essential. The lesion, if papillary in type, is removed sub-totally with the diathermy loop. Needles of low radium content are used,

varying from 0.6 mg. to 2 mg. The base of the growth is underlined with the needles and the periphery surrounded by a corona of small needles. If removable seeds or needles are used, the threads are tied in a bunch and cut short except for one thread, which is brought out through the suprapubic wound beside the drainage-tube, and which acts as a guide for the subsequent removal of the needles (fig. 823). The needles are left in position 10 days. Following irradiation, bleeding is arrested in the majority of cases. In successful cases the ulcer takes about two months to heal, and the end result shows a pale thin scar.

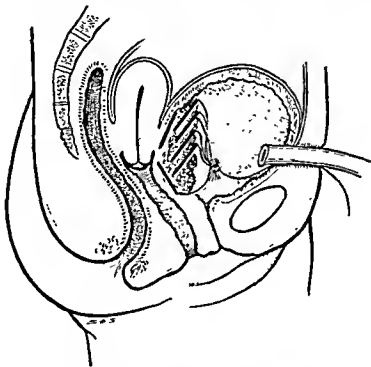


Fig. 823.—NEEDLING OF CARCINOMA OF BLADDER.

Neither permanency of results nor a high percentage of cures can be claimed, but it is an established fact that the papillary type is sensitive to radiation and that the ulcerative type if sufficiently early (not involving the extra-vesical tissues) can be benefited from radiation. As contrasted with total cystectomy or partial cystectomy necessitating the transplantation of both ureters, radium offers a safer method of treatment in a disease in which neither method can claim very much in ultimate results.

#### RADIUM TREATMENT OF CARCINOMA OF THE PROSTATE

Radium occupies a useful place in the treatment of malignant disease of the prostate. Although only very few permanent cures are

recorded, palliation is obtained in the majority of cases. Histologically the prostatic neoplasm is not unlike that of the breast, and the response to radiation is good. The ultimate lack of permanent results is due not so much to the local condition as to the development of skeletal metastases. Inoperability and extra-prostatic extension are not always contra-indications to treatment; on the contrary, in such cases radiation is the first line of treatment.

Methods of treatment vary with individual cases. In every case preliminary X-radiation is indicated. If obstruction is present an excellent alternative to suprapubic drainage is per-urethral resection with the

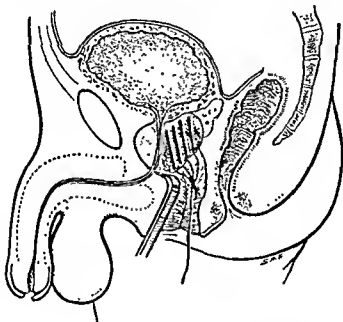


Fig. 824.—PERINEAL NEEDLING OF CARCINOMA OF THE PROSTATE.

diathermy. The scheme of treatment in such a case should be preliminary X-radiation, per-urethral diathermy, and perineal insertion of needles. If the general health and adequate urinary function permit it, an "open" needling should be done. The perineal exposure of the prostate as described by Winsbury White is the best method of approach (fig. 824), it gives an excellent exposure and permits an accurate distribution of needles. Adequate screenage is essential, as the needles are left in position up to ten days. Each lobe of the prostate is needled both at the periphery and near the inner part of the gland; in this way two circles of radium foci are inserted into the prostate and the bladder is not perforated. The average number of needles used is 12, and the total quantity of radium varies from 18 to 30 mg. As a

palliative measure, radium fills a useful place in the treatment of these cases.

Blind needling through the perineum with a guiding finger in the rectum, or the insertion of seeds, is not such an accurate method of treatment, and is used when the open method is contra-indicated.

By rectal examination it is possible to follow the gradual shrinking of the prostate, and supplementary treatments can be given if recrudescence of the growth is noticed. Post-radiation fibrosis does not as a rule affect the urethra or ureteric orifices if the perineal approach is used.

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## SECTION 2

### RADIO-THERAPY IN DISEASES OF WOMEN

by

MALCOLM DONALDSON

At first sight it might seem a little unusual to devote a chapter to a special type of treatment rather than to discuss such treatment under the disease to which it is applicable. The reason for this procedure is that radio-therapy is comparatively new, and although good work has been done in certain centres in England, yet it has not been tried out in this country to the extent that it has on the Continent. The result of the half-hearted attempts at treatment carried out in many places, and insufficient knowledge of the correct technique, has led to poor results and gained for radio-therapy an adverse criticism which is not justified.

Although any gynæcologist who intends to treat patients with radium must already have a fair knowledge of the physics of radium, yet it may be a help to state briefly a few physical and biological facts, the knowledge of which is essential to the carrying out of the proper technique.

*Radium Element.* Radium is a white metal which rapidly becomes oxidised and has an atomic weight of 226.5. It is one of a series of substances known as "radio-active." All "radio-active" substances have the characteristic of disintegration into other substances at the same time as a large amount of energy is liberated in the form of rays. Although this disintegration is going on continuously, in the case of radium it takes 1590 years for a given quantity to lose half its activity. The result of the first stage of disintegration of radium is a substance which is gaseous at ordinary temperatures called radium emanation or radon. With the production of this radon an alpha particle is given off.

*The Alpha Particle.* The  $\alpha$ -particle has a mass equal to four times that of hydrogen, and is, in fact, an atom of helium with a positive charge of electricity. Its velocity is 15,000 miles per second, but in the air it is stopped after travelling a few inches, or if it encounters any material as thick as a cigarette paper. From this it is seen

that it can be of no value as a therapeutic agent, as it can never get outside the walls of its container.

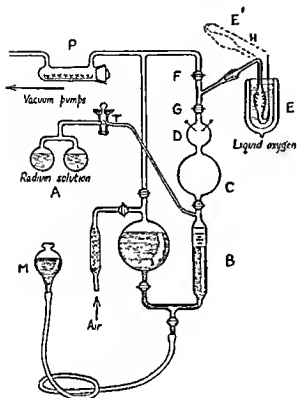


Fig. 825.—RADON OR EMANATION PLANT.

Radon is drawn off from the radium in A by lowering the reservoir of mercury M. The radon is then forced from B into C and D where any free hydrogen and oxygen is got rid of by an electric spark. The radon is then forced into the capillary tube H and solidified by placing the capillary tube H in liquid oxygen E. All the air is then extracted from H by vacuum pumps and the radon allowed to become gaseous once more. Finally, the glass capillary tube H is cut into short lengths placed in gold or platinum covers and used as seeds.

3.825 days. During this disintegration it gives off  $\alpha$ -particles and becomes Radium A.

*Radium A. B. & C.* Radium A. has a very short life, and in three minutes is reduced to half its value. It gives off  $\alpha$ -particles and becomes Radium B., which in turn becomes Radium C., and Radium C'.

These in turn break down into other radio-active bodies which, however, from a therapeutic point of view, are of no interest (fig. 826).

In radio-therapy the radio-active bodies which are of most importance are Radium B. and Radium C. It is from these bodies that the Beta and Gamma rays are emitted. It will be seen, therefore, from what has been said in the foregoing paragraphs that whether radium element, or emanation gas, i.e. radon, is put into a container the same therapeutic agents, namely Beta and Gamma rays, are emitted. The great difference in using these two sources of radiation is the fact that

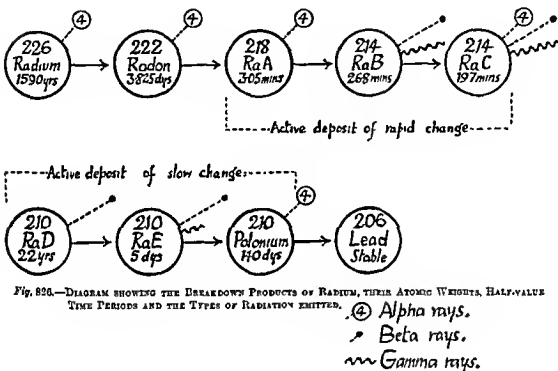


Fig. 826.—DIAGRAM SHOWING THE BREAKDOWN PRODUCTS OF RADIUM, THEIR ATOMIC WEIGHTS, HALF-VALUE TIME PERIODS AND THE TYPES OF RADIATION EMITTED.

the amount of Beta and Gamma rays given out by radium element is constant, whereas that given out by a container filled with radon is diminishing very rapidly so that at the end of 3.825 days only half the amount of energy is being emitted.

**Beta Rays.** Beta rays, sometimes spoken of as  $\beta$ -particles, are electrons travelling at a high velocity, approximately 186,000 miles per second. This is very nearly the speed of light waves. These  $\beta$ -radiations are more readily absorbed than the Gamma radiations, and for this reason have a much greater effect on living tissue. For the same reason they have a much more limited area of action. These Beta radiations can be obstructed by 0.5 mm. platinum. Thus, if radon or radium is placed in a container which has a wall the thickness of which is equivalent to 0.5 mm. platinum, no Beta rays will be

emitted from the radium or radon contained therein. On the other hand, it must not be forgotten that if the wall of the container is made of a dense material such as metal, the radiation from the contained radium will hit the container wall and set up secondary  $\beta$ - and  $\gamma$ -radiations of smaller intensity but of longer wave-length. This will be referred to again in the paragraph discussing filters to be used in radio-therapy.

**Gamma Rays.** These radiations are electro-magnetic waves which have a uniform velocity but vary in wave-length, the shorter waves being more penetrating. The amount of energy given out by these

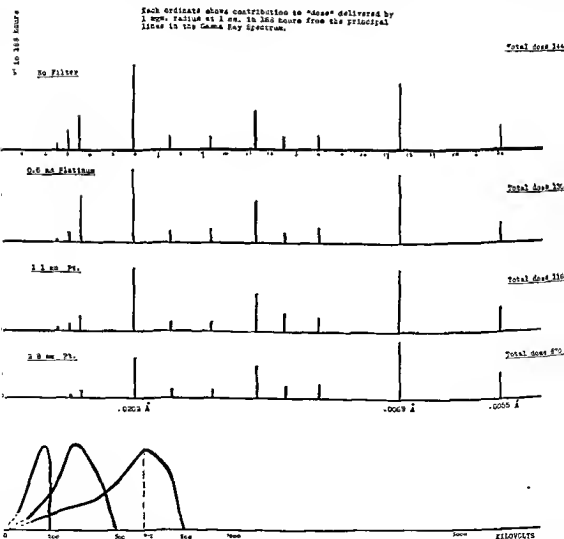


Fig. 827.—THE UPPER PORTION OF THE DIAGRAM SHOWS THE RELATIVE INTENSITIES OBTAINED FROM THE DIFFERENT LINES IN THE GAMMA RAY SPECTRUM WHEN DIFFERENT THICKNESSES OF FILTER ARE USED.

THE LOWER PORTION OF THE DIAGRAM IS PUT IN TO SHOW A COMPARISON BETWEEN THE WAVE-LENGTHS OBTAINED BY X-RAYS OF VARIOUS VOLTAGES AND THOSE OBTAINED BY RADIUM.

different wave-lengths is not equal in all parts of the radium spectrum (fig. 827). It will be seen from this figure that the energy is chiefly given out by wave-lengths of  $0.0202\text{\AA}$  and  $0.0069\text{\AA}$ .

It is these Gamma radiations that are most used in radio-therapy.

In the same way that Beta radiations give rise to secondary Beta radiation when they come in contact with a metal of high density, so Gamma radiations give rise to Beta radiations and other Gamma radiations when passing through the walls of the container. The secondary Gamma radiation is of a longer wave-length than the original.

*Radiation from an X-ray Tube.* The radiation from an X-ray tube is of the same nature as Gamma radiation, but of a different wave-length. These X-radiations are produced by detaching the electrons

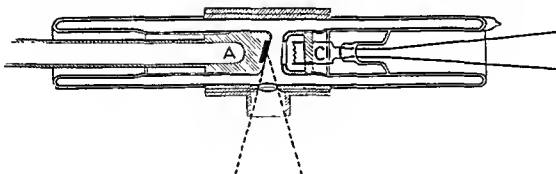


Fig. 828 — A SIMPLIFIED DIAGRAM OF AN X-RAY TUBE SHOWING THE CATHODE C AND ANODE A.

from atoms and projecting these electrons at a very high velocity against a piece of metal (fig. 828). The electrons are detached from the atoms of the metal composing the cathode (C), and by means of an electric current of high voltage they are passed through the vacuum and impinge on the metal anode marked A, the X-rays emerging through the side of the tube marked X. The velocity of these electrons passing through the vacuum depends on the voltage. The wave-length of the X-rays which are produced by the impact of the electrons on the anode depends on the velocity of the electrons hitting the anode. *The higher the voltage the greater the velocity of the electrons and the shorter the length of the waves produced.* The general tendency in radio-therapy is to use radiation of short wave-lengths.

In the case of radium therapy the short wave-lengths are obtained by cutting out the long waves by means of filters. In the case of X-ray therapy the short wave-lengths are obtained by using very high voltage currents and cutting out the longer wave-lengths by means of filters.

*Comparison of Gamma- and X-Radiations.* It is not uncommon to hear clinicians speak of radium therapy and X-ray therapy as if they were entirely different, or even antagonistic to each other. Nothing is farther from the truth. X-radiation as will be seen by the diagram (fig. 827) has wave-lengths which, as stated above, vary with the voltage used to propel the electrons through the tube. The rays produced, however, are not homogeneous, so that they can best be represented as a curve. For instance, the radiation given out by a machine which works at 800,000 volts would give a small proportion of rays equivalent to 0.0159 ångström units, but a far bigger proportion of a slightly longer wave-length, namely, 0.0202 ångström units.

If a comparison is made of the intensity—by that is meant the amount of energy falling on a unit area during a unit of time—it is found that the energy that can be produced by such an X-ray tube is equivalent to something like 40 grammes of radium. It will be seen, therefore, that with X-rays it is easy to obtain large quantities of radiated energy, but difficult to get very short wave-lengths. With radium, on the other hand, the short wave-lengths are easily obtained, but it is difficult to obtain a large amount of radiated energy.

One of the problems in the treatment of cancer with which radiotherapists are confronted at the present time is to obtain evidence as to the relative importance of a short wave-length compared to longer wave-lengths, and whether a large intensity of irradiation used over a short time is superior to a small intensity over a longer time. Experience up to the present time suggests that radiations of very long wave-lengths, such as Beta radiations, are too readily absorbed and have a necrotic effect on the tissues, which is in most cases detrimental. On the other hand, there is at present no evidence that the beneficial effects of radium are due to the very shortest wave-lengths, namely, those of 0.0055 ångström units. It is possible that most of the effect is due to the wave-length of 0.02 ångström units. If this should be proved in years to come, then it is highly probable that X-ray therapy will to a very large extent take the place of treatment by Gamma rays produced by radium.

It is hoped that in the near future a very interesting series of observations will be made at St. Bartholomew's Hospital, the Radium Institute, London, and Mount Vernon Hospital, Northwood. The work which is to be carried out at the Radium Institute consists of treatment by means of a mass of radium of 3 grammes and 5 grammes, and the work proposed to be carried out at St. Bartholomew's Hospital and Mount Vernon will consist of a number of observations on treat-

ment by X-rays of very short wave-length, produced by a machine worked by a current up to 1,000,000 volts.

*Biological Action of Radium.* Much work has been done and continues to be done on the biological action of radium. When radium was first used for the treatment of malignant disease many people considered that it acted as a cautery and destroyed all tissues within its range with equal facility.

It is true that in the case of Beta rays there is very little difference in their action on the various types of tissue through which they pass and are absorbed. In the case of Gamma rays histological sections suggest that the effect on some cells is greater than on others. The problem arises as to whether these effects produced by Gamma rays are directly on the cells or are an indirect result caused by changes in the blood-vessels, which in turn is the result of direct action on the endothelial cells of a vessel. Without going into detail of the evidence in favour of *direct* or *indirect* action of radium on malignant disease, it will be useful just to give a short summary of such evidence as there is.

*Direct Action.* In the case of tissue culture it has been proved beyond doubt by Strangeways, Spears and others, that the cells are at first inhibited from dividing and that later they die. The dose required to bring about the death of the cells in tissue culture is very great compared to that used in radio-therapy, but the dose required to upset the mechanism of mitosis is quite small.

This change in mitotic activity forms a very sensitive indicator, and it has been demonstrated that the results differ with an exposure varying by only 30 seconds.

*Indirect Action.* There is also considerable experimental evidence to show that the indirect action of radiation is very important. Strangeways and Fell in 1927 proved that there was a different action on the chick embryos after they had developed a vascular system from that on embryos at an earlier stage in their development. The importance of the indirect action is emphasised by the recent work of Dr. Pullinger, who appears to believe that the indirect action brought about by the changes in the blood-vessels is more likely to be the cause of the disappearance of the growth rather than the direct action on the malignant cells.

For many years it has been suggested that the indirect action may be partly due to the formation of immunising bodies. Russ irradiated



animal tumours outside the body and then innoculated them into other animals. Subsequent inoculation of living tumours into these animals failed, in a large number of cases, to produce growth. The same author states that animals treated with a general X-radiation have definitely raised resistance to transplanted tumours.

Clinically, it is impossible to solve the question of direct versus indirect action. It is true that histological changes can be demonstrated in carcinomatous tissue at the end of twenty-four hours' irradiation. Mitosis by this time has ceased, and sections taken two days later show further changes in the cancer cells. This, however, is not absolute proof of the direct action on the cells, as by this time there are also marked changes in the blood-vessels.

Tod has suggested that the action of radio-therapy takes place locally at the junction of normal and malignant tissues by stimulating some defensive action.

The Swedish School, among others, holds the view that radio-therapy affects the tumour sufficiently to allow the normal tissues to resist and to absorb the irradiated growth. Further, they believe that any overdose to these normal tissues tends to diminish the power of resistance, and that the growth temporarily checked by the direct action may ultimately increase quicker than before.

In addition to the above problem of whether the action of radium is chiefly direct or indirect on the cancer cells, there are many other questions to which an answer has not yet been given. For instance, do waves of different wave-lengths have varying effects on living tissue? It has already been stated that the longer wave-lengths, either from radium in the form of Beta radiation or from X-rays, are absorbed more readily than Gamma rays and have a greater necrosing effect.

Warnford Moppet did some experiments with X-radiations which suggested that certain wave-lengths caused destruction and others specific stimulation. These results, however, have not yet been confirmed.

Browning and Russ showed that the germicidal action of ultra-violet light on bacteria stopped abruptly at a wave-length of 2960 Å.

*Clinical Observations on the Action of Different Wave-Lengths.* To obtain any clinical evidence of the different results obtained by different wave-lengths is extremely difficult. It is quite impossible at the present time to obtain sufficient energy from an homogeneous beam of X-rays. It is, however, the general impression of all clinicians that by using shorter wave-lengths better results are obtained. There are

several possible explanations for this beneficial result. First, it may be due to the absence of the necrosing effect of the Beta radiation, or long wave X-rays. Secondly, it may be that by using short wave-lengths the cells are not absorbing energy so quickly, and that the continued spraying of the cell by small amounts of energy leads more readily to its ultimate death. Thirdly, the short wave-length may have a specific action on some of the chemical changes taking place within the cells.

*Radio-sensitivity.* Yet another difficulty confronts the clinician when treating malignant disease in different parts of the body. He finds that a dose which is sufficient for a certain growth in a certain part of the body is of little value in producing an effect on other growths elsewhere. Even among the normal tissues of the body there is a marked difference in the effect of a given dose of irradiation. For example, a nerve fibre is practically unaffected by a large dose of irradiation, whereas the ovaries and lymphoid tissues are very sensitive.

It is well known that quickly-growing tissues are more radio-sensitive than those in which metabolism proceeds at a slower rate. This greater radio-sensitivity, sometimes spoken of as the law of Bergonié and Tribondeau, must not be misinterpreted as is so often done, to mean that a cell is more radio-sensitive during the actual process of mitosis. The whole problem of radio-sensitivity is of very great importance, but it is not likely that it will be really understood until the fundamental changes in tissues, be they chemical or electrical, brought about by radiation have been discovered.

*Clinically, some tumours respond to radiation far more readily than others.* Speaking generally, squamous-celled carcinomata respond more readily than columnar-celled, and sarcomata more readily than either. It is unlikely, however, that it is the type of cell that affects the radio-sensitivity, as Mottram has shown that cells of different radio-sensitivity in the animal are themselves equally sensitive *in vitro*. Clinically I have observed that secondary deposits of columnar carcinoma in the vagina are very radio-sensitive.

Three possible explanations for these differences have been suggested :

- (1) The rate of growth.
- (2) Difference in response of the tissues surrounding the tumour, i.e. the tumour bed.
- (3) Difference in the radio-sensitivity of individual cells.

This last idea has led many people to classify the sensitivity of growth according to the histological findings. For instance, Broders in 1920 classified growth in four grades :

- Grade I. Where three quarters of the growth is differentiated epithelium and one quarter undifferentiated.
- Grade II. Where the differentiated and undifferentiated are about equal in number.
- Grade III. Where the undifferentiated epithelium forms about three quarters and the differentiated about one quarter of the growth.
- Grade IV. Where there is no tendency at all for the cells to differentiate.

It does not appear that these classifications are of any very great practical value, because sections taken from different parts of the same tumour vary considerably in the relative proportions of the differentiated and undifferentiated tissues.

The radio-sensitivity of glandular metastases is of very great practical importance, as at the present time glandular metastases are holding up the advance of radio-therapy, and might be compared to the "pill boxes" built by the Germans during the war.

If a number of clinicians are asked as to whether glandular metastases are radio-sensitive there may be a divergence of opinion, but all will agree that they are less sensitive than the primary growth, and even those who say that they disappear rapidly under the effect of radiation agree that recurrences in the glands after treatment are rather the rule than the exception.

There is no doubt that a gland with carcinoma in it rapidly gets smaller, but it is more than probable that the diminution in volume is due to the diminution in the gland tissue which is extremely radio-sensitive, rather than to the destruction of the carcinoma cells. Indeed, it may well be that the difficulty in treating secondary deposits in the glands is due to the fact that the tumour bed is so sensitive that the resistance of the normal tissue is destroyed more readily than the malignant cells.

In summing up the biological action of radium it may be said that :

- (1) There is experimental evidence of direct and indirect action, but there is at present no evidence as to which of these factors is the more important.

- (2) There is clinical evidence that indirect action, i.e. cutting off of blood supply, takes place with the formation of fibrous tissue, and that the malignant cells are affected by this process to a greater extent than the surrounding more stable cells. There is, however, no evidence of how great an effect the direct action has on the malignant cells.
- (3) There is abundant evidence that the final result of radio-therapy, whether due to direct or indirect action, is selective against the malignant growth, and in certain cases leaves little or no scar tissue.
- (4) There is no experimental evidence to prove that there is any specific action in the different wave-lengths on individual cells, but there is a great deal of clinical evidence to suggest that short wave-lengths are far more efficient in treating the growth than long wave-lengths.
- (5) There is abundant evidence of the difference in radio-sensitivity of different tissues and possibly of the same type of tissue in different parts of the body. There is no definite evidence whatever to show on what this radio-sensitivity depends, although many suggestions have been made. It is a subject which is of very great importance to radio-therapy, and the solution of which might be the turning point in the treatment of malignant disease quite apart from radio-therapy.

*Method of Applying Radium.* From what has been said it will be realised that whether radium or radon is used, the action is exactly the same, but in the latter case the intensity diminishes rapidly and is only half the initial intensity at the end of three and a half days. There are four distinct methods by which radium or radon can be applied :

(1) *Surface Irradiation.* If the growth is subcutaneous the radium can be applied in a flat box or plaque directly to the surface of the skin (fig. 829, photograph of plaques).

(2) *Cavitary Method.* The applicator in this case is put into one of the natural cavities of the body, for example, the antrum of the nose, the vagina, or the cavity of the uterus (figs. 830 and 831, X-ray photos showing tube in position). In theory this method has a serious defect in that the radiation is unequally distributed, the tissues near the applicator getting a very large dose compared with those at a distance. In practice, however, some of the most successful techniques in gynaecology

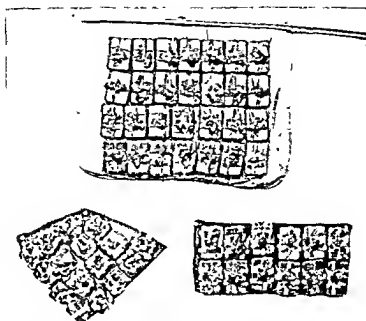


Fig. 820.—SHOWS THREE PLAQUES EACH MADE UP OF A NUMBER OF UNITS



Fig. 821—SHOWS AN X-RAY PHOTOGRAPH OF A SINGLE RADIUM TUBE IN THE CERVIX AND BODY OF THE UTERUS. THIS IS THE USUAL METHOD EMPLOYED IN PRODUCING AN ARTIFICIAL MENOPAUSE.

are based on this method. (For example, the Stockholm technique, the Paris technique, etc.)

(3) *Interstitial Irradiation.* By this method the radium or radon is implanted into the tissues. The radium is placed in hollow needles which are generally made of platinum (fig. 832, photo of X-ray showing needles in position). There are several advantages in using this metal, since it has a very great density and the walls

of the needle do not require to be so thick as if other metals are used. These needles are sterilised by boiling or by putting into

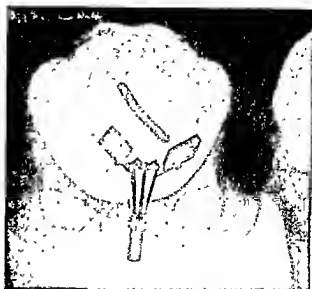


FIG. 831.—SHOWS AN X-RAY PHOTOGRAPH OF A RADIUM TUBE IN THE CERVIX AND BODY OF THE UTERUS, AND A "BUTTERFLY" PESSARY CONTAINING RADIUM, SPREAD OUT IN THE LATERAL FORNICES OF THE VAGINA. THIS IS USED IN CASES OF CARCINOMA OF THE CERVIX



FIG. 832.—X-RAY PHOTOGRAPH OF NEEDLES USED IN A BREAST CASE. THEY ARE NOT SO MUCH USED IN GYNECOLOGICAL CONDITIONS.

antiseptic solution, but care must be taken to avoid any mercury solution which would erode the platinum. Instead of using needles, sometimes small cells of platinum, gold, or silver are used to contain the radon and are inserted deeply in the tissues, and left permanently *in situ* or withdrawn by means of an attached thread at the end of a number of days. These containers are spoken of as "seeds," and are never used except with radon (fig. 833, "Seeds").

The interstitial method of applying radium is used in such conditions as carcinoma of the lip, the breast,

and the tongue, etc., but is not used extensively in gynæcology. The advantage of this method is the more homogeneous irradiation obtained. The disadvantage is the slight necrosis which takes place in the tissues immediately adjacent to the needle, and this ill-effect is increased if the needles are used in a septic area like carcinoma of the cervix.

(4) *Distance Therapy.* The ideal way of irradiating any tumour or portion of tissue is to use a parallel beam of rays from a distance. By this means the difference of intensity on the surface of the tumour and in its depth is considerably less than when the source of irradiation is close to the tumour. Since, however, the intensity falls off rapidly when the source of irradiation is at a distance, it requires a very large quantity of radium to get sufficient intensity falling on the tumour to be of any value. To obtain sufficient intensity, at least one gramme of radium is necessary (fig. 834), but it is better to have 4 or 5. When it is necessary to irradiate a large area of skin and not necessary to irradiate deeply in the tissues, a shorter distance between the source of irradiation and the skin can be used. A convenient way of carrying this out is to place the radium in needles or tubes on the surface of a wax or rubber mould about 1 or 1.5 cm. in thickness.

*Dosage in Radium Therapy.* Clinicians have become so accustomed to the word "dose" in connection with the administration of medicine as meaning the quantity of a drug, that it may not be easy at first to realise exactly what the term dosage means when used in connection with radio-therapy. The quantity of radium that is used gives no indication whatever of the amount of energy received by the tissues, and merely to state that a certain amount of radium has been used is of no value unless many other factors are already known. The most important of these factors is the distance that the radium is from the tissue irradiated. In this respect the radiations from radium are similar to the radiations from a source of visible light, that is to say, if in both cases the source is a point source. The intensity of the rays in both cases diminishes inversely as the square of the distance from the point source. Although radium when used for therapy can never be a true point source of irradiation, nevertheless the importance of the law can be shown by quoting an actual example. If a tube of radium 15 mm. long with walls 0.5 mm. thick is buried in a mass of tissue, the cells in contact with that tube will be irradiated with an intensity at least a thousand times greater than a cell lying two inches away. It

*Guns used for implantation  
of Radium Seeds*



*Radium Seeds*

*Gold seed, with string for removal*



*Platinum seeds, 1000, 1000, 1000*



Fig. 833.—PHOTOGRAPH OF RADON SEEDS AND INTRODUCER.

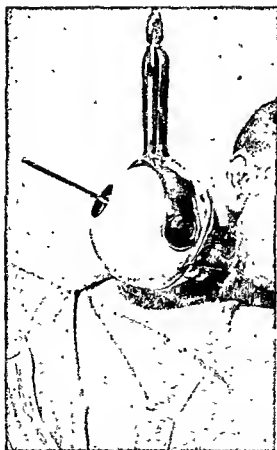


Fig. 834.—PHOTOGRAPH OF THE RADIIUM DOME USED AT THE WESTMINSTER ANNEX.



will be readily understood, therefore, how difficult it is to calculate the amount of irradiated energy that is received by the tumour. The total amount of energy received will depend not only on the intensity, but also upon the time during which the irradiated energy is falling on the tissues. The correct calculation of a "dose" of radium is made by multiplying the intensity of the irradiation in the tumour by the time that the tumour is being irradiated. As it is very difficult to calculate the exact intensity of irradiation in the tumour, or in different parts of the tumour, the "dose" is frequently expressed by multiplying the mass of radium used in the applicator and expressed in milligrammes by the number of hours during which the exposure is made. The result so obtained is spoken of as milligramme hours, but even this in itself is of little value if all the other factors are not known. For instance, if one milligramme of radium element is used for 100 hours, the biological effect will not be the same as if 10 milligrammes are used for 10 hours, or 100 milligrammes for one hour, although the amount of energy received at any one point would be the same in each case if measured by physical standards. There is yet another complication in calculating the dosage of a radium treatment, namely, the thickness of the wall of the radium container. This factor will make a great difference both to the actual amount of energy received by the tissues and the quality of the radiations received. For instance, if the walls of the container are more than 0.6 mm. platinum all the Beta rays will be absorbed and possibly some of the longer Gamma rays. In using a radium container near the tumour another complication is introduced, namely, the size and shape of the container. As mentioned above, the inverse square law is only true where the source of radiation is a point. It follows from all that has been said that at the present time it is very difficult, if not impossible, to calculate the exact dosage received by the tissues, but for convenience the dose is expressed as so many milligrammes of radium, or millicuries of radon used for a specified number of hours. The best method, therefore, of recording dosage is to state the mass of radium element or radon that is present in the container, the exact dimension of the container, and the thickness of its walls, the number of hours during which the radium is used, and, as far as possible, the exact position of the container or containers in relation to the tumour.

The calculation of the amount of radiated energy received from a source at a distance from the body is not quite so difficult, and in the case of X-ray therapy is calculated in the terms of erythema dose, or by the principles of unit designated "r."

*Units of Dosage.* Although the "erythema dose," or unit skin dose (U. S. D.), varies to a great extent according to how the radiation is given and other factors, yet it is sufficiently constant, according to Levitt in his book on Deep X-ray Therapy, to justify the following definition :

"That dose of X-rays which when applied, in one sitting lasting from half to one hour, to the normal abdominal skin of a healthy adult, with a port of entry of 64 sq. cm., will produce a bright red erythema without vesication after an interval of 10 to 21 days followed by pigmentation and desquamation."

The "r" unit is the international unit, and is the quantity of X-radiation which, when the secondary electrons are fully utilised and the wall effect of the chamber is avoided, produces in 1 cubic centimetre of atmospheric air at 0° C. and 76 cm. Hg. pressure such a degree of conductivity that one electrostatic unit of charge is measured at saturation current.

Having decided on the optimum amount of irradiated energy to be given to a tumour, it is then necessary to consider whether the energy shall be given continuously or at intervals. There is a certain amount of evidence, both experimental and clinical, that the effect of a given dose of radiation can be enhanced by interrupting the application of the radiation. The problem whether this "split dose" is better in all radio-therapy, or is better only in certain types of cancer, is one that has not been settled at the present time. Likewise, it is not possible to be dogmatic as to what is the optimum intensity and the optimum time of exposure, but there is some clinical evidence which suggests that in the case of undifferentiated neoplasms, for example, sarcoma, a large intensity for a short time is more effective than a small intensity for a long time. On the other hand, experience suggests that a markedly keratinised carcinoma requires a longer exposure, with a correspondingly small intensity.

To summarise, therefore, the problems involved in "dosage in radio-therapy," it may be said :

(1) That the ideal is to express the amount of irradiated energy actually received by the tumour.

(2) In the case of radium, this is so difficult to calculate that at present the more practical method must be adopted of stating :

(a) The amount of radium element in the applicator.

(b) The exact dimensions of the container.

- (c) The thickness and nature of the filter.
- (d) The position of the container or containers in relation to the tissues irradiated.
- (e) The duration of the application.

(3) The question of whether a large intensity used for a short time is better than a small intensity used for a longer period seems to depend on the nature of the tumour, but there must always be a threshold of time and of intensity. That is to say, however great the intensity, if the application only lasts for a fraction of a second little or no results will be seen, and, conversely, that with a very small intensity, no result would be obtained by an application kept in position for months.

(4) The problem of whether it is more beneficial to give a certain dose of radiation in one continuous period, or to split the dose into a number of applications, is not absolutely solved, but there is considerable experimental and clinical evidence in favour of split doses.

#### THE CLINICAL ASPECT OF RADIO-THERAPY IN GYNÆCOLOGY

The conditions in gynæcology for which radio-therapy is indicated may conveniently be divided into malignant and non-malignant. To the public, and even to a large number of medical men, the treatment of malignant conditions by radio-therapy is well known, but the great value of such treatment in non-malignant conditions is as yet not realised. It is said that the starting-point of radium therapy in connection with malignant disease was due to an accident. Soon after radium had been isolated, some experimenter carried about a tube of the precious element in his pocket, with the result that considerable radio-necrosis took place in his skin. This suggested the possibility of using it in the treatment of malignant neoplasms. First, this resulted in the dramatic disappearance of the growth locally, and people immediately considered that the cure of all malignant disease had been found. Later, when it was realised that metastases at a distance from the primary growth were not affected, the pendulum of opinion swung back the other way, and many people said that radio-therapy was of no value. It is only now that some true evaluation of radio-therapy is beginning to become evident. The present state of affairs can be summed up as follows :

That, in a great many types of cancer, radio-therapy is equal to or superior to excisional surgery. In the case of inoperable growth, it far

surpasses any treatment that has yet been tried. Although radium therapy is of some value in any malignant condition found in the female genital tract, yet some of these are better treated by excisional surgery, followed by X-rays, whilst in other types of disease, radium followed by X-rays is the right treatment.

#### CARCINOMA OF THE CERVIX

The commonest malignant condition in gynaecology is *carcinoma of the cervix*.

*Ætiology.* Statistics of a number of cases of carcinoma of the cervix show that occurrence of this condition varies in different countries. For instance, it appears that it is about twice as common in England as in Holland. It is of some interest to note that if the statistics of a number of sites of malignant disease are stated, it will be found that in different countries certain types are much more prevalent than in others, but the total number of deaths from malignant disease in all countries where statistics are properly kept is about the same. It has been suggested that the difference in the prevalence of carcinoma of the cervix in England and Holland is due to the trauma brought about by the too frequent use of forceps in the former country. There is no doubt that one of the outstanding ætiological factors in carcinoma of the cervix is the frequency with which it is found in multiparæ as compared to nulliparæ. It is extremely rare to find this condition in a patient who has never been pregnant, and it is difficult to escape from the conclusion that laceration of the cervix must be of importance as a predisposing factor. Nothing yet is definitely known as to why it should be so, but it has been suggested that the trauma at the time of childbirth increases the incidence of chronic infection. Further, it has been suggested that, following on this infection, the epithelium is destroyed, and that the columnar epithelium has a greater power of recovery and grows down from the lining of the cervical canal over the affected area. The squamous epithelium then tries to grow over the same area, and in doing so gets farther and farther from the surface, and the individual cells acquiring the property of living in an area which is poorly supplied by blood-vessels thus adapt themselves to a more anaerobic life. Following this the cells gradually pass into a state having all the characteristics of malignancy. It has also been suggested that the increased secretion due to the chronic cervicitis stimulates the growth of the squamous epithelium to such an extent that it becomes malignant.

There is no evidence that multiple pregnancies are more likely to result in carcinoma than a single pregnancy, but the evidence that it is more liable to occur in multiparæ than in nulliparæ is overwhelming.

Although it is true that malignant disease in general is associated with old age, yet in the case of carcinoma of the cervix no age can be said to exclude the possibility. The peak of incidence occurs between the ages of 45 and 60. There are a number of cases reported about the age of 18, but the disease is rare after 75.

*Pathology.* The normal histology of the cervix and the uterus is too well known to need a special description. The proportion of cases in which the malignant growth starts in the columnar epithelium lining the canal is about 5 per cent. These growths are endocervical, but there are other endocervical growths which originate in the squamous epithelium. The squamous-celled carcinomata generally consist of columns of polyhedral or rounded cells, and they rarely show any keratinisation. Clinically, there are three varieties :

- (1) The hypertrophic type, or cauliflower growth.
- (2) The infiltrating type, generally starting as an endocervical carcinoma, which produces an expanded and enlarged cervix.
- (3) The ulcerative type.

Of these three types, the hypertrophic seems to be the most radio-sensitive, and the prognosis appears to be better than in the other two types. It is of interest to speculate as to why this type of growth should give better results than the others. It is possible that there is a resistance (using the word in its broadest sense), which, preventing the cells from spreading up the cervix, allows them to multiply outwards, thus producing the cauliflower type of growth, whereas in the ulcerative type, the resistance being poor, the cells grow into the surrounding tissues forming early metastases. The disease spreads locally either along the vaginal walls or into the parametric tissues surrounding the cervix. In addition to this local spread, there is a lymphatic spread to the internal iliac glands. It is difficult to get statistics as to whether this lymphatic spread occurs at an early stage of the disease or late. For instance, Bonney states that in a series of 339 cases on whom he did Wertheim's hysterectomy, 42 per cent had carcinomatous deposits in the lymphatic glands, whilst other authorities state that only 34 per cent of patients who die from the disease show glandular involvement when examined at a post-mortem. It would appear from these two series that early cases may have glandular involvement, whilst in other

eases the disease may be in an advanced stage locally without having spread to the glands.

*Diagnosis.* Unfortunately the diagnosis of carcinoma of the cervix at the time that the majority of patients seek advice is by no means difficult. The first symptom that is likely to call attention to this type of growth is irregular uterine hæmorrhage. Whether before this occurs there is in the majority of cases a non-sanious discharge, it is difficult to determine, as it is not uncommon for women to ignore such a mild symptom. The importance of the symptom of irregular hæmorrhage, although common to many other diseases, cannot be too strongly stressed. It will take many years to educate the public on such matters, but medical men can and should, where possible, lay stress on the importance of this symptom. For example, when they are confronted with a case of menopausal hæmorrhage, they should take great care to explain to the patient that although in her case it is nothing serious, yet it is abnormal to have irregular hæmorrhages at the "change of life." If this precaution is not taken the patient may take upon herself to make a similar diagnosis in the case of other women suffering from the same symptom, the cause of which is very different. When the disease has advanced still further, the symptoms will depend on whether the bladder or the rectum is involved. If the former, cystitis and, later, hæmaturia will occur. In the last stage of the disease, the growth may constrict the ureters, giving rise to hydronephrosis, or may cause a fistula in the rectum or bladder. By this time the patient is suffering from a profound toxæmia, and the heart, poisoned by the toxins and weakened by the recurrent hæmorrhages, finally fails. The signs found in carcinoma of the cervix in the *very* early stages (fig. 835) are readily confused with a simple erosion (fig. 836). It is so important that treatment should be carried out at this stage of the disease that very great care must be taken to make a correct diagnosis. If the disease starts on the outside of the cervix, a small red area will be seen which bleeds readily at touch, and the probe pushed against the surface may show that the tissue is friable. When the disease starts in the inside of the cervix, the diagnosis cannot be so readily made, but any patient in whom there is irregular hæmorrhage which does not readily respond to treatment should be examined under an anæsthetic, and the interior of the uterus and cervix explored with a curette. At a later stage the local signs of disease are very obvious, either as a large cauliflower mass measuring even several inches across, or as a large ulcerated growth. Changes in

the general condition of the patient, such as anæmia and cachexia appear late in the disease and are generally associated with an ulceration and infection of the growth. Enlargement of the liver due to metastases is not common, and secondary deposits outside the pelvis are rare except in the very late stages.

*Treatment.* The treatment of carcinoma of the cervix has improved very greatly in the last thirty years. The working out of the technique for abdominal hysterectomy by Wertheim in 1900 marked a great advance, but the immediate mortality in the first hundred operations was exceedingly high. Since that time, other surgeons have practised and improved the technique, so that the immediate mortality has

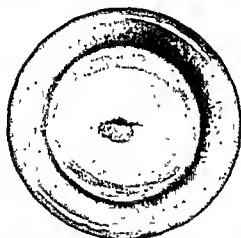


Fig. 835.—DRAWING OF A VERY EARLY CARCINOMA OF THE CERVIX AS SEEN THROUGH A SPECULUM.

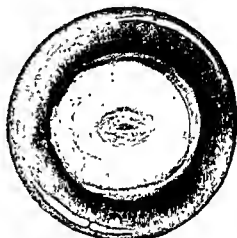


Fig. 836.—DRAWING OF A SIMPLE EROSION AS SEEN THROUGH A SPECULUM.

dropped to about 10 per cent, and in some hands even lower than this. The average immediate mortality due to the operation, taken from the figures of a number of different surgeons, is, according to the committee set up at Geneva, 17 per cent.

The next great advance in treatment was radio-therapy, and this first showed signs of being really successful in 1910. Since that date, further improvements have been brought about by combining radium and X-ray, so that at the present time in most countries excisional surgery for carcinoma of the cervix uteri has been entirely replaced by radio-therapy. There is still some controversy in this country as to which method should be used in those cases where the disease is still in its early stage, i.e. when it is still operable. At first sight it might appear an easy matter to decide by a statistical enquiry as to which of these two methods is the better. When, however, attempts have been

made, the number of factors involved are so great that, unless the enquiry is carried out having for its basis thousands of cases, the value of any conclusion is doubtful. Lane-Claypon, in 1927, reported on 6,661 cases operated on by vaginal or abdominal hysterectomy. Of these, the five years survival rate was 34.1 per cent. She also investigated 1,117 cases of carcinoma of the cervix which were technically operable, that is early cases, but had been treated by radio-therapy alone. The five years survival rate obtained by this method of treatment was 35.8 per cent. This method of comparing excisional surgery and radio-therapy has been criticised by some authorities on the ground that surgeons differ so much in their standards of what should be considered early, i.e. operable, and what should be considered inoperable. It has been suggested that the radiologist and gynaecologist practising radium therapy may judge a case as being inoperable which in point of fact would be found to be operable if the abdomen was opened. This, however, is extremely unlikely. Indeed, it is exceptional for a surgeon on opening the abdomen to say that a case is less advanced and more readily operable than he had anticipated, whereas it was not at all uncommon in the days before radio-therapy for a surgeon, having opened the abdomen, to express disappointment at finding things far more advanced than he had anticipated, and to be compelled to close the abdomen without doing anything. The radiologists working on carcinoma of the cervix have tried to define four stages to indicate the extent to which the disease has advanced. At the conference at Geneva, which reported in 1929, these stages were laid down for international use and have since been accepted by the majority of clinics and surgeons working on the subject. The National Radium Commission have made it one of the conditions for allowing their radium to be used, that statistics shall be kept on the basis of spread of the disease.

*Definition of International Stages 1-4.*

Stage 1. Growth strictly limited to cervix uteri.

Stage 2. Lesions spread into one or more fornices with or without infiltration of the parametrium to the uterus, the uterus still retaining some degree of mobility.

Stage 3. (a) Infiltration of parametrium on one or both sides extending to the wall of the pelvis, with fixation of the uterus.



- or (b) A more or less superficial infiltration of a large part of the vagina, with a mobile uterus.
- or (c) Isolated metastases in pelvic glands, with a relatively small primary growth.
- or (d) Isolated metastases in the lower part of the vagina.

Stage 4. (a) Massive infiltration of both parametria extending to walls of pelvis.

- or (b) Carcinoma involving bladder and rectum.
- or (c) Whole vagina infiltrated, or one vaginal wall infiltrated along its whole length, with fixation of primary growth.
- or (d) Remote metastases.

Stages 1 and 2 of the above correspond to those cases where the disease is still operable, although there are a number of cases technically Stage 2, which fall into the "borderline" condition under which beading some surgeons describe certain of their cases.

In discussing the method of treatment, whether it shall be excisional surgery or radio-therapy, the figures quoted above and the figures from other authorities suggest that the five years survival rate is slightly better in the case of patients treated by radio-therapy. In addition to statistics there are some other factors which can hardly help influencing a gynaecologist in favour of using radio-therapy. For instance, the immediate mortality due to radium treatment in the early stages of the disease, i.e. Stages 1 and 2, is negligible, and can only be due to the anaesthetic or other complications quite unconnected with the treatment. The suffering which results to the patient from radio-therapy, if a recognised technique such as is practised in Paris or Stockholm is used, is negligible compared to that following Wertheim's hysterectomy. In considering the choice of treatment, there are still other advantages in the use of radio-therapy. The fear of a big operation is entirely removed and patients, therefore, may be more willing to report to their medical adviser at an early stage of the disease. Again, the technique of Wertheim's hysterectomy requires a lifelong experience to become really efficient, whilst the technique of radium therapy can be readily acquired in the course of a few months. It must be admitted, however, that if good results are to be obtained from radio-therapy much more is needed than a mere knowledge of technique.

With all these points in favour of radium therapy, it may well be

asked what are the arguments against such treatment. The answer given by those surgeons who favour Wertheim's hysterectomy is one and one only, namely, that if the internal iliac glands are involved (and it is practically impossible from a clinical examination to prove or to disprove such glandular involvement) radium in the uterus and vagina will have little, if any, effect on deposits in the glands, whereas by Wertheim's hysterectomy, a certain percentage of patients will be saved by the removal of the glands in which carcinomatous deposits have been found. On the other hand, in a series of 214 cases published in 1926 in which Wertheim's hysterectomy had been performed, 34 died as a result of the operation, and among these fatalities 41.1 per cent had no carcinomatous deposits in the glands. It might be argued, therefore, that most, if not all, of these 14 other patients would have been saved by radio-therapy. Since the technique of X-ray therapy has been improved there is very little doubt that the five years survival rate of patients suffering from carcinoma of the cervix has risen considerably, and Professor Lacassagne, in a paper published in 1932, states that the improvement in the five years survival rate of patients in whom the disease had reached the third stage was due largely to irradiation by means of X-rays or by tele-therapy, with massive doses of radium.

To sum up, therefore, the arguments in favour of radio-therapy are :

- (1) That the immediate mortality is negligible.
- (2) That the statistics of survival rate in the early cases are in no way inferior to those of hysterectomy.
- (3) That the patients are not deterred from seeking early advice by the prospect of a severe operation.
- (4) A certain number (Heyman says 12 per cent) of patients who, because the disease is very far advanced, are beyond all surgical aid, are restored to health and survive more than five years.
- (5) As a palliative measure in very advanced cases there is no other treatment at present so beneficial.

*Technique.* In an earlier part of the chapter it was mentioned that radium can be used either interstitially, that is by putting radium or radon into the tissues by means of needles or seeds, or by putting radium into a natural cavity of the body, or by putting it at a distance from the surface. The most successful treatment in carcinoma of the

cervix appears to be by technique based on the cavitary method of applying radium.

The first efforts at radium therapy in cases of carcinoma of the cervix were made in 1905. In those days, however, the radium was contained in glass containers, allowing both Beta and Gamma rays to escape and affect the tissues. Dominici, in 1907, introduced containers made of silver, the walls of which were .05 mm. thick. This constituted a very big advance in the technique of radium therapy, and between 1909 and 1919 Cheron and Duval published statistics of 158 inoperable cases of carcinoma of the cervix treated by radium. The growth disappeared completely as far as could be ascertained clinically in 77 patients.

There are now, however, several well-recognised techniques, but it will be sufficient to describe in detail those that are better known.

#### THE STOCKHOLM TECHNIQUE

In Stockholm the treatment of carcinoma of the cervix was first started by Forssell in 1910. The treatment in those days consisted of small quantities of radium—for example, 20 milligrammes of the element—repeated on many occasions during a long time, such as three to six weeks. Later, when more radium was acquired by the Institution, the treatments were reduced and gradually the present technique was evolved.

Heyman, in the League of Nations Report, 1929, says that different applicators are used according to the extent of the growth, but always involving the same principle, namely, to cover as much of the growth as possible with radium and at the same time to stretch the vagina sideways so as to place the radium as near the lateral pelvic wall as possible. For the intra-uterine treatment an applicator is selected corresponding to the length of the uterine cavity so that the latter is completely filled from the fundus down. The radium is kept in place by means of wedges and gauze plugging (fig. 837).

The day before treatment the patient is admitted to the hospital and given a vaginal douche, and a tampon of iodoform gauze soaked in hydrogen peroxide is plugged into the vagina. As a rule no anæsthetic is given, but the patient receives an injection of morphia. The surface of the tumour is cleaned with benzine, the cervix dilated up and the radium put in position. Enucleation, cauterisation, or similar operations are never undertaken. In the case of pyometra, drainage by means of glass tubes is carried out for a few days before treatment.

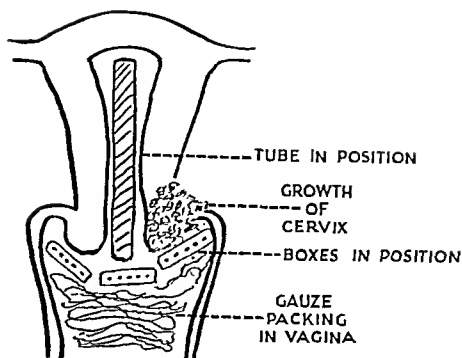


Fig. 837.—DIAGRAM SHOWING TUBE OF RADIUM IN THE CERVIX AND BODY OF THE UTERUS AND BOXES OF RADIUM KEPT IN POSITION BY MEANS OF GAUZE.

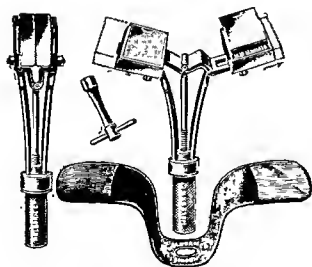


Fig. 838.—PHOTOGRAPH OF "BUTTERFLY" PESSARY USED TO KEEP BOXES IN POSITION IN THE VAGINA.

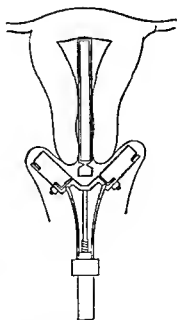


Fig. 839.—DIAGRAM OF "BUTTERFLY" PESSARY IN THE VAGINA.

The application is repeated on three occasions, the second being a week after the first, and the third three weeks after the second. Each application lasts for 20 to 22 hours. In addition to this, X-ray is now given as a routine, but this additional treatment has only been carried out in recent years.

As an example of the doses of radium, Heyman quotes the following:

*First Treatment.*

In the uterus (4 tubes), 40 mg. el.  $\times$  19 hours = 760 mg. el. hrs.

In the vagina (12 tubes), 78 mg. el.  $\times$  10 hours = 1480 mg. el. hrs.

*Second Treatment.*

In the uterus (1 tube), 43 mg. el.  $\times$  21 hours = 900 mg. el. hrs.

In the vagina (10 tubes), 71 mg. el.  $\times$  21 hours = 1490 mg. el. hrs.

*Third Treatment.*

In the uterus (1 tube), 39 mg. el.  $\times$  19 hours = 720 mg. el. hrs.

In the vagina (10 tubes), 80 mg. el.  $\times$  19 hours = 1520 mg. el. hrs.

The aggregate dose in this series will be :

In the uterus, 2380 mg. el. hrs.

In the vagina, 4500 mg. el. hrs.

The above "Stockholm Technique" is used at a number of centres in this country, but at the Mount Vernon Hospital we have modified this in order to get the radium treatment over more quickly and to start the additional X-ray treatment at the earliest possible date after the cervix has healed. The technique as modified is as follows :

The patient has a vaginal douche on admission to the hospital and for the first application a general anæsthetic is given. The advantage of a general anæsthetic on the first occasion is to make a more thorough examination of the pelvis. The amount of radium inserted is the same in nearly every case, namely, 50 mgrs. in the canal, with a filter corresponding to 1.5 mm. of platinum; and in the vagina 62.5 mgrs. of radium element in two boxes. These boxes have a filtration equivalent to 1.5 mm. platinum, and are attached to a pessary similar in type to the "Zwanche Pessary" (figs. 838 and 839), which years ago was used for prolapse. The two wings of the pessary are pushed well out into the fornices and kept in position by the screw in the handle of the stem. The whole pessary is kept in position by a metal "T" piece pressing into the stem of the pessary, and by gauze plugging

soaked in dilute solution of flavine (fig. 840). Each application lasts for 24 hours with an interval of one week between. Every patient is seen again one month after leaving hospital, on which date it is decided when the X-ray treatment should be carried out.

#### THE PARIS TECHNIQUE

The Paris technique is on an entirely different principle. The radium is kept in position for a week, but is taken out and replaced each day. The amount of radium used is very much less than in the case of the Stockholm technique. When the patient is admitted to hospital careful investigations are made to see what organisms are present in the cervix. At the same time the cervix is dilated up, but



Fig. 840.—PHOTOGRAPH SHOWING THE METAL "T" PIECE PRESSING INTO THE STEM OF THE PESSARY.

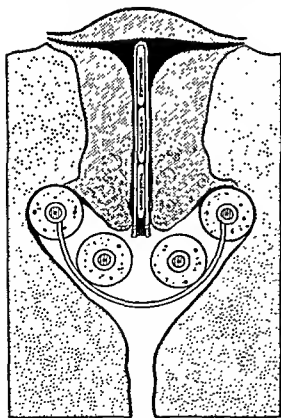


Fig. 841.—DIAGRAM SHOWING THE PARIS TECHNIQUE FOR RADIUM IN THE UTERUS AND IN THE VAGINA.

no radium is put in position until 24 hours have elapsed in order to see whether the dilatation of the cervix produces a temperature. If all goes well, after the preliminary dilatation three or four tubes of radium are placed in the vagina (the tubes having a filter equivalent to 1.5 mm. platinum) in a special colopostat (fig. 841). The amount of radium in the vagina equals 33.3 mgrs. At the end of three days, radium is placed in the uterus as well, and at the end of seven days all the radium is removed. The patients treated by this method have, in addition, either X-ray treatment to the whole pelvis or irradiation from a mass

of radium, for example, 4 grammes used at a distance. In Paris when X-rays are used the voltage is 180 to 200 KV., and the current 4 or 5 milliamperes—the skin distance being 60 to 80 centimetres. The filter varies according to the patient, but is as a rule between 1 and 2 millimetres of copper. It is usual to have eight fields of irradiation, each being 250 square centimetres. Each field eventually has 5 to 8 hours exposure split up into daily doses, thus the total number of days in which the X-ray treatment is carried out is about 25. In Paris the radium application is very often carried out after the X-ray treatment.

Another pioneer centre in Europe is the Brussels Radium Institute. In this centre the amount of radium used is very much less, but it is kept in position between one and two weeks, being taken out for cleaning every two days.

In Munich, at the University Gynaecological Clinic, where every case of carcinoma of the cervix since 1913 has been treated by radiotherapy alone, the treatment is very different to those already described. At this centre the first day is devoted to case taking, clinical examination, etc. The second day the pituitary gland is treated by means of X-rays. The third day X-ray treatment is used for the pelvis. On the fourth day no treatment is carried out. On the fifth day, if necessary, some of the growth may be removed by cautery. On the sixth day no treatment is carried out. On the seventh day radium treatment is given. Soon after this the patient leaves hospital, and eight weeks later returns and has a shorter treatment of X-rays combined with radium, and leaves the hospital at the end of a week. Usually two series of treatment are sufficient, but in certain cases it may be considered advisable to give further applications. The voltage used at this centre is 200 KV. and the current 2.5 milliamperes, the filter being 1 mm. copper.

In America the tendency in most of the centres is to use a very much larger quantity of radium or radon, but to apply the treatment for a shorter time. For instance, at the Memorial Hospital, New York, the patient is admitted and treated by X-rays. This is followed immediately by the application of 3000 millicurie hours in the body of the uterus and cervix. The duration of this application varies from twelve to twenty-four hours according to the amount of radon in the applicator. The walls of the filter are 0.5 mm. gold with a secondary filter of rubber. In addition, a "homb" consisting of a lead cup containing 1000 millicuries is applied to the cervix and fornices for three hours.

## 1655

Stage I.			Stage II.			Stage III.			Stage IV.		
Years.	No. of Patients Treated.	Irradiated.	Cured.	Irradiated.	Cured.	Irradiated.	Cured.	Irradiated.	Cured.	Total of Patients Cured.	Percentage of Cures.
1910	83	4	2	23	5	38	2	18		9	10
1920	89	12	3	36	8	38	3	3		14	15
1921	30	30	1	20	6	8	-	1		7	19
1922	03	7	4	25	9	27	4	4		17	20
1923	74	7	6	25	10	37	7	5		23	31
1924	68	3	3	19	9	36	12	10	1	25	35
1925	88	22	6	25	7	152	15	47		28	32
1926	87	4	4	27	15	41	12	15		31	35
Totals	588	52	29	200	69	263	55	73	1	154	20
Percentage of apparent Cures		55.7		34.5		20.9		1.3			



## MUNICH

STATISTICAL RESULTS OF RADIO-THERAPY IN CARCINOMA OF THE CERVIX  
YEARS OF TREATMENT, 1924-1926

	<i>No. of Patients treated.</i>	<i>No. of Patients surviving 5 Years.</i>	<i>Percentage.</i>
Group I . . .	99	49	49.4
Group II . . .	91	21	23.1
Group III . . .	129	23	17.9
Group IV . . .	85	4	4.9

## STOCKHOLM

## YEARS OF TREATMENT, 1914-1923

	<i>No. of Patients treated.</i>	<i>No. of Patients surviving 5 Years.</i>	<i>Percentage.</i>
Group I Operables . . .	188	76	40.4
Group II Borderline and Inoperables . . .	519	88	16.0

## MAYO CLINIC

## YEARS OF TREATMENT, 1915-1924

(Total cases treated, 1,001, but only 625 were confirmed by  
microscopic examination)

	<i>Percentage of Patients surviving 5 years.</i>
Group I . . . . .	75
Group II : borderline . . .	61.54
Group III : inoperables . . .	21.49
Group IV : "Modified" . . .	24.82

## MARIE CURIE HOSPITAL

Year.	Interval since irradi- ation.	No. of Cases treated.	STAGE							
			I.		II.		III.		IV.	
			L	D	L	D	L	D	L	D
1925 (3 mths)	8	14	-	-	2	1	2	6	0	3
1926	7	59	2	0	2	5	8	34	0	8
1927	6	57	1	0	5	4	9	19	0	19
1928	5	85	6	2	10	7	17	29	3	11
1929	4	109	4	0	13	4	20	49	2	17
1930	3	136	5	1	23	9	35	46	3	14
1931	2	126	7	2	16	3	35	42	3	18
1932	1	113	7	0	30	3	33	27	1	12
1933	-	107	5	0	33	0	47	5	8	9

## STATISTICS FROM THE MOUNT VERNON HOSPITAL

## INTERNATIONAL DEGREE

Year.	Interval since treatment.	1		2		3		4		Total.		Not Radiated.	
		Alive	Dead	Alive	Dead	Alive	Dead	Alive	Dead	Alive	Dead	Alive	Dead
1930	3 years	4	3	4	6	2	11	1	9	11	29		1
1931	2 years	4	-	7	7	10	14	1	10	22	31		-
1932	1 year	7	2	6	1	11	6	6	15	30	24		1
1933	-	3	0	5	0	19	3	6	5	31	9		

## RESULTS OF TECHNIQUE

The difficulty of comparing the results of radio-therapy and excisional surgery have already been referred to, but the five years' results of the former must convince the most conservative-minded medical man of the value of radio-therapy. It is almost impossible to judge which of the more well-known techniques is the most successful, but there is no doubt in my mind that when radio-therapy is combined with X-ray treatment the final results are superior to either of these methods of radiation used alone.

## CONCLUSIONS

Although there is considerable difference in the details of technique of treatment for carcinoma of the cervix in the various big centres, yet the total number of milligramme hours used works out very much the same. Judging by the published results it would appear that the growth is satisfactorily dealt with in the cervix, and that the failures are due to inefficient irradiation of the glands and deeper parts of the pelvis. It is still a matter of controversy as to why these deeper parts of the pelvis and glands should be more radio-resistant and difficult to deal with. At first sight it might be thought it was entirely a mechanical difficulty of getting sufficient energy through the skin and other structures to the affected portion of the pelvis. Although this may be a very important factor, in my opinion the question of radio-resistance due to the situation of the tumour and the character of the tumour bed may be equally important. Two interesting instances may be quoted in support of this idea. I was asked to treat two cases which had been diagnosed as carcinoma of the vagina, but on further examination proved to be very advanced cases of carcinoma of the rectum. In both these cases the growth in the vagina proved to be very radio-sensitive, whereas that in the rectum was much more radio-resistant. It is hoped that now that large masses of radium are available, and now that an X-ray machine is being designed which is capable of producing short waves comparable to those given out by radium, that this problem of radio-sensitivity may be solved.

## MALIGNANT CONDITIONS

*Carcinoma of the Corpus Uteri.* This condition occurs as a rule in women who are past the menopause, and although it is sometimes stated that it occurs as frequently in women who have had children as in those who have not, this, in my experience, is far from the truth. It may be true that in a small series of cases of carcinoma of the body, half the patients will have had children and half will be nulliparous, but since the majority of women in the world are multiparous, it is obvious that the disease occurs more frequently in nulliparous women. In my own experience it is comparatively rare to find carcinoma of the body in a patient who has had children.

The growth is a columnar-celled carcinoma (fig. 842) and is confined to the uterus for a greater time than in the case of carcinoma of the cervix.

When the disease is situated entirely in the fundus, it spreads to the lumbar glands, but when, however, it is in the lower part of the uterus the internal iliac and presacral glands may be involved.

*Diagnosis.* Uterine hæmorrhage is as a rule the earliest symptom complained of, and malignant disease of the body should always be suspected if there is the slightest uterine hæmorrhage in a patient past the menopause, and where there is no evidence of disease in the cervix. In very elderly patients a purulent discharge coming on suddenly should suggest a pyometra which has discharged itself, and this condition is not uncommonly associated with malignant disease of the body. The probable explanation of a pyometra being associated with carcinoma of the body is that the growth gives rise to intra-uterine hæmorrhage which is unable to escape owing to the narrowing or complete obliteration of the cervical canal. The blood thus pent up becomes infected and gives rise to pyometra.

In examining a patient it cannot be too strongly emphasised that she may look the picture of health, and frequently these patients are very stout. The only definite sign in a routine clinical examination is a slightly enlarged body of the uterus. Such a clinical examination must be followed within a very short time by a diagnostic curettage. A "wait and see" policy must never be adopted in the case of post-menopausal hæmorrhage. At the same time that a diagnostic curettage is carried out, in patients over 40 years of age 50 milligrammes of radium element should be placed in the body of the uterus and cervical canal for 48 hours whilst awaiting the result of the histological examination.

In a certain percentage of cases the report is negative, but if there is much thickening of the endometrium a granulosa tumour of the ovary must be suspected. Sometimes, however, there is very little endometrium, and in such cases the radium that has been placed in position for 48 hours will probably give rise to sufficient endarteritis of the vessels in the wall of the uterus to prevent further hæmorrhage.

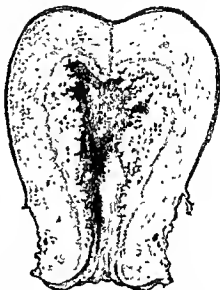


Fig. 842.—DRAWING OF A CARCINOMA OF THE BODY OF THE UTERUS.

Sometimes in such a case definite fibroids are present, and the existence of these tumours may lull the practitioner into a feeling of security and prevent him from carrying out a diagnostic enurettage. It cannot be too emphatically stated that carcinoma of the body is not infrequently present in a patient suffering from fibroids.

*Treatment.* There is no doubt that carcinoma of the body of the uterus is less radio-sensitive than carcinoma of the cervix, and since the operation of panhysterectomy has a comparatively low mortality this line of treatment should whenever possible be carried out. In my opinion, however, there is no doubt that every case of carcinoma of the body treated by hysterectomy should have X-ray treatment of the whole of the pelvis as soon as the abdominal wound has healed.

In patients who are inoperable either from the extent of the growth, or who are technically inoperable because of their general condition, radio-therapy alone must be undertaken. The number of these cases is hardly sufficient to justify dogmatism on the technique, but usually the same radium technique is carried out as that employed in the case of carcinoma of the cervix. At the Marie Curie Hospital attempts have been made to place extra radium in the cornua of the uterus. It is obvious that a greater intensity of radium should be used in the cavity of the uterus than is generally used in the Stockholm technique for carcinoma of the cervix. This radium therapy must in every case be followed up by X-ray treatment of the whole pelvis.

*Results of Radio-therapy.* The figures for the results of treatment by pure radio-therapy in carcinoma of the body of the uterus are small, but at the Radiumhemmet at Stockholm 36.6 per cent of 77 cases treated between 1915-1922 have lived for more than five years. In Munich, 66 per cent of the early cases survived five years, but only 7.9 per cent of the advanced cases were successfully treated.

*Chorion Epithelioma.* This is not a common malignant condition. It arises as a rule in patients who have had a hydatidiform mole, but it is also found after a simple abortion or even after the birth of a normal child. The histology shows that it is definitely a malignant disease of the syncytium and of Langhan's layer in the chorionic villus. There are many theories as to the aetiological factors concerned in the formation of a hydatidiform mole and of chorion epithelioma, and there is considerable evidence that it is associated with changes in the ovary, in which organs a lutein cyst is not infrequently found.

*Diagnosis.* In a typical history of a chorion epithelioma there is an abortion of a hydatidiform mole. This may be followed by normal menstruation for one or two months and then the patient complains of irregular hæmorrhage.

On physical examination in the early stages of the disease, nothing is found except a slightly enlarged uterus and possibly a cyst in one of the ovaries. In the later stage of the disease, the patient may complain of slight hæmoptysis. Indeed, this symptom is sometimes the first complained of, and is due to secondary deposits in the lungs. The possibility of the formation of chorion epithelioma following a hydatidiform mole is sufficiently great to justify a periodic examination of a patient for a year or eighteen months after evacuation of the mole. At these periodic examinations a specimen of urine should be tested by the Ascheim-Zondek test. In any suspected case of chorion epithelioma an Ascheim-Zondek test should be made of the urine and a diagnostic curettage of the uterus carried out. The disease tends to spread by the blood and secondary deposits are found in the lungs, liver, and not infrequently also in the vagina.

*Treatment.* At whatever stage the disease is discovered the first line of treatment should be X-ray therapy to the whole pelvis. This may be followed by panhysterectomy and bilateral salpingo-oophorectomy. The growth is extremely radio-sensitive, and in cases where there are secondary deposits in the vagina, radium therapy to the body of the uterus and vagina should follow the preliminary X-ray treatment.

I have seen a case where the vagina was full of metastases and two years later nothing abnormal could be detected in the uterus or vagina. In another case treated entirely by radio-therapy, but with only a small deposit in the vagina, the patient has lived for over five years.

*Sarcoma of the Uterus.* There is considerable difference of opinion as to the frequency with which this disease is found in women. It has been stated that 2 per cent of all fibroids removed show sarcomatous changes. On the other hand, sarcomata not infrequently arise without any previous history or sign of fibroids. The majority of such tumours are found in the body of the uterus though occasionally the growth may start in the cervix and fill the vagina as a pedunculated mass.

*Diagnosis.* When the neoplasm is found in the uterus it must be differentiated from a fibroid. This is not always possible before an

operation, but irregular bleeding associated with rapid growth of the tumour must give rise to the suspicion that a sarcoma is being dealt with. A positive diagnosis can only be obtained by microscopic section.

*Treatment.* The treatment of all sarcomata developing in the female genital tract is radiation followed, if possible, by excision. As a rule these masses are so large, and the risk of secondary deposits in other parts of the pelvis is so great, that it is advisable to irradiate the whole pelvis by means of X-rays. Such growths are very radio-sensitive, though not as radio-sensitive as chorion epithelioma. If the diagnosis is only made after excision, then the operation should be followed up as soon as the wound is healed by a full course of X-ray treatment.

*Carcinoma of the Vagina.* Primary carcinoma of the vagina is very much more rare than carcinoma in other parts of the genital tract. It is a squamous-celled carcinoma and is very radio-sensitive.

*Diagnosis.* The patient generally complains of bleeding and discharge, and on examination a friable mass is discovered. If the growth is large and high up in the fornices it is not always easy to differentiate it from a carcinoma starting in the cervix. If the section shows the growth to be a columnar-celled carcinoma the possibility of it being a secondary deposit from carcinoma of the corpus uteri must be borne in mind. Another differential diagnosis is a carcinoma of the rectum penetrating deeply into the vagina. I have seen two such cases, and it is of no little interest that although the growth was columnar-celled rectal carcinoma, yet that portion of the growth which extended into the vagina was found to be very radio-sensitive and the vagina was readily healed.

*Treatment.* In the majority of cases of carcinoma of the vagina there is no question of excision as the growth is too far advanced. Even if it were operable—for instance, high up in the fornices—the correct treatment is to use radium, although an excision of the growth following treatment can do no harm. The growth does not spread rapidly into the glands, and the prognosis is fair.

*Technique of Irradiation.* If the growth is small the best method of dealing with it is to bury needles under it, each needle having 1 mgr.

per centimetre of length and a filter of 0.8 mm. platinum. Another method is to make a wax stent of the vagina and to place the needles on the surface of the stent so that they come as close as possible to the growth. The needles should be placed a centimetre apart and should be left *in situ* for seven days. The irradiation by radium should be followed up by X-ray therapy to the whole pelvis.

*Carcinoma of the Vulva* (fig. 843). This condition is usually found in elderly women, and it is always a matter of surprise that the growth is so often allowed to advance until it is quite a big mass before the patient seeks advice. There are two probable explanations—first, the fear of being told that the condition is malignant and, secondly, that the patient being old may feel that she cannot be bothered to undergo treatment.

In many cases a pre-carcinomatous condition is present, namely, leucoplakia.

This disease starts as a chronic inflammatory condition of the vulva and passes through two or more stages. In the first stage, there is great irritation associated with thickening of the epidermis. In the second stage, cracks appear in the thickened epidermis which reaching



Fig. 843.—DRAWING OF A CARCINOMA OF THE VULVA.

the papillæ cause great pain and slight bleeding. It is in these cracks that carcinoma of the vulva is likely to develop. Should the cracks heal, then the last stage is an atrophied condition in which the skin looks a translucent white, from which the condition derives its name.

*Diagnosis.* The diagnosis of a carcinoma of the vulva even in the early stages is not difficult, but should be confirmed by section at the time of treatment.



*Treatment.* It is not possible to be dogmatic as to the best way of treating carcinoma of the vulva, but in the early stages excision of the primary growth, together with the superficial glands, and X-ray therapy before or after the excision, is probably the safest course to adopt in our present knowledge of the disease. If, as so often happens, the primary growth has extended too far for excision, then the problem is how best to treat by radio-therapy. In such cases the primary growth should be treated by means of radium followed by X-ray therapy with or without excision of the glands. The radium can be applied by means of needles, but since the growth is very radio-resistant it requires a considerable duration of exposure which is liable to injure the normal tissue as well. In addition to this, the growth is often grossly infected and the results of needle treatment are not without risk.

Another method is to make a wax stent and apply the radium for seven or eight hours a day until such time as a radium reaction is seen. This may take three weeks, as it is unwise to use a very large intensity.

The difficulty of treating the glands is the same as is experienced in the treatment of lymphatics elsewhere in the body. It is one of the most urgent problems that now confront radio-therapists.

*Malignant Tumours of the Ovary.* The malignant condition may be primarily in the ovary or may be a secondary deposit from a growth elsewhere in the body. It is an interesting fact that sometimes the ovaries appear to be the only place in which secondary deposits have occurred. This is particularly true of carcinoma of the alimentary canal. The secondary deposits from such primary sites as the stomach and intestines often take on a characteristic histological appearance and are known as Krugenberg's tumours.

*Diagnosis.* When the growth is really small it is only by accident that the tumour is diagnosed, and it is for this reason that the prognosis of carcinoma of the ovary is so hopeless. The presence of an ovarian tumour is not, as a rule, difficult to discover, but it is rarely possible to be quite certain of the malignant condition without an exploratory laparotomy. Among the signs suggesting malignancy are rapid growth, ascites, œdema of the legs, and finally hard masses felt on vaginal examination, or per rectum. Wherever possible a laparotomy should be performed, as occasionally a suspicious case turns out to be a papilliferous cyst, removal of which will cure the patient.

*Treatment.* There is some doubt as to whether in an advanced case of carcinoma of the ovary there is any value in removing the majority of the tumour. Indeed, such cases, as one might expect, seem to grow rather more rapidly if some of the tumour has been removed than previously. On the other hand, the prognosis is so poor that the psychological effect when the patient finds that the mass in the abdomen seems to have disappeared may justify removing as much of the growth as possible. The treatment after excision of as much of the growth as possible, or after a definite diagnosis has been made, is X-ray therapy. I have seen patients with large malignant masses made more comfortable for a number of years and the growth diminished in size, but the prognosis is very poor.

#### BENIGN CONDITIONS

So far this chapter has dealt entirely with the radiological treatment of malignant disease of the female genital tract, but its value in non-malignant conditions is very great. The chief benign conditions in which radio-therapy has been employed are as follows :

- (1) Menorrhagia or irregular hæmorrhage associated with the menopause.
- (2) Certain cases of menorrhagia in young patients.
- (3) Menorrhagia or irregular hæmorrhage caused by fibroids.
- (4) Tuberculous disease of the cervix or vagina.
- (5) Leucoplakia of the vulva.

*Abnormal Menopause.* It is in cases of abnormal menopause that radio-therapy perhaps finds its most satisfactory results. The normal termination of menstrual life, that is to say the menopause, is so often associated with menorrhagia (flooding) and irregular hæmorrhage, that the majority of women consider that such symptoms are "natural," and must be patiently borne. It is the frequency of these symptoms between the ages of 40 and 50 that prevents women very often from seeking medical advice, and thus malignant disease of some part of the genital tract is often ignored until it is far advanced. If once the public could be educated to realise that the symptoms of irregular hæmorrhage and menorrhagia should be reported to a medical man and not accepted as a natural part of the menopause, then thousands of lives would be saved each year. In the past the medical profession may have been responsible for confirming this erroneous

idea. It was not uncommon in the old days for a patient to be given a bottle of medicine and told "it is only the change of life," which comforting words were repeated to neighbours suffering from the same symptoms though not always due to the same cause. There are few medical men nowadays who would be guilty of treating such symptoms without a thorough pelvic examination, but there are still some who do not realise the importance of explaining to the patient that although it is only "the change of life" such symptoms are abnormal and the patient did well to report them at once.

Every medical student is taught to-day that the normal menopause consists in an abrupt cessation of menstruation, or an increased cycle, or in a diminished loss, the menstrual cycle remaining the same. Any other termination, such as menorrhagia or irregular hæmorrhage, demands a careful pelvic examination, and very frequently the diagnosis of abnormal menopause cannot be confirmed without a diagnostic curettage. If, on a general physical examination in a patient who is between 40 and 50, nothing abnormal can be found to account for menorrhagia, it is safe to treat the patient by means of drugs for the next one or two menstrual periods. In the case of a patient complaining of irregular hæmorrhage, however, no time should be lost in exploring the uterus and cervical canal.

There are two methods by which menorrhagia and irregular hæmorrhage at the menopause can be treated, namely, radium or X-rays. The problem then arises as to which of these is the better. In the case of irregular hæmorrhage there can be no doubt whatever that radium should be used, since it is impossible to confirm the diagnosis of abnormal menopause in such a case without doing a diagnostic curettage, and if the cervix is dilated for diagnostic curettage then a tube of radium can readily be placed in the body and cervix. In the case, however, where the symptom is purely one of menorrhagia it is not so essential to do a diagnostic curettage and therefore X-rays may be used. The advantage of X-ray treatment is that it does not require an anæsthetic and the patient is not obliged to go to bed. On the other hand, it is sometimes a great disadvantage not to be able to examine the patient under an anæsthetic, and there is a risk that an inflammatory condition of the pelvis may be overlooked which will be very seriously affected by the X-ray therapy. When, therefore, there is any doubt as to the diagnosis, or when the patient is stout and therefore difficult to examine without an anæsthetic, radium should be used. X-rays should be reserved for those patients in whom the diagnosis is certain and the pelvic examination easy.

*Technique of Radio-therapy.* There has been considerable discussion as to the exact details of technique when applying radium to the uterus for an artificial menopause, but in my experience the following method has proved extremely satisfactory :

The patient is given a general anæsthetic and placed in a lithotomy position. The vagina and vulva are cleaned, and a non-metallic antiseptic such as ether applied to the vagina and cervix. After a thorough manual examination of the pelvic viscera, the cervix is pulled down by means of a vulsellum and dilators are passed into the canal. As a rule it is not necessary to dilate further than a 9/12 Hegar's dilator. The body of the uterus and the cervical canal are then explored and gently curetted. Following this 50 mgrs. of radium element enclosed in a tube, the walls of which are equivalent to 1 or 1.5 mm. platinum, are inserted into the cavity of the uterus and cervical canal. The tube should be covered with rubber, and a convenient length is 2½ inches. The vagina must then be carefully plugged, to prevent the possibility of the tube being dislodged and falling back into the vagina. Gauze packing soaked in flavine 1 in 4000 is generally used for the vaginal plugging, and it is carefully inserted high up into the posterior fornix before the rest of the vagina is filled. Should the radium slip even a short way into the vagina, the rectum, which is very radio-sensitive, will be kept well away from the tube of radium. The radium is kept in position for 48 hours and then removed without an anæsthetic. The patient stays in bed for three or four days, after which the vagina should be douched once or twice a week with normal saline for three or four weeks. The patient must be warned that one or even two more periods may occur, and that there may be a slight vaginal discharge due to radium reaction which will last for two or three weeks. She must also be made to realise that menopausal symptoms such as flushings are just as frequent when the menopause is brought on artificially as when it comes naturally in the course of time.

*Treatment by X-rays.* There are two methods employed in creating an artificial menopause by means of X-rays, namely, a single large dose or repeated smaller doses. My colleague, Dr. N. S. Finzi, has kindly sent me the following note on the treatment he uses :

"The method I have often used in the treatment of menorrhagia is to treat the patient through three ports of entry, each about 100 to 150 square cm., two anterior and one posterior, on successive days, using X-rays at a kilovoltage of 150 undulating

current and a filter in most cases of 5 mm. of aluminium: the focus-skin distance is 24 cm. I give 85 to 90 per cent of a full erythema dose in the first series. The second series is started four weeks from the date of commencement of the first, and a slightly smaller dose is given. The third series is started eight weeks from the first. In stout patients or those with large fibroids I use four or five ports of entry. The rays in all cases are directed very obliquely with considerable compression so as to attempt to get both ovaries into each field.

The advantages of the method are that the patient need not be in a hospital or nursing home, but can attend as an out-patient; there is very little or no disturbance to the general health, and post-radiation nausea and vomiting are exceedingly rare, in contrast to the single-dose method, where they are not uncommon. The disadvantages are that it is a little slow, and in a few very stout patients I have seen permanent skin changes, but these are very unusual and can, I think, be completely avoided.

I use the single-dose method when the patient comes from a great distance, or when she prefers it."

#### RESULTS OF TREATMENT OF ABNORMAL MENOPAUSE BY MEANS OF RADIO-THERAPY

The statistics published by various clinics all show equally satisfactory results. For instance, in a series of 122 cases from St. Bartholomew's Hospital 96.3 per cent had permanent amenorrhœa. At Mount Vernon Hospital in a series of 85 patients, 98 per cent were complete successes. At Mount Vernon Hospital statistics have been kept on details other than merely the production of a permanent amenorrhœa. For instance, such annoying symptoms as flushings, nocturnal sweatings, and sex instinct have been noted. As a result of these statistics it is possible to state that when the patient is over forty the risk of having flushings, nocturnal sweating, or loss of sex instinct is no greater than might be found in an equal number of patients in whom the menopause has occurred as a result of nature.

*The Treatment of Fibroids by Radio-therapy.* The indication for radio-therapy in the case of fibroids may be dogmatically stated as follows: Any patient who is over 40 years of age in whom the fibroid is not larger than a full-time foetal head and in whom there is no evidence of pelvic inflammation. It may be asked why one sets a limit to the

size of the fibroid. The answer is twofold. First, that very large fibroids are more likely to undergo degeneration and, secondly, that with a very large fibroid it is sometimes difficult to exclude inflamed tubes, etc. In addition to the above indications, there are, of course, those patients in whom operation is contra-indicated although the fibroid is very large. In such patients one must risk the possibility of degeneration, etc.

The action of the radio-therapy is precisely the same as in the case of menorrhagia, or irregular hæmorrhage caused by an abnormal menopause, that is to say the ovaries are affected and an artificial menopause brought about. The result will depend very largely on the proportion of fibrous tissue and muscular cells in the tumour. If muscle predominates the fibroids will become so small that it may be difficult to recognise their existence by an ordinary bimanual palpation. If, on the other hand, there is a large amount of fibrous tissue very little change will be noted in the size of the tumours. In one of my cases a uterus which originally reached to within half an inch of the umbilicus had receded to the size of a twelve weeks' pregnancy three and a half months later.

The technique for radium treatment of fibroids is similar to that employed when creating an artificial menopause, but where the uterus is very large it is preferable to leave the radium in position for 72 hours instead of 48.

*Production of Artificial Menopause in Young Patients suffering from Menorrhagia.* There has been a considerable amount of discussion as to the advisability of treating young patients who are suffering from intractable menorrhagia by means of radium or X-rays. It is of very considerable interest to note that it sometimes requires very high doses of radium or X-rays to produce a permanent amenorrhœa in such patients. For instance, in one patient, aged 21, who was recently treated by means of X-rays for chorion epithelioma, menstruation recurred and has been quite normal for the last six months.

A considerable number of these younger patients have been treated at the Royal Free Hospital with apparently satisfactory results, but the following arguments have as a rule prevented me from using this type of treatment in patients under the age of forty :

- (1) If a permanent amenorrhœa should be obtained with the accompanying artificial menopause, the symptoms—flushing, etc.—seem to be rather more severe than is the case when an artificial menopause is produced in patients over forty.

- (2) It does not seem justifiable to bring about "the change of life" in young women.
- (3) If a dose is used so that the periods return (and in very young patients, as already mentioned, the periods do as a rule return) then the question arises as to whether the ova which are produced by such irradiated ovaries will be normal.

*Conclusions.* From the above it may be concluded that :

- (1) The production of an artificial menopause by radio-therapy is indicated in patients over the age of 40 who are suffering from menorrhagia or irregular hæmorrhage.
- (2) The treatment by radio-therapy of menorrhagia or irregular hæmorrhage due to fibroids is indicated in patients aged 40 or over when the fibroid is not larger than a foetal bead and there is no inflammatory condition in the pelvis.
- (3) That the optimum dose seems to be 50 mgrs. of radium element applied in the uterus for 48 hours, although in the case of large fibroids a somewhat longer application may be given.

#### TREATMENT OF OTHER NON-MALIGNANT CONDITIONS

The other non-malignant conditions in which radio-therapy may be employed are tubercular disease of the cervix and vagina and leucoplakia. The former of these is extremely rare, and a definite diagnosis can only be made by examining a histological section. The condition is so seldom met with that it is impossible to be dogmatic as to the best technique. Tubercular disease in the glands or elsewhere is very amenable to radio-therapy, and it is as well to start with a dose of 50 mgrs. of radium element, with 1 mm. platinum, kept at a distance from the vaginal walls by means of a stent and left in for not longer than 24 hours. Further treatment must depend on the result of this initial dose, but at least six weeks should elapse before deciding to give any further radio-therapy.

In the case of leucoplakia, treatment can be carried out either by X-rays or radium. On the whole, the treatment is more readily and more efficiently carried out by X-rays and such cases, if the affected area is too large for excision, should be handed over to the radiologist.

AN OUTLINE OF THE TREATMENT OF MALIGNANT DISEASE, IN  
GYNÆCOLOGICAL CASES, BY HIGH VOLTAGE X-RAYS, AS CARRIED  
OUT IN THE RADIOLOGICAL DEPARTMENT OF THE MOUNT  
VERNON HOSPITAL

by  
G. C. FAIRCHILD

THE description given below is of the routine treatment at present in use at this Hospital, and does not include special methods used in certain cases for research purposes, and which are at present *sub judice*.

It is convenient to consider the matter under the following headings:

- (1) The patient.
  - (a) General condition.
  - (b) Local condition of skin.
  - (c) Position, extent, and pathology of lesion.
  - (d) Primary objective of treatment.
- (2) The apparatus used to produce the requisite radiation, and that used for the measurement of its quality and intensity. The biological unit of quantity, the "U.S.D." (unit skin dose).
- (3) The methods employed in applying the radiation.
  - (a) Physical factors.
  - (b) Field selection.
  - (c) Dosage.
  - (d) Technique. Protection of patient from stray primary and secondary radiation.
- (4) Precautions taken during irradiation, and the immediate and after-effects of irradiation.

THE PATIENT

*General Condition.* The general condition of the patient should be comparatively good, although poor condition does not of necessity



contra-indicate treatment, but it must be remembered that a course of X-ray treatment involving the heavy dosage required in these cases should not be lightly undertaken without weighing up the pros and cons as to whether the patient will be able to tolerate it.

Patients with marked asthenia, and where the natural defensive mechanism has apparently broken down, and where distant metastasis has taken place, are probably better left untreated, except as a purely palliative measure for relief of pain, etc. All cases should have pelvis (spine) and chest skiagraphed before treatment, in order to discover the presence or absence of metastases in these regions.

A preliminary blood count should be done in all cases, partly in order to see if it is safe to administer the requisite dose, and partly to have a record of the blood count as it was before irradiation.

Should the blood count be unsatisfactory, especially as regards a low lymphocyte count, it does not contra-indicate treatment, but this should be undertaken with caution and frequent blood examinations made, or if the patient's condition will permit, preliminary treatment may be undertaken to try and improve the blood condition before irradiation is started. The patient should be examined with a view to ascertaining the presence of any concurrent disease, such as would affect her sensitivity to radiation, e.g. Graves' disease, diabetes, eczema, etc. The age of the patient is also a factor for consideration, as the older the patient is, the larger the erythema dose will be, and vice versa.

*Local Condition of the Skin Areas to be Irradiated.* First and of great importance is the question: Has the skin of these areas received any previous irradiation, and if so, when, how much, how given, and at what voltage and filtration? This question may not affect more than 1 per cent of cases, but in that 1 per cent it may be the means of saving the patient from serious damage, especially in those cases where large doses of soft radiation have been administered. The presence of the results of trauma, such as burns, cicatrization resulting from surgery, vaccination, etc., or pathological conditions such as ventral hernia, the presence of a colonic or vesical fistula, etc., may also prove limiting factors as to the amount of radiation the skin will tolerate.

Further, although it does not strictly come under this heading, should be mentioned the varying degree of radio-sensitivity exhibited by different individuals. In the majority of cases this does not exceed a matter of plus or minus 10 per cent, but there are occasionally seen those cases where there is a definite idiosyncrasy, and in these even a

60 per cent Unit Skin Dose has produced serious effects. Any cases showing a marked erythema towards the end of their course of treatment should be proceeded with with the greatest care.

In this connection may also be mentioned the very rare case, where early in the dose a temporary erythema may appear, which completely fades within 48 hours of its appearance. This condition may be disregarded.

#### THE POSITION, EXTENT, AND PATHOLOGY OF THE PRIMARY LESION

*The Position of the Primary Lesion.* The usual sites are as under, given in their order of frequency :

- (1) Uterine cervix.
- (2) Uterine fundus.
- (3) Vulva.
- (4) Ovaries.
- (5) Chorion (epithelioma).
- (6) Fallopian tubes.

*Extent and Environment of Primary Lesion.* The actual extent of the primary lesion does not of necessity influence the technique to be employed, provided it is not so extensive as to render the case hopeless, e.g., one where there is extensive involvement of rectum and bladder. The reason for this attitude is that, however early any individual case comes under observation, one is never safe in assuming that there is no malignant tissue present outside the primary lesion, or, in other words, that local metastasis has not already taken place. This being so, it is of paramount importance that a wide area outside the primary lesion should be regarded as constituting a danger zone, and should therefore be included in the area of irradiation, especially those lymphatics draining the area involved.

In all gynaecological cases, therefore, an area is included in the field of irradiation reaching from a line joining the crests of the ilia above, to a point well below the vulva below, regardless of the extent of the primary lesion. There is also a physical reason for the use of such an extensive area of tissue, this being that there is an appreciable increase in the quantity of secondary radiation set up at a depth.

The environment of the lesion is a matter of importance and careful examination should be made in order to exclude the possibility of any septic focus, such as a pyosalpinx, breaking-down glands, or fibroma, and should any such focus be present, it should be dealt with before

irradiation is commenced, as otherwise very serious complications may ensue.

*Pathology.* The main varieties of growth met with in these positions are :

- (1) Carcinoma.
- (2) Sarcoma.
- (3) Chorion epithelioma.
- (4) Endothelioma.

It is the present practice in this hospital to treat all malignant growths with a maximum dose, regardless of the varying degrees of sensitivity to radiation which they may exhibit.

It is true that, in a large percentage of cases, sarcomata and endotheliomata are more radio-sensitive than carcinomata, but on the other hand there are cases where the reverse condition holds. It is almost impossible to foretell with any degree of certainty how any individual growth will respond to irradiation.

*The Immediate Objective of Treatment.* The immediate objective is to administer to a predetermined area of tissue such a quantity of radiation as will prove lethal to every malignant cell within that area, and which at the same time will permit all non-malignant tissues within that area to resume their normal functions within a certain interval of time.

THE APPARATUS USED TO PRODUCE THE REQUISITE RADIATION, AND THAT USED TO MEASURE ITS QUALITY AND INTENSITY, TOGETHER WITH SOME PHYSICAL CONSIDERATIONS. THE BIOLOGICAL UNIT OF MEASUREMENT.

A closed core transformer fed from A.C. mains is used to produce a H.T. current at 200 K.V. constant potential, by means of valve rectification and condenser circuit. This H.T. current is used to excite the X-ray tube. The X-ray tube used in routine treatment is a hot-cathode tube of the sealed-off glass variety. Dissipation of heat generated in the anode of the tube is procured by feeding water to the anode under pressure, a water-pump and radiator being placed in the H.T. circuit for this purpose. The tube is capable of passing a current of 15 MA. at 200 K.V. continuously. The H.T. current is led through an elaborate anti-corona system, in order to avoid brush

discharge and consequent air contamination. A heavy type treatment tube stand carries the X-ray tube, which is capable of movement in all directions.

For the physical measurement of intensity of the X-ray beam, the Mekapion Iontogrammeter is used.

The standard unit of radiation used is the International "r" unit, the definition of which will be found on page 1641 of this book. For the measurement of surface and depth intensity, the Mekapion instrument is used in conjunction with a wax phantom, the wax being made of the same specific gravity as the body tissues, and capable of alteration in volume and shape. Depth and surface intensity are also measured directly by the Mekapion, the ionisation chamber being inserted into suitable body cavities, and placed on the body surface.

The quality or wave-length of the X-rays produced under varying conditions of filtration and voltage is investigated directly by means of the X-ray spectrograph, and indirectly by means of the "half value layer" method, also by direct measurement of voltage by means of the sphere gap, the wave-length of the X-rays being inversely proportional to the voltage used in their production. The biological standard of measurement of quantity of any given radiation is the "Unit Skin Dose."

The "Unit Skin Dose" represents that quantity of radiation which, when applied to the normal skin of the abdomen of a healthy adult, to an area measuring  $8 \times 8$  cm., and when given in one application lasting under one hour, will be followed in from ten days to three weeks by a bright red erythema, without vesication, but followed by desquamation and pigmentation.

#### METHODS EMPLOYED IN APPLYING THE RADIATION

*Physical Factors.* In routine treatment of these cases, the physical factors are as follows :

200 K.V. constant potential.

10-15 MA tube current.

1.5 mm. Cu. and wood filtration.

40-76 Cm. focus skin distance.

*Field Selection relative to the Site of Lesion and Habitus of Patient.*  
As it is impossible to treat a lesion at a depth of several centimetres with an adequate dose of X-rays through one area of skin, it becomes

necessary to select several ports of entry, through which the rays will converge in the desired area.

To accomplish this it becomes necessary to make a sketch of the cross-section of the patient's body at the level of the lesion. To do this the patient is placed supine on the treatment couch, and by means of calipers the antero-posterior diameter of the pelvis is measured at the level of the symphysis pubis in front and of the coccyx behind. The lateral diameter is then measured at the same level. Two lines are then drawn on a large sheet of paper, at right angles to one another. The antero-posterior diameter is then marked on the vertical line, its mid-point coinciding with the intersection of the horizontal and vertical lines, and the lateral diameter is then marked on the horizontal line in the same manner. A strip of malleable metal is now carefully moulded to the patient's body around the pelvis at the same level and then transferred to the paper, the antero-posterior and lateral points being made to coincide with those already marked, and the outline drawn in. We now have an outline of the cross-section of the patient's pelvis, with all its various contours.

The lesion is next sketched in, as nearly as possible in the position it occupies in the patient, this having been previously estimated by clinical examination and where possible by skiagrams taken with opaque indicators in position. The above procedure may be carried out on the illuminated glass of the Holfelder Field Selector instead of on paper. In either case the Holfelder tinted gelatin strips may be used to estimate the correct position of fields and concentration of rays.

In using the paper sketch, it is now necessary to make measurements on the surface line of the varying surface lesion distances at different points. From this data are calculated the number of ports of entry, the angles at which the central ray for individual ports will have to be projected, and the total surface radiation that will be necessary to produce a lethal dose at the depth of the lesion. The various fields selected are now marked in on the patient's skin with skin ink.

The above information having now been obtained, the patient is ready for treatment.

*Dosage.* There are three main methods of dosage :

- (1) The intensive single dose.
- (2) The intensive split dose.
- (3) The protracted fractional dose.
- (4) The saturation method.

The procedure that has given the best results up to date in this hospital is a combination of the intensive split dose combined with the saturation dose methods. This is carried out as follows :

Daily doses (Sunday excepted) are given till the required depth dose has been attained, which usually occupies a period of from 14 to 21 days. Following this, at selected intervals, repeat doses are given equal to the fall off in value with time of the original depth dose, which is thus kept up to its original value. In other words, supposing 100 per cent depth dose has been given, this depth dose is kept at a 100 per cent value for a predetermined time.

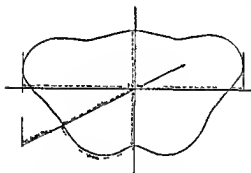


Fig. 844.—DIAGRAM OF THE SURFACE OUTLINE OF THE CROSS-SECTION OF A PELVIS, AT THE LEVEL OF THE OS PUBIS, OBTAINED BY THE METHOD DESCRIBED. IN THIS CASE THE LESION IS PLACED CENTRALLY IN THE PELVIS.

THE PROJECTION OF THE CENTRAL LAY OF ONE PORT OF ENTRY ONLY HAS BEEN INCLUDED (THAT OF A POSTERIOR OBLIQUE) IN ORDER NOT TO COMPLICATE THE DIAGRAM.

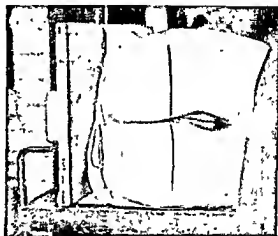


Fig. 845.—PHOTOGRAPH OF A PATIENT IN POSITION FOR THE TREATMENT OF A PERINEAL FIELD AS DESCRIBED.

*Technique.* All these cases are treated in the horizontal position, either prone or supine, except in the case of a perineal field, when the procedure is as follows : The patient is placed across the treatment couch, the head resting on an extension, and the back supported and fixed by a special arrangement, with the transverse diameter of the body in the vertical position. The buttocks are placed level with the lateral side of the couch, the thighs being well flexed with the legs at right angles to them. Large pads are then placed between the legs, to keep them parallel, and thus assist in maintaining the transverse diameter of the pelvis in the vertical position. The buttocks are then separated, revealing the anus and vagina, and a line is drawn vertically

through the anus over the buttocks, for purposes of centring. An examination is now made per rectum and per vaginam to locate exactly the site of the lesion with reference to the above line and to the tip of the coccyx. The tube is now centred both in relation to the line through the anus and the plane of the coccyx and inclined slightly downwards.

It is found that this method of treating a perineal field is much safer from H.T. dangers, and is more comfortable for the patient than the use of the lithotomy position.



Fig. 846.—THE SAME PATIENT AS IN FIG. 845, BUT COMPLETELY COVERED WITH LEAD RUBBER AND WITH TUBE CENTRED READY FOR TREATMENT.

In cases of large and obese patients, when treating through the abdomen, it is advantageous to have the patient in the Trendelenburg position, as this helps to keep the movable abdominal viscera away from the field of irradiation, and also permits of greater compression.

In all cases, wherever possible, compression is used by means of rigid applicators and retaining devices, in order to lessen the surface-lesion distance, which in some cases may amount to as much as

10 cm. or more. Additional compression is secured, when necessary, by means of adjustable hands passing over the abdomen.

In treating gynaecological cases it is well to have the bladder and lower bowel empty before each treatment.

All irregularities in contour, such as the triangular space between buttocks and thighs posteriorly, and that between thighs and groins anteriorly, are carefully filled up with powder bags, containing a mixture of Fuller's earth and sod. bicarb. made up to a specific gravity of 1, in order to avoid overdosing of such parts as the vulva, etc.

Protection of the patient from stray primary and secondary radiation is provided by covering all parts, with the exception of the area being irradiated, with lead or lead-rubber of equal lead equivalent. The weight of the protective material is carried by specially constructed cradles.

In treating small or average-sized patients, it is usual to select five fields :

- (1) Anterior vertical ( $20 \times 15$ — $20 \times 20$  cm.).
- (2) Posterior vertical ( $20 \times 15$ — $20 \times 20$  cm.).
- (3) Right lateral vertical ( $15 \times 11$  cm.).
- (4) Left lateral vertical ( $15 \times 11$  cm.).
- (5) Perineal ( $8 \times 8$ — $10 \times 10$  cm.).

In obese patients with large pelves, up to nine fields are selected :

- (1) Anterior vertical ( $20 \times 20$  cm.).
- (2) Posterior vertical ( $20 \times 20$  cm.).
- (3) Right lateral vertical ( $20 \times 15$  cm.).
- (4) Left lateral vertical ( $20 \times 15$  cm.).
- (5) Right anterior oblique ( $20 \times 11$  cm.).
- (6) Right posterior oblique ( $20 \times 11$  cm.).
- (7) Left anterior oblique ( $20 \times 11$  cm.).
- (8) Left posterior oblique ( $20 \times 11$  cm.).
- (9) Perineal ( $11 \times 11$  cm.).

In some cases where there is the possibility of very widespread abdominal metastasis, such as malignancy of ovary or Fallopian tube, the pelvis and a selected area of abdomen are treated as one open field, centrings being taken at different levels, so that a larger area may receive a uniform dose.



PRECAUTIONS TAKEN DURING IRRADIATION AND THE EFFECTS  
OF IRRADIATION

*During Irradiation.* During irradiation and for three weeks following, washing of the irradiated areas is contra-indicated, bathing with warm normal saline being only permitted in the case of discharges. The skin over the irradiated surfaces should remain untouched by any preparation other than a powder composed of a mixture of equal parts of talc and starch, which should be used freely. Pressure and friction from clothes or other sources should be avoided.

In patients confined to bed, pressure should be evenly distributed by frequent change of position. The application of hot water bottles, etc., to the region under treatment should be avoided.

In the vast majority of cases there are no untoward symptoms whatever during treatment.

In a very large proportion of cases patients state that they feel a general improvement in their condition when about half their dose has been given, followed later in a small percentage of cases by slight nausea and anorexia towards the end of their course, symptoms which very rarely call for any interference.

In about 1 per cent of cases there may be serious nausea and vomiting, but in these there has usually been some indication of this before treatment was started. In these cases, chloretone 3 grs. p.r.n. and/or daily intramuscular doses of liver extract usually prove effective. The absence of X-ray sickness amongst these cases is probably due to :

- (1) Adequate protection from adventitious radiation.
- (2) Adequate ventilation of treatment theatre.
- (3) Efficacy of anti-corona system.
- (4) The use of relatively high intensity of X-radiation. This ensures the minimum of time spent in the theatre, and consequently a minimum of theatre air breathed, with also perhaps a psychological element. The shorter the time taken in treatment, the smaller the volume of blood passing through the irradiated area during that treatment.

Towards the end of a course, patients usually develop some degree of a radiogenic colitis, proctitis, and cystitis, which, however, rarely give rise to anxiety, as they are easily controlled by simple medical measures.

In this connection should be mentioned the avoidance of any foods containing undigestible solid material, and the administration of

purges, during and for three weeks after treatment. The best laxative to employ as a routine in all cases is liquid paraffin.

Towards the end of a course patients usually complain of some irritation and soreness in the region of vulva and anus, which is best treated with liberal applications of lanoline. At this time there are usually early signs of radiogenic erythema and signs of epilation. The mucous membranes of the vulva and vagina show varying degrees of filming. In cases where there was hæmorrhage from the vagina or rectum this is usually controlled or stopped. Pain is almost universally relieved or suppressed. Discharges are usually checked or obliterated, and this is especially noticeable in those with offensive odours. About a week after treatment there is frequently, in addition to a well-marked and general erythema, some slight degree of vesication, which is treated with lanoline applications.

At the end of about three weeks after treatment a large majority of patients admit improvement in general condition, sleep, appetite, energy, etc., and many are quite fit to carry on their usual occupation. At this time the skin has recovered to a great extent, and is now quite dry, showing extensive desquamation, pigmentation, and complete temporary epilation. Diarrhœa and cystitis have also ceased.

Three months after treatment, in hopeful cases, the general improvement is maintained, weight has increased, and the patient has no symptoms or complaints. The skin over the area treated has now quite recovered, showing only varying degrees of pigmentation, and complete epilation. On examination, if a tumour was originally palpable, this has usually shrunk in varying degrees or may have completely disappeared. On vaginal examination, if there was originally an active ulcer visible, this has usually almost, or quite, healed. Mucous membrane where involved has become smooth and movable over subjacent structures, induration where present has become less rigid, diminished in extent, and less craggy, and has probably been or is becoming replaced by scar tissue. Pelvic viscera, which before treatment were fixed and immovable, have regained varying degrees of mobility. The above findings, of course, apply to hopeful cases, but in those that are doubtful or hopeless, examination at this period shows little, if any, change from the original condition. Absence of change in size of tumours or extent of induration at this time is not, *per se*, evidence of a hopeless prognosis, as cases are by no means infrequent where there have been no observable changes in the local condition for long periods, yet where the patient has definitely improved in general condition and freedom from

symptoms, and no evidence of activity in the region of the primary lesion. It is our practice to give every case a second complete course of radiation within a period of three months from the cessation of the first treatment. Further treatment is undertaken as indicated by the progress of the case.

Each case is seen in the "Follow-up Department" at 3-monthly intervals.

## INDEX

## INDEX

- Abdomen, burst, *see* Burst abdomen  
 enlargement of, 99  
 injuries of, *see* Abdominal wounds  
 local pigmentation of, 100  
 local swelling of, 101  
 movements of, 101  
 operations upon, *see* Abdominal operations  
 palpation of, 103, 108  
 percussion of, 103, 108, 109  
 physical examination of, 99  
 reflexes, 103  
 surgical incisions, *see* Incisions  
 visceral enlargement, 110  
 visible peristalsis, 102  
 wounds of, *see* Abdominal wounds
- Abdominal abscess, *see* Peritoneal abscess
- Abdominal diseases, ascites in, 100, 113  
 auscultation in, 113  
 cutaneous hyperæsthesia and hyper-  
 algesia in, 105  
 physical examination in, 99  
 rectal examination in, 114  
 reflex tenderness in, 105  
 syphilis in relation to, 138  
 vaginal examination in, 115  
 visceral pain in, 104  
 visceral tenderness in, 106
- Abdominal examination, 99
- Abdominal incisions, *see* Incisions
- Abdominal movements, 101
- Abdominal operations, anaesthesia in, 53  
 complications following, 1215  
 acute suppurative parotitis, 1242  
 bed sore, 1249  
 burst abdomen, 1222  
 fæcal fistula, 1238  
 femoral thrombosis, 1257, 1258  
 infected wounds, 1215  
 pain, 1253  
 peritonitis, 1132  
 persistent abdominal sinus, 1221  
 phlebitis, 1256  
 retention of urine, 1245  
 thirst, 1251  
 diabetes in relation to, 790  
 Abdominal reflexes, 103  
 Abdominal sinus, persistent, 1221  
 Abdominal wall, anaesthesia in operations  
 upon, 53  
 protective response of, 104  
 retraction of, in dyspeptic states, 100  
 Abdominal watersheds, 1100, 1199, 1212  
 Abdominal wounds, disruption of, *see*  
 Burst abdomen  
 infection of, 1215  
 incidence, 1215  
 localised hæmatoma in, 1219  
 persistent abdominal sinus in, 1221  
 predisposing factors in, 1215  
 treatment of, 1219
- Abel on survival rate after radical opera-  
 tion for cancer of the rectum,  
 1473
- Abscess, amœbic, of liver, 740, 764  
 appendix, 928  
 iliac, 1145  
 intra-peritoneal, 1142  
 liver, 740  
 pancreatic, 784, 787  
 pelvic, 930, 1150  
 peri-colic, 947  
 perigastric, 221  
 peritoneal, 1142  
 residual, 1142  
 secondary, 1142  
 splenic, 842  
 staphylococcal, of liver, 743  
 stitch, 1217, 1219  
 subphrenic, 221, 1153  
 tropical, of liver, 740
- Acetyl-choline, and intestinal peristalsis,  
 1070, 1088  
 in treatment of paralytic ileus, 1202

- Acetylene anaesthesia, 9
- Achlorhydria, significance of, in gastro-intestinal diseases, 122
- Acholic jaundice, *see* Hemolytic jaundice
- Acidity, gastric, significance of, 122  
tests for, 121, 122
- Acoine as local analgesic, 29
- Actinomycosis, of appendix, 937  
of colon, 947  
X-ray diagnosis of, 1521  
of liver, 747  
of the right iliac fossa, 937
- Adams on acute jejuno-gastric intussusception, 400, 401
- Adeno-carcinoma of colon, 955, 956  
of rectum, 1411
- Adenoma of colon, 952  
of rectum, 1394  
of small intestine, 595  
of stomach, 418, 521
- Adhesions, perigastric, 255
- Adrepalin in treatment of cardiac failure, 25
- Alcohol and gastro-intestinal disorders, 70
- Alcohol test meal in diagnosis of ulcer-cancer of the stomach, 563
- Alexander on acute glossy cedema of pancreas, 784
- Alimentary tract, *see* Gastro-intestinal tract
- Alkalosis in infantile pyloric stenosis, 148
- Allis's forceps, 327
- Alypin as local analgesic, 29
- Ametox in treatment of toxic jaundice, 779
- Amoebic abscess of liver, 740  
as cause of obstructive jaundice, 764
- Amoebic hepatitis, 740
- Amylocaine as local analgesic, 29
- Anaemia following gastric operations, 402  
in carcinoma of the stomach, 91, 439  
in gastro-intestinal diseases, 92  
pernicious, haemolytic jaundice in, 765  
splenic, *see* Splenic anaemia.
- Anaesthesia, acetylene, 9  
basal narcosis in, *see* Basal narcosis  
blood changes in, 3  
body temperature in, 23, 24  
cardiac failure during, 13, 21, 25  
chloroform, 13  
acute yellow atrophy of liver in, 13  
administration of, 14  
as cause of death, 13  
delayed poisoning in, 13  
effect on blood-pressure, 13  
indications for use of, 14  
insulin premedication in, 13  
metabolic changes in, 13  
mixtures of chloroform and ether, 14  
mode of action, 3  
muscular relaxation obtainable with, 4  
overdosage in, 13  
paralysis of respiratory centre in, 13  
rate of elimination, 4  
toxicity of, 4, 13  
choice of methods, 39
- cyclopropane, 9
- di-ethyl ether, 10
- di-vinyl ether, 13
- endotracheal, 16
- ether, 10  
administration of, 11, 12  
atropine in pre-operative medication, 12  
convulsions in, 12  
de-etherisation after, 12  
ethyl chloride as preliminary to, 10, 11  
excessive sweating in, 12  
hyperpnoea in, 11  
insulin in premedication, 13  
metabolic changes in, 12  
mixtures of chloroform and ether, 14  
muscular relaxation obtainable with, 4  
nitrous oxide as preliminary to, 11  
overheating in, 12  
pre-operative medication in, 12, 13  
pulmonary complications in, 12  
rate of elimination of, 4  
salivation in, 12  
toxicity of, 4  
tremor in, 12  
with nitrous oxide and oxygen, 12
- ethyl chloride, 10  
administration of, 10  
as a preliminary to open ether, 10  
freezing of skin by, 10  
in children, 10  
masseteric spasm in, 10  
muscular relaxation obtainable with, 4  
rate of elimination of, 4

- "single dose" method, 10
- toxicity of, 4
- ethylene, 9
  - administration of, 9
  - advantages and disadvantages of, 9
  - muscular relaxation obtainable with, 4
  - rate of elimination of, 4
  - toxicity of, 4
- evipan, 14, 15
  - administration of, 14
  - contra-indications to use of, 15
  - dangers of, 15
  - effect on blood-pressure, 15
  - effect on respiration, 15
  - evaluation of, 15
  - sequelæ of, 15
- gas, mode of action, 3
- gas-oxygen, 6
  - adjuvants to, 9
- general consideration of, 3
- heart failure during, 24, 25
- hydrocarbon gases, 5
- in abdominal surgery, 53
- in appendicectomy, 54
- in cholecystectomy, 55
- in cranial surgery, 41
- in diabetics, 795
- in excision of rectum, 54
- in gastrectomy, 55
- in gastro-enterostomy, 55
- in gastrostomy, 54
- in hysterectomy, 54
- in operations for hernia, 53, 54
- in operations for hydrocele, 53
- in operations for internal hæmorrhoids, 1284
- in operations for peritonitis, 1116
- in operations for relief of acute intestinal obstruction, 58, 1003
- in operations for relief of perforated gastric ulcer, 58
- in operations for varicocele, 53
- in perineal surgery, 59
- in prostatectomy, 51
- in radical operation for cancer of rectum, 1456
- in removal of ovarian cyst, 54
- in resection of ribs, 50
- in splenectomy, 902
- in suprapubic cystotomy, 54
- in surgery of the neck, 44
- in thoracic surgery, 48
- intra-bronchial, in thoracic surgery, 51
- intravenous, 12
  - with evipan, 14, 15
- local, *see* Analgesia, local
- modern ideal in, 4
- morphia as preliminary to, 19
- narcysten, 9
- nitrous oxide, administration, 5
  - mode of action, 3, 5
  - muscular relaxation obtainable with, 4
  - preparation of, 5
  - rate of elimination, 4
  - toxicity of, 4
- nitrons oxide and air, administration, 5
  - advantages of, 6
  - contra-indications to use of, 5
- nitrons oxide and oxygen, 6
  - advantages of, 6
  - administration of, 6
  - apparatus for, 6
  - closed-circuit apparatus, 6
  - continuous-flow apparatus, 6
  - in abdominal surgery, 53, 58, 59
  - intermittent-flow apparatus, 6
  - premedication in, 6
- operative shock during, 22
- premedication in, 19
- propylene, 9
- rectal instillation of oil-ether, 12
- restorative measures during, 22
- resuscitation after, 22
- scopolamine as preliminary to, 19
- sedative drugs before, 19
- shock during, 22
- sodium evipan, *see* Anæsthesia, evipan
- somnoform, 10
- splanchnic, *see* Analgesia, splanchnic
- temperature in, 23, 24
- theories of action, 3
- vinethene, 13
- (*see also* Analgesia, local; Basal narcosis)
- Anæsthetics, choice of, 39
  - in abdominal surgery, 53
  - in cranial surgery, 41
  - in perineal surgery, 59
  - in surgery of the neck, 44
  - in thoracic surgery, 48
- effect upon metabolism, 3
- lipoid-soluble, 3, 4

Anæsthetics, mode of action, 3  
 muscular relaxation obtainable with, 4  
 oxygen-replacing, 3, 4  
 rate of elimination, 3, 4  
 theories of action, 4  
 toxicity of, 3, 4  
*(see also under names of various anæsthetic agents)*

Anal canal, carcinoma of, *see* Rectum, carcinoma of  
 effect of presence of hæmorrhoids upon tissues of, 1279  
 fistula of, *see* Ano-rectal fistula  
 narrowing of lumen of, following operations for hæmorrhoids, 1290  
 radium therapy in carcinoma of, 1619  
 stricture of, *see* Stricture of anus and rectum

Anal fissure, 1292  
 ætiology of, 1292  
 differential diagnosis of, 1295  
 from diseased conditions of neighbouring pelvic viscera, 1296  
 from submucous fistula, 1296  
 from syphilitic fissures, 1296  
 operations for, 1299  
 Gabriel's operation, 1302  
 operation by incision (sphincterotomy or Boyer's operation), 1299  
 pectenotomy (Miles' operation), 1300  
 stretching the sphincters (Récamier's operation), 1302  
 pathological anatomy of, 1294  
 pathological conditions complicating, 1296  
 symptomatology of, 1293  
 treatment, 1297  
 operative, 1299  
 palliative, 1298  
 post-operative, 1301

Anal fistula, *see* Ano-rectal fistula

Analgesia, local, 26  
 cocaine, 29  
 advantages and disadvantages of, 26, 27  
 alypin, 29  
 amylocaine, 29  
 apothesine, 29  
 apparatus for, 29, 30  
 borocaine, 29  
 butyn, 29  
 caudal block, 35, 37, 59  
 cocaine, 28

combined with basal narcosis, 27  
 combined with general anæsthesia, 27  
 contra-indications to use of, 26, 27  
 co-operation of surgeon in, 27  
 definition of, 26  
 diothane, 29  
 drugs used in, 28, 29  
 eucaine, 29  
 field blocking in abdominal surgery, 55, 56  
 novocaine in, 28  
 percaine in, 29  
 technique of, 32, 33  
 freezing, 33  
 holocaine, 29  
 in cranial surgery, 41, 42  
 in Finocchetto's operation of partial gastrectomy, 527  
 in herniotomy, 1050  
 in operations upon the abdominal wall, 53  
 in perineal surgery, 59, 60  
 in rib resection, 50  
 in surgery of the neck, 44  
 infiltration method, novocaine in, 28  
 percaine in, 29  
 technique of, 31, 32  
 intra-arterial, 34  
 intra-neural block, 33  
 intravenous, 34  
 ischæmia in, 26  
 methods of producing, 31  
 neothetin, 29  
 nerve blocking in, 33  
 by novocaine, 28  
 novocaine, 28  
 in abdominal surgery, 56, 57  
 in cranial surgery, 42  
 pantocain, 29  
 para-neural block, 33  
 paravertebral and splanchnic block in abdominal operations, 56, 57  
 percaine, 29  
 in abdominal surgery, 55  
 quinine and urea hydrochloride, 29  
 sedative drugs as adjuvants to, 27  
 spinal analgesia, 35  
 anatomy and physiology, 35  
 blood-pressure in, 37  
 contra-indications to use of, 36  
 disadvantages of certain methods, 35  
 discomfort during, 38



- headache after, 38  
 hypoharic percaine method of  
   Howard Jones, 36  
 in abdominal surgery, 55  
 in operations for peritonitis, 1116  
 in operations for relief of perforated  
   gastric ulcer, 58  
 in operations for relief of acute  
   intestinal obstruction, 58  
 in perineal surgery, 60  
 in prostatectomy, 54  
 in treatment of paralytic ileus, 1092  
 in vaginal hysterectomy, 60  
 indications for use of, 36  
 morphine and scopolamine prepara-  
   tory to, 36  
 nausea and vomiting in, 38  
 paralyses after, 38  
 percaine in, 29, 35, 36  
 position of patient in, 37  
 respiration in, 37  
 sequelæ of, 38  
 solutions used in, 35  
 technique of, 36  
 vomiting after, 38  
 splanchnic block, Braun's anterior  
   technique, 56  
   in abdominal surgery, 55, 56  
   in treatment of paralytic ileus, 1093  
   Kappis's method, 56  
 stovaine, 29  
 subarachnoid spinal block, 35  
 surface or permeation analgesia, 31  
 technique of, 31  
 trans-sacral block in perineal surgery,  
   60  
 tropacocaine, 29  
 tutocaine, 29  
 Analgesics, local, 28, 29  
 Anastomotic ulcer, *see* Gastric and duo-  
   denal ulcer, secondary  
 Aneurysm of splenic artery, 842  
 Angina pectoris, simulation of gastro-  
   intestinal disease by, 93  
 Angioma of colon, 952  
   of stomach, 522  
 Ano-rectal fistula, 1327  
   ætiology of, 1329  
   classification of, 1330  
   complete, 1331  
   external opening of, 1331  
   intermuscular, 1350  
   clinical course of, 1351  
   differential diagnosis of, 1351  
   operative treatment of, 1351  
   post-operative treatment of, 1352  
 internal opening of, 1332  
 ischio-rectal, 1366  
   clinical course of, 1369  
   differential diagnosis of, 1369  
   operative treatment of, 1370  
   post-operative treatment of, 1371  
   varieties of, 1367  
 main track of, 1334  
 offshoots or extensions from the main  
   track of, 1334  
 operative treatment of, 1335  
   general principles in the technique of,  
   1340  
 palliative treatment of, 1335  
 para-rectal, 1352  
   clinical course of, 1356  
   differential diagnosis of, 1356  
   operative treatment of, 1357  
   post-operative treatment of, 1360  
   varieties of, 1353  
 post-operative treatment of, 1341  
 subcutaneous, 1342  
   clinical course of, 1344  
   differential diagnosis of, 1343  
   operative treatment of, 1344  
   post-operative treatment of, 1345  
   varieties of, 1342  
 submucous, 1345  
   clinical course of, 1348  
   differential diagnosis of, 1348  
   operative treatment of, 1348  
   post-operative treatment of, 1348  
   varieties of, 1346, 1347  
 sub-sphincteric, 1361  
   differential diagnosis of, 1361  
   operative treatment of, 1365  
   post-operative treatment of, 1365  
   varieties of, 1362  
 treatment of, 1335  
 varieties of, 1330  
 Anorexia in carcinoma of the stomach, 82,  
   439  
   in dyspepsia, 81, 82  
   in gastritis, 82  
 Anschütz and Konjetzny on the incidence  
   of sarcoma of the stomach, 517  
 Anti-gas gangrene serum in treatment of  
   appendicitis, 914, 932

- Antivenin in treatment of purpura hemorrhagica, 877
- Anus, benign tumours of, 1390
- carcinoma of, 1408
- fibro-cellular tumour of, 1392
- lipoma of, 1393
- papilloma of, 1390
- symptoms, 1391
- treatment, 1392
- pruritus of, *see* Pruritus ani
- soft fibroma or fibro-cellular tumour of, 1392
- stricture of, *see* Stricture of anus and rectum
- (*see also* Anal Canal)
- Apoplexy, pancreatic, 783
- Apothesine as local analgesic, 29
- Appendicectomy, adhesive obstruction following, 1073
- anaesthesia in, 54
- drainage of the peritoneal cavity in, 923
- drainage of the wound in, 924
- incisions for, 920
- post-operative complications, 934, 935
- prevention of post-operative obstruction in, 1083
- retrograde appendicectomy, 923
- sequelæ of, 934, 935
- technique of, 920, 921
- Appendices epiploicæ, torsion of, 932
- Appendicitis, 909
- acute, acute appendicitis proper, 914
- acute appendicular obstruction, 914, 915
- age incidence of, 915
- atypical, 916
- causing peritonitis, 1131
- clinical features of, 915
- complications and sequelæ of, 928, 934, 935
- appendix abscess, 928
- internal faecal fistula, 934
- paralytic ileus, 935
- pelvic abscess, 930
- peritonitis, 932
- post-operative, 934, 935
- pregnancy, 933
- pylephlebitis, 934
- subphrenic abscess, 1163
- diagnosis, 916
- differential diagnosis, 918
- from abdominal influenza, 918
- from early pneumonia and pleurisy, 918
- from early pregnancy, 919
- from ectopic pregnancy, 919
- from right renal colic or pyelitis, 918
- from ruptured lutein cyst (apoplectic ovary), 919
- from salpingitis, 919
- from suppurating deep iliac glands, 919
- hamatemesis in, 82
- high temperature in early cases of, 917
- in pregnancy, 933
- leucocyte count in, 917, 918
- pain in, 76
- pathology of, 914
- pelvic abscess in, 930
- peritonitis complicating, 1131
- sex incidence of, 915
- simulation of, in children, by lobar pneumonia, 95
- subphrenic abscess complicating, 1163
- symptoms of, 916
- time factor in treatment of, 924
- treatment of, 920
- anti-gas gangrene serum in, 914, 932
- appendicectomy, 920
- Ochsner-Sherren (delayed) treatment, 924
- time factor in, 924
- when to operate in, 924
- with diarrhoea, 917
- with general peritonitis, 932
- with local abscess, 928, 929
- without abdominal rigidity, 917
- etiology of, 911
- abuse of purgatives, 913
- concretions and strictures, 912
- familial susceptibility, 912
- race and diet, 912
- role of fats and fatty acids, 912
- worms and other foreign bodies, 912
- as an endemic disease, 912
- bacteriology of, 913
- Aschoff's organism, 914
- bacillus *terrestris* capsulatus (B. Welchii), 914
- bacillus coli communis, 913

- streptococcus faecalis, 913  
 chronic, 936  
   constipation in, 84  
   diagnosis of, 936  
   " hunger pain " in, 77  
   nausea as symptom in, 77  
   pain in, 75  
   pathology of, 936  
 hæmatemesis associated with dyspepsia  
   in, 82  
 in apes, 909, 913  
 McBurney's point in, 909  
 mortality of, 909  
 psoas spasm in, 919  
 radiological diagnosis of, 936, 1517  
 recurrence of, 936  
 Rovsing's sign in, 916  
 Sherren's skin triangle in, 916  
 sub-acute, 935  
 X-ray diagnosis of, 936, 1517  
 Appendicostomy in treatment of ulcer-  
   ative colitis, 945, 946  
   technique of, 945  
 Appendicular obstruction, acute, 914, 915,  
   916  
 Appendix, abscess of, 928  
   (see also Peritoneal abscess)  
   actinomycosis of, 937  
   anatomy of, 909, 911  
   blood-vessels of, 911  
   carcinoma of, 938  
   carcinoid tumour of, 938  
   diseases of, 909  
   diverticula of, 938  
   duplication of, 910  
   globocellularis or carcinoid tumour of,  
     613  
   inflammation of, see Appendicitis  
   lymphatics of, 911  
   McBurney's point, 909  
   minute anatomy of, 911  
   misplacements of, 910  
   positions of, 910  
   retrocæcal position of, 910  
   surface anatomy of, 909  
   surgical anatomy of, 909  
   variations in position of, 910  
   vascular supply of, 911  
   X-ray examination of, 1515  
 Appendix abscess, 928  
 Appendix dyspepsia, 73  
 Appetite in dyspepsia, 82  
   in gastric ulcer, 197  
   in hour-glass stomach, 272  
   in pyloric obstruction, 291  
 Argentaffian tumour of small intestine,  
   613  
 Arsenic in treatment of purpura  
   hæmorrhagica, 877  
 Aschoff on the causation of gall-stones,  
   727  
   organism associated with appendicitis,  
     914  
 Ascites, 100, 113  
   due to portal cirrhosis, 744  
   in splenic anæmia, 868  
   treatment of, 744  
 Asthenic constitution in dyspepsia, 90  
 Atresia, congenital, of colon, 944  
 Atropine in pre-operative medication, 12,  
   13  
 Auerbach's plexus in relation to paralytic  
   ileus, 1067  
 Autogenous blood, by intramuscular  
   injection, in purpura hæmo-  
   rrhagica, 876  
 Autolytic peritonitis in injury of the  
   liver, 739  
 Avertin basal narcosis, 19, 20  
   contra-indications to use of, 20  
   in cranial surgery, 42  
   in operations for toxic goitre, 27, 45, 46
- B
- Back's results in posterior gastro-  
   jejunostomy, 384  
 Backache in dyspepsia, 75  
   in peptic ulceration, 75  
 Bacteriology of appendicitis, 913, 914  
   of peritonitis, 1106  
 Bacteriophage in treatment of peritonitis,  
   1124  
 Bag, tripartite rubber, for operating  
   tables, 666  
 Bailey's apparatus for delivery of anæ-  
   sthetic solution under pressure,  
   30  
 Baker (Morant) forceps, 921  
 Balfour's cautery method for destruction  
   of gastric ulcers, 361  
   method of ante-colic Polya anastomosis,  
     495

- Balfour's modification of Mayo's operation of direct implantation of the hepatic duct into the duodenum, 711
- on gastric polyposis, 522
- on hereditary factor in gastric carcinoma, 417
- on incidence of anastomotic ulcer, 404
- on incidence of gastric carcinoma, 417
- operation for gastric ulcer, 311
- results in operations for gastric carcinoma, 443
- (*see also* Polya-Balfour operation)
- Balfour and Henderson's results of operations for benign tumours of small intestine, 600, 621
- Bands, intestinal, as cause of acute obstruction, 1011
- Banti's disease, *see* Splenic anæmia
- Barbiturates as basal narcotics, 20, 21
- Bargen's serum in treatment of ulcerative colitis, 945
- Basal narcosis, 19, 20
- avertin, 19
- in cranial surgery, 42
- in operations for toxic goitre, 45, 46
- combined with local analgesia, 27
- coramine as antidote in overdose, 21
- emergencies in, 21
- evipan, 21
- hebaral sodium, 20
- in operations for toxic goitre, 45, 46
- nembutal, 20
- overdosage in, 21
- paraldehyde, 19
- pernocton, 20
- sodium amytal, 20
- sodium evipan, 21
- sodium soneryl, 20
- (*see also* Anæsthesia; Analgesia)
- Battle's incision in appendicectomy, 920
- Beard's reducing valve for gas-oxygen apparatus, 7
- Bed-lifter, Hoskin and Sewell's, 926
- Bedsore, 1249, 1250
- Belching in dyspepsia, 79
- Benedict and Allen on adenomata of the stomach, 521
- Benzidine test for occult blood, 128
- Bertrand on sarcoma of the stomach, 517
- Berven's results in radium treatment of cancer of the tongue, 1587
- Bier's method of pyloric occlusion for gastric and duodenal ulcer, 336, 337
- Bile-duct, common, carcinoma of, as cause of obstructive jaundice, 83, 762
- cholecysto-enterostomy in diseases of, 688
- congenital cystic dilatation of, 733
- complications of, 734
- diagnosis of, 733
- operations for, 734
- operative mortality in, 735
- rupture of cyst in, 734
- symptoms of, 733
- treatment of, 734
- X-ray diagnosis of, 733
- exploration of, in cases of gall-stones, 652
- gall-stones in, 650, 759
- (*see also* Gall-stone disease)
- Moynihan's operation of rotation of the duct, 694
- obstruction of, 658
- causes, 658
- loss of weight in, 81
- treatment, 659
- obstructive jaundice due to stenosis of, 758
- operations upon, *see* Bile-ducts, operations
- operative trauma to, 632, 633
- reconstruction operations upon, 710
- results of operations for stones in, 721
- rotation of (Moynihan's operation), 694
- Bile-ducts, abnormalities of, 628
- anatomy of, 629
- calculi in, *see* Gall-stone disease
- carcinoma of, 638
- diagnosis, 640
- symptoms, 640
- treatment, 640
- diseases, 627
- duodenal intubation in diseases of, 139
- gall-stones in, *see* Gall-stone disease
- inflammation of, *see* Cholangitis
- injuries of, 627, 635
- operations upon, 664, 692
- anastomosis between coned-out

- biliary fistula and the duodenum, 710  
 Balfour's modification of Mayo's operation of direct implantation of hepatic duct into duodenum, 711  
 causes of death after, 722  
 choledocho-choledochostomy, 706  
 choledochotomy and choledochostomy, 692  
 Coffey's modification of Sullivan's operation of end-to-side choledochoduodenostomy, 712  
 end-to-side choledocho- or hepaticoduodenostomy, 710  
 exploratory laparotomy in, 668  
 general considerations, 661  
 hepatico-jejunostomy, 718  
 incisions, 667  
 instruments, 664  
 isolation of operative fields, 669  
 Kocher's operation of trans-duodenal choledochostomy, 704  
 lateral choledocho-duodenostomy, 709  
 McBurney's operation of duodeno-choledochotomy or ampullary choledochostomy, 702  
 Mayo's operation of direct implantation of hepatic duct into duodenum, 711  
 Moynihan's operation of rotation of the duct, 694  
 position of patient upon operating table, 664  
 post-operative treatment, 718  
 reconstruction operations, 710  
 retro-duodenal choledochotomy, 700  
 rotation of the liver in, 668  
 supra-duodenal choledochotomy, 694  
 technique of, 664  
 trans-duodenal choledochotomy, 702  
 Walton's operation of indirect implantation of common bile-duct into duodenum, 712  
 operative trauma to, 628, 632, 633  
   treatment of, 634  
 pre-operative treatment in diseases of, 661  
   rupture of, 635, 636  
 Bile-pigment, formation of, 748  
   Fouchet's test for, 754  
   icterus index, 754  
   Van den Bergh reaction, 752  
     (see also Jaundice)  
 Bilharzia mansoni and Egyptian splenomegaly, 898  
 Biliary calculi, *see* Gall-stones; Gall-stone disease  
 Biliary fistula, 657  
   cholecystectomy in, 678  
   external, 657  
     transplantation of, 710  
     treatment of, 658  
   internal, 657  
     causes of, 657  
     treatment of, 658  
   operation for anastomosis between coned-out fistula and duodenum, 710  
 Biliary passages, anatomy of, 629  
   causes of death after operations upon, 722  
   gall-stones in, *see* Gall-stones, Gall-stone disease  
   operative trauma to, 628  
   results of operations upon, 719  
 Bilirubin, 748  
   formation of, 748  
   Fouchet's test for, 754  
   icterus index, 754  
   renal threshold for, 763  
   Van den Bergh reaction for, 750, 752, 763  
 Billroth I operation, *see* Péan-Billroth I operation  
 Biochemical investigations in dyspepsia, 116  
 Bladder, radium therapy in carcinoma of, 1619  
   X-ray diagnosis in diseases of, 1553  
   X-ray examination of, 1540, 1552  
 Bleeding time in purpura hæmorrhagica, 875  
 Blood analysis in jaundice, 752, 754  
   autogenous, by intramuscular injection, in purpura hæmorrhagica, 876  
   changes after splenectomy, 817  
   cholesterol content of, 133  
   coagulation time in jaundice, 775, 779  
   dyscrasias, hæmatemesis in, 82  
   effect of anæsthesia upon, 3  
   effect of X-ray irradiation of the spleen upon, 820

- Blood, in Gaucher's disease, 864  
 in hæmolytic jaundice, 884, 886, 887  
 in splenic anæmia, 866  
 occult, examination of stools for, 127  
   in diagnosis of ulcer-cancer of the stomach, 562  
   significance of, 129  
   radio-therapy in diseases of, 821  
   regeneration of, after splenectomy, 818  
 Blood count, in diagnosis of apparent dyspeptic states, 138  
   in Egyptian splenomegaly, 899  
 Blood-platelets, 829  
   count, after operations, 834, 836  
   after splenectomy, 832  
   in purpura hæmorrhagica, 875  
   functions of, 833  
   in relation to splenectomy, 837  
   in relation to thrombosis, 831  
   in splenic anæmia, 835  
   increase of, after splenectomy, 831, 832  
   morphology of, 829  
   origin of, 829  
 Blood-pressure, during cranial operations, 43  
   effect of chloroform anæsthesia upon, 13  
   effect of ovarian anæsthesia upon, 15  
   effect of spinal analgesia upon, 37  
 Blood-transfusion, after radical operation for carcinoma of the rectum, 1470  
   before operations for strangulated hernia, 1049  
   in pre-operative treatment of carcinoma of the stomach, 445  
   in purpura hæmorrhagica, 873, 876  
   in treatment of hæmatemesis from gastric and duodenal ulcer, 582  
   in treatment of operative shock, 24  
 Blood urea, clearance test, 135  
   estimation of, 132  
 Body-build in dyspepsia, 90  
 Body heat, loss of, from exposed viscera, 23  
   maintenance of, during operations, 24  
 Body weight, *see* Weight  
 Bohmansson on gastritis and stomal ulceration, 407  
 Bond on increase of the blood-platelets after splenectomy, 831  
 Bone-marrow, changes in, following splenectomy, 818  
 Bonney on cholecystotomy in gall-stone disease, 670  
   self-retaining retractor, 510  
 Borocaine as local analgesic, 29  
 Bormann on duodenal involvement in cancer of the pylorus, 603  
 Boyer's operation for anal fissure, 1299  
 Boyle's continuous-flow apparatus for gas-oxygen anæsthesia, 7  
 Bozemann's needle holder, 327  
 Brain, anæsthesia in surgery of, 41, 42, 43  
   local analgesia in surgery of, 41, 42  
 Braun's anterior technique for splanchnic block, 56  
   operation of gastro-jejunostomy, 360  
 Breast, radium therapy in cancer of, 1599  
 Brenner's operation of gastro-jejunostomy, 360  
 Brinton's description of leather-bottle stomach, 423  
   on duodenal involvement in cancer of the pylorus, 603  
 Brockman on cases of intestinal injury, 616  
 Bromsulphthalein in the testing of liver function, 775  
 Brunn and Pearl on gastric polyposis, 522  
 Brunner's glands, adenoma of, 596  
 Buccal mucosa, radium therapy in cancer of, 1588  
 Bumm's results in sleeve resection of gastric ulcer, 346  
 Burst abdomen, 1222  
   after splenectomy, 817  
   causes of, 1222  
   errors in operative technique, 1225  
   failure of tissues to heal soundly, 1222  
   post-operative complications, 1224  
   contributory factors in, 1224  
   diagnosis of, 1227  
   incidence of, 1227  
   incisions in relation to, 1235  
   mortality of, 1227  
   prophylaxis of, 1228  
   treatment of, 1229  
   re-suture, 1229  
   suture by silver wire, 1232  
   tampon method, 1236  
   use of drainage-tubes in relation to, 1226  
 Butyn as local analgesic, 29

## C

- Cachexia in gastric carcinoma, 91
- Cade's results in radium therapy of cancer of the breast, 1607
- results in radium therapy of cancer of the tongue, 1587
- Cæcectomy, technique of, 963
- Cæcostomy, 969
- after-care of, 970
- for acute intestinal obstruction, 1007
- in treatment of ulcerative colitis, 945, 946
- technique of, 969
- Cæcum, carcinoma of, 958
- excision of, 963
- volvulus of, 1030
- Calcium in treatment of purpura hæmorrhagica, 877
- Calculi, biliary, *see* Gall-stones; Gall-stone disease
- pancreatic, 788
- Cancer, *see* Carcinoma
- Carcinoid tumour, of appendix, 938
- of small intestine, 613
- Carcinoma, high voltage X-rays in treatment of, 1671
- of anus, 1408
- of appendix, 938
- of bile-ducts, 638
- of cæcum, 958
- of cervix uteri, 1643
- of colon, 954
- of duodenum, 603
- of gall-bladder, 638, 730
- of liver, 745
- of rectum, 1409
- of small intestine, 602
- of stomach, 416
- of uterus, 1658
- radio-therapy of, 1628
- radium therapy of, 1557
- Cardiolysis for pericardial adhesions, anaesthesia in, 49
- Catarrhal jaundice, 766
- aetiology, 758, 766
- course of, 767
- differential diagnosis of, 768
- symptoms of, 767
- Caudal block, 35, 37
- in perineal surgery, 59
- Cantery excision of gastric ulcer, 341
- Post electric, 328
- Cellulitis of stomach, *see* Gastritis, acute phlegmonous
- Cervix uteri, carcinoma of, 1643
- aetiology of, 1643
- definition of International Stages 1-4, 1647
- diagnosis of, 1645
- pathology of, 1644
- radio-therapy of, 1643, 1646
- history of, 1650
- Paris technique, 1653
- results of, 1655, 1656, 1657
- Stockholm technique, 1650
- technique of, 1649
- treatment of, 1646
- radio-therapy in carcinoma of, 1643
- radio-therapy in tuberculosis of, 1670
- Cheate's gall-stone scoop, 665
- Chlides' forceps Kifa clips, 489
- Chloroform anaesthesia, 3, 13
- action of, 3
- acute yellow atrophy of liver in, 13
- as cause of death, 13
- by means of Junker's inhaler, 14
- cardiac failure in, 13
- delayed poisoning in, 13, 769
- effect on blood-pressure, 13
- effect on peristalsis, 1070
- indications for use of, 14
- insulin premedication in, 13
- metabolic changes in, 13
- mixtures of chloroform and ether, 14
- muscular relaxation obtainable with, 4
- open lint method, 14
- overdosage in, 13
- paralysis of respiratory centre in, 13
- rate of elimination of, 4
- toxicity of, 4, 13
- Cholæmia, 771
- aetiology, 771
- and "liver deaths" after operations, 772
- diagnosis of, 772
- jaundice in, 772
- medical treatment of, 780
- pathology of, 772
- regeneration and repair of liver in, 772
- sub-acute necrosis of liver in, 772
- symptoms of, 771

- Cholangitis, cholecystostomy in, 672  
 duodenal intubation in, 140  
 in hæmolytic jaundice, 885  
 obstructive jaundice due to, 758  
 with jaundice, 663
- Cholecystectomy, 678  
 advantages of, in cholecystitis, 653, 654  
 anaesthesia in, 55  
 in acute pancreatitis, 786  
 in chronic cholecystitis, late results of, 720  
   operative mortality, 719  
 indications for, 678  
 operative mortality of, 646  
 operative trauma during, 628  
 partial, 678  
 technique of, 678  
   with choledochostomy, operative mortality of, 721
- Cholecystendysis in gall-stone disease, 670
- Cholecystitis, acute, 654  
 advantages of immediate cholecystectomy for, 654  
 complications of, 729  
   empyema of the gall-bladder, 729  
   general peritonitis, 729  
   local peritonitis, 729  
   subphrenic abscess, 729  
   suppurative pyelphlebitis, 729  
 expectant or delayed treatment, 661  
 indications for conservative measures in, 655  
 intravenous salines in, 661  
 operative mortality in, 721  
 pre-operative treatment of, 661  
 starvation during treatment of, 661  
 treatment of, 654
- association with dyspepsia, 72  
 causation of, 727  
 choice of operation for, 644, 648  
 cholecystectomy in, 678  
 cholecystostomy in, 672  
 chronic, 653  
   complications of, 729  
   fistula due to, 729  
   in hæmolytic jaundice, 885  
   medical treatment of, 662  
   operative mortality of cholecystectomy in, 719  
   pre-operative treatment of, 662  
   treatment of, 653  
 due to gall-stones, 728
- duodenal intubation in, 140  
 "hunger pain" in, 77  
 in relation to carcinoma of the gall-bladder, 639  
 indications for operation in, 642  
 pain in, 76  
 pre-operative treatment of, 661  
 toxic effects of, 613  
 treatment of, 653  
 X-ray diagnosis of, 1534
- Cholecysto-duodenostomy, technique of, 692
- Cholecysto-enterostomy, 687  
 complications of, 687  
 indications for, 688
- Cholecysto-gastrostomy, for inaccessible ulcers of the stomach, 312  
 technique of, 689
- Cholecystogram, normal, 724
- Cholecystography, 1527
- Cholecysto-jejunostomy, technique of, 692
- Cholecystostomy, 672  
 biliary fistula after, 657  
 in acute cholecystitis, operative mortality of, 721  
 in chronic cholecystitis, late results of, 720  
   operative mortality of, 720  
 indications for, 672  
 operative mortality of, 721  
 technique of, 672
- Cholecystotomy in gall-stone disease, 670
- Choledcho-choledochostomy, 706
- Choledcho-duodenostomy, Coffey's modifications of Sullivan's operation, 712  
 lateral, 709  
 Walton's operation of indirect transplantation, 712
- Choledochostomy, for removal of calculi, 692  
 Kocher's operation, 704  
 McBarney's operation, 702  
 operative mortality of, 721
- Choledochotomy, for removal of calculi, 692  
 McBarney's operation, 702  
 retro-duodenal, 700  
 supra-duodenal, 694  
 trans-duodenal, 702
- Cholelithiasis, *see* Gall-stones; Gall-stone disease



- Chorion epitelioma, radio-therapy of, 1660
- Cirrhosis of liver, *see* Liver, cirrhosis of, 855
- Clamps, for gastrectomy operations, 460  
Friedrich-Petz, 329, 330  
Payr's stomach, 328  
de Petz, 329  
Sherren's, 328
- Clausen's face-piece retainer for inhalation anaesthesia, 47, 49
- Clover's inhaler, 10, 11
- Coagulents in treatment of purpura hæmorrhagica, 877
- Cocaine as local analgesic, 28
- Coffey's modification of Sullivan's operation of end-to-side choledocho-duodenostomy, 712  
operation of jejunostomy, 458  
two-stage abdomino-perineal operation for carcinoma of the rectum, 1453, 1454
- Cohen and Colp's results in local excision of duodenal carcinoma, 607, 621
- Cokkinis on mesenteric embolism and thrombosis, 1030
- Colectomy, partial, Devine's technique for, 968
- Colic, renal, *see* Renal colic
- Colitis, chronic, pain in, 75  
diarrhoea in, 84  
ulcerative, 944  
bacteriology of, 945  
perforation in, 946  
sequelæ of, 947  
sigmoidoscopy in, 945  
treatment of, 945  
appendicostomy in, 945, 946  
Bargen's serum, 945  
cæcostomy, 945, 946, 969  
ileostomy, 945, 946  
in cases of perforation, 946  
operative, 945  
rectal oxygen insufflations, 945  
tuberculous, 947  
value of surgical treatment of, 945  
X-ray diagnosis of, 1520
- Collapse under anaesthesia, 22
- Colloid carcinoma of rectum, 1412
- Colon, actinomycosis of, 947  
X-ray diagnosis, 1521  
adeno-carcinoma of, 955, 956  
adenoma of, 952  
anatomy of, 943  
angioma of, 952  
atresia of, congenital, 944  
appendices epiploicæ, torsion of, 952  
ascending, carcinoma of, 958  
benign tumours of, 952  
X-ray diagnosis, 1523  
blood and lymph supply of, 943  
carcinoma of, 954  
acute obstruction in, 960, 961  
clinical types of, 960  
chronic obstruction in, 960  
diagnosis of, 958  
left-sided carcinoma, 960  
obstruction in, 957  
of cæcum and ascending colon, 958  
operative treatment of, 961  
contra-indications to, 956  
in absence of acute obstruction, 961  
in presence of acute obstruction, 961  
results of, 956  
sites of, 956, 957  
spread of, 962  
ulcerative, 960  
X-ray diagnosis of, 1524  
congenital affections of, 944  
congenital atresia, 944  
congenital mesenteries, 944  
megacolon, 944  
diverticula of, 950  
and malignant disease, 950  
incidence of, 950  
treatment of, 952  
varieties of, 950  
X-ray diagnosis of, 1521  
functions of, 943  
inflammation of, *see* Colitis  
left-sided, X-ray diagnosis of, 1526  
lipoma of, 952  
myoma of, 952  
new growths of, 952  
operations on, 963  
cæcostomy, 963  
cæcostomy, 969  
colostomy, 969  
Devine's operation of partial colectomy, 968  
Mikulicz-Paul operation, 963, 966  
resection of the sigmoid colon, 965  
perforation of, in ulcerative colitis, 946

Colon, polyposis of, 954  
 p-endo-polyposis of, following ulcerative colitis, 947  
 recto-sigmoid, stricture of, 947  
 sigmoid, carcinoma of, 960  
 resection of, 965  
 stricture of, 947  
 stricture of, in ulcerative colitis, 917  
 surgical diseases of, 944  
 swelling of, 112  
 tuberculous infection of, 947  
 volvulus of, 948  
 X-ray diagnosis in diseases of, 1520  
 X-ray examination of, 1518  
 Colostomy, 969  
 openings, after-care of, 969  
 technique of, 969  
 Colp on burst abdomen, 1223, 1227  
 Common bile-duct, *see* Bile-duct, common  
 Congenital pyloric stenosis, *see* Pyloric stenosis, infantile  
 Connell's suture, 330  
 Constipation, and anal fissure, 1292  
 as ætiological factor in production of hæmorrhoids, 1279  
 in acute intestinal obstruction, 992  
 in carcinoma of the rectum, 1414  
 in dyspepsia, 84  
 in gastro-intestinal diseases, 84  
 in infantile pyloric stenosis, 148  
 in pyloric obstruction, 292  
 Convulsions during ether anæsthesia, 12  
 Cope on acute dilatation of the stomach, 159  
 Coprosthesis in acute intestinal obstruction, 992  
 Coramine as antidote in barbiturate poisoning, 21  
 in treatment of operative shock, 24  
 Coronary thrombosis simulating abdominal disease, 94  
 Cough, chronic, as a cause of abdominal pain, 96  
 Cullen's sign in acute pancreatitis, 786  
 Cushing's stitch, 489  
 Cyclopropane anæsthesia, 9  
 in thoracic surgery, 9, 49  
 Cytotropin in treatment of acute cholecystitis, 661  
 Cystic artery, abnormalities of, 630  
 operative trauma to, 628  
 Cystic duct, abnormalities of, 628

absence of, 628  
 operative trauma to, 632  
 Cystotomy, suprapubic, anæsthesia in, 54  
 Cysts, dermoid, *see* Dermoid cysts  
 hydatid, *see* Hydatid cysts  
 of the spleen, 845  
 of the stomach, 523

## D

D'Annoy and Zoeller on incidence of sarcoma of the stomach, 517  
 Deaver's retractor, 665  
 Debenham on retrograde intussusception of the jejunum following gastro-jejunostomy, 402  
 Dehiscence of abdominal wounds, *see* Burst abdomen  
 Depressor, Sargent's, 920  
 Dermoid cysts of the spleen, 846  
 Desjardins' gall-stone forceps, 665  
 D'Espère on incidence of gastric carcinoma, 416  
 Devine's enterotome, 967  
 method of pyloric occlusion for gastric and duodenal ulcer, 336, 337  
 operation for gastric carcinoma, 459  
 technique for partial colectomy, 968  
 Diabetes, effect of operations upon insulin requirement in, 800  
 hypoglycæmia in surgical cases, 799  
 in relation to abdominal surgery, 790  
 in relation to acute surgical conditions, 796  
 post-operative treatment, 797  
 pre-operative treatment, 797  
 precautions during operation, 797  
 in relation to non-urgent surgical conditions, 791  
 anæsthesia, 795  
 operative precautions, 795  
 post-operative care, 795  
 pre-operative treatment, 791  
 surgical considerations in, 790  
 Diaphragmatic hernia, *see* Hernia, diaphragmatic  
 Diarrhœa in dyspepsia, 84  
 Diathermy in surgery of the neck, 45  
 in thoracic surgery, 52  
 Diathesis in peptic ulceration, 70, 181

- Diet as ætiological factor in appendicitis, 913  
 in dyspepsia, 70  
 Fraser's course of diet after gastro-enterostomy, 389, 390  
 in treatment of jaundice, 776  
 in treatment of peptic ulcer, 575  
 Lenhartz, 388, 390
- Di-ethyl ether anaesthesia, 10
- Dietl's crisis and pyloric obstruction, 289
- Dilatation of common bile-duct, *see* Bile-duct, common, congenital cystic dilatation  
 of stomach, *see* Stomach, dilatation of
- Diothane as local analgesic, 29
- Diverticula, of the appendix, 938  
 of colon, 930  
 of duodenum, 212  
 X-ray diagnosis of, 1511  
 of stomach, X-ray diagnosis of, 1489
- Diverticulitis, X-ray diagnosis of, 1521
- Di-vinyl ether anaesthesia, 13
- Douglas, pouch of, abscess in, *see* Pelvic peritoneal abscess
- Drainage-tubes in relation to burst abdomen, 1226
- Dukes' classification of rectal carcinomata, 1412
- Dunn's continuous-flow syringe for local analgesia, 30
- Duodenal hands, 218
- Duodenal delay, X-ray diagnosis of, 1513
- Dnodenal drainage in pre-operative treatment of strangulated hernia, 1049
- Duodenal fistula, external, 262  
 diagnosis of, 263  
 expectant treatment of, 263  
 following operations, 262  
 mortality of, 265  
 operative treatment of, 264
- Duodenal fistula, internal, 265
- Duodenal ileus, as a predisposing cause of duodenal and gastric ulcer, 622  
 associated with chronic peptic ulceration, 187  
 associated with visceroprosis, 622, 624  
 causes of, 622  
 chronic, 215, 622  
 pathology of, 622  
 simulating pyloric stenosis, 294  
 symptoms of, 623  
 treatment of, 624  
 X-ray diagnosis of, 623  
 (*see also* Ileus)
- Dnodenal intubation, 139, 771  
 in diagnosis of biliary obstruction or infection, 775  
 in jaundice, 771
- Dnodenal obstruction, in secondary peptic ulceration, 413  
 X-ray diagnosis of, 1512  
 (*see also* Duodenal ileus; Ileus)
- Duodenal ulcer, and duodenal carcinoma, 604  
 chronic, choice of operation for, 314  
 complications of, 319  
 indications for operation in, 306  
 operation for chronic ulcer with stenosis, 314  
 operation for chronic ulcer without stenosis, 314  
 operations for complications of, 319  
 water-brash in, 199  
 constipation in, 84  
 dyspeptic symptoms in, 72  
 family history in, 70  
 hæmatemesis in, 82, 319  
 "hunger pain" in, 77  
 pain in, 74  
 after meals, 72  
 perforation in, 220  
 abscess of liver after suture of, 743  
 pyloric obstruction in, 283  
 X-ray diagnosis of, 1508  
 (*see also* Gastric and duodenal ulcer)
- Duodenitis, constipation in, 84  
 "hunger pain" in, 77  
 X-ray diagnosis of, 1508
- Duodenostomy for perforated ulcer, 248
- Duodenum, benign tumours of, 598  
 X-ray diagnosis of, 1510
- carcinoma of, 603  
 ampullary, 603  
 treatment of, 606  
 and cancer of the pylorus, 603  
 and duodenal ulcer, 604  
 as a cause of obstructive jaundice, 762  
 diagnosis of, 605  
 distribution of, 603  
 incidence of, 603  
 infra-ampullary, 603  
 treatment of, 607  
 pathology of, 603

Duodenum, carcinoma of, supra-ampullary, 603  
 symptoms, 605  
 treatment of, 606  
   by radium, 607  
   operative, 606  
   pre-operative, 606  
 X-ray diagnosis of, 1510  
 chronic dilatation of, *see* Duodenal ileus  
 diverticula of, 212  
   X-ray diagnosis of, 1511  
 drainage of, by Wangensteen's method, 998  
 fistula of, *see* Duodenal fistula  
 ileus of, *see* Duodenal ileus  
 inflammation of, *see* Duodenitis  
 injuries of, 615  
 intubation, *see* Duodenal intubation  
 mobilisation of, by Kocher's method, in retro-duodenal choledochotomy, 700, 701  
 perforation of, pain in, 76  
 rupture of, 619  
 tumours of, 593  
 X-ray diagnosis in diseases of, 1508  
 X-ray examination of the normal, 1507  
 Duracaine as local analgesic, 28  
 Dyspepsia, abdominal examination in, 99  
   alcohol in relation to, 70  
   analysis of individual symptoms in, 74  
   anorexia in, 81, 82  
   appendix, 73, 205  
   appetite in, 77  
   asthenic constitution in, 90  
   belching in, 79  
   biochemical investigations in, 116  
   blood count in, 138  
   body-build in, 90  
   condition of bowels in, 84  
   constipation in, 84  
   description of attacks, 73  
   diarrhoea in, 84  
   diathesis in, 70  
   diet in relation to, 70  
   duodenal intubation in, 139  
   duration and progress of symptoms in, 71  
   enlargement of viscera in, 110  
   eructations in, 79, 80  
   examination of urine in, 131

family history in, 70  
 fauces in, 99  
 flatulence in, 79  
   due to gall-stones, 731  
 form for case-histories, 64  
 "functional dyspepsia," 203  
 gastric analysis in, 116  
 hæmatemesis in, 82  
 heartburn in, 79, 80  
 "hunger pain" in, 77  
 importance of case-history in investigation of, 69  
 in chronic cholecystitis, 72  
 in cirrhosis of liver, 98  
 in duodenal ileus, 623  
 in gall-stone disease, 72  
 in gastritis, 72  
 in peptic ulceration, 72  
 in renal disease, 98  
 in visceroptosis, 72  
 initial symptoms in, 71  
 intermittent, in peptic ulceration, 72  
 intermittent symptoms in, 72  
 investigation of a case of, 63  
   biochemical investigations, 116  
   case-history, 69  
   exploratory laparotomy, 89  
   gastric analysis, 116  
   gastro-photography, 88  
   gastroscopy, 87  
   physical examination, 90  
   suggested form for case-history, 63  
   test meals, 119  
   with special instruments, 87  
   X-ray examination, 1483  
 jaundice in, 71, 92  
 Kahn test in, 138  
 long history of, 72  
 melæna in, 82  
 methods of investigation in, 63  
 mouth and throat in, 98  
 multiple organic lesions in, 86  
 nausea in, 77  
 necessity of systematic physical examination in, 92  
 neck in, 99  
 nutrition in, 91  
 pain in, 74  
   accompanied by collapse, 76  
   associated with pyrexia, 76  
   character of, 75  
   constant, 76

"hunger pain," 77  
   position of, 74  
   radiation of, 74  
 personal habits in, 70  
 physical examination in, 90  
 physique in, 90  
 previous illnesses of patients with, 71  
 pyrosis in, 79, 80  
 reflex, 205  
 regurgitation in, 79, 80  
 renal efficiency tests in, 131  
 sense of fullness in, 79, 80  
 sex factor in, 91  
 short history of, 71, 72  
 simulation of, by cardiac and pulmonary diseases, 93  
 skin in, 92  
 social and personal state in, 70  
 sthenic constitution in, 90  
 stools in, 82, 127  
 symptoms of, 71  
 syphilis in relation to, 70, 138  
 teeth in, 99  
 test meals in investigation of, 119  
 tobacco smoking in relation to, 70  
 tongue in, 98  
 urine examination in, 131  
 visceral enlargement in, 110  
   of colon, 112  
   of gall-bladder, 112  
   of kidney, 111  
   of liver, 110  
   of lymphatic glands, 99  
   of pancreas, 112  
   of spleen, 111  
   of suprarenal gland, 112  
 vomiting in, 77  
   amount, 78  
   character of vomit, 78  
   frequency, 79  
   relation to food, 79  
   relief of pain following, 79  
   self-induced, 79  
 Wassermann reaction in, 138  
 water-brash in, 80  
 weight in, 81, 91  
 Dysphagia in carcinoma of the stomach, 440

## E

Eccles and Freer on growth of splenulcus after splenectomy, 809

Echinococcus cysts, *see* Hydatid cysts  
 Eclampsia, jaundice in, 768  
 Egyptian splenomegaly, *see* Splenomegaly, Egyptian  
 Ehrlich's diazo reagent, 752  
 Einhorn's stomach tube, 117  
 Electric cautery, Post, 328  
 Eliason and Ferguson's results in splenectomy for purpura hæmorrhagica, 873, 878  
 Eliason and Wright on adenomata of the stomach, 521  
 Embolism of mesenteric blood-vessels, 95, 1030  
   (*see also* Thrombosis)  
 Emetine in treatment of liver abscess, 741  
 Empyema, anaesthesia in operations for, 50, 51  
   of gall-bladder, in acute cholecystitis, 729  
 Endotracheal anaesthesia, 16  
   and respiratory obstruction, 16  
   apparatus for, 17  
   in abdominal surgery, 16, 55, 57  
   in cranial surgery, 42  
   in operations for relief of acute intestinal obstruction, 58  
   in surgery of the head and neck, 16, 45  
   in thoracic surgery, 51  
   indications for use of, 16  
   inhalation method, 16  
   insufflation method, 16  
   intubation in, 17, 18  
   technique of, 16  
   (*see also* Anaesthesia)  
 Enteritis, diarrhoea secondary to, 85  
 Enteroliths as cause of acute intestinal obstruction, 1018  
 Enterostomy for acute intestinal obstruction, 1005  
   for post-operative obstruction, 1094  
 Enterotome, Devine's, 967  
 Epiplocele, 1043, 1045  
 Epithelioma, radium therapy of, 1610, 1612  
 Eructations in dyspepsia, 79, 80  
 Essential purpura hæmorrhagica, *see* Purpura hæmorrhagica  
 Essential thrombopenia, *see* Purpura hæmorrhagica

Ether anaesthesia, 10

administration of, 11, 12

atropine in pre-operative medication, 12

closed method in, 11

Clover's inhaler, 11

convulsions in, 12

de-etherisation after, 12

effect on peristalsis, 1070

ethyl chloride as a preliminary to, 10, 11

hyperpnoea in, 11

insulin premedication in, 13

intravenous infusion, 12

metabolic changes in, 12

mixtures of chloroform and ether, 14

muscular relaxation obtainable with, 4

nitrous oxide as a preliminary to, 11

open and semi-open methods, 11

overheating in, 12

pre-operative medication in, 12, 13

pulmonary complications in, 12

rate of elimination of, 4

rectal instillation, 12

salivation in, 12

sweating in, 12

toxicity of, 4

tremor in, 12

warm ether-vapour apparatus, 11

with nitrous oxide and oxygen, 12

(see also Anaesthesia)

Ethocaine, see Novocaine

Ethyl chloride anaesthesia, 10

administration of, 10

as preliminary to ether, 10, 11

freezing of skin by, 10, 33

in children, 10

masseteric spasm in, 10

muscular relaxation obtainable with,

4

rate of elimination of, 4

"single-dose" method, 10

toxicity of, 4

(see also Anaesthesia)

Ethylene anaesthesia, 9

muscular relaxation obtainable with, 4

rate of elimination of, 4

toxicity of, 4

(see also Anaesthesia)

Eucaine as local analgesic, 29

Evipan anaesthesia, 14

administration of, 14, 15

as basal narcotic, 21

contra-indications to use of, 15

dangers of, 15

effect on blood-pressure, 15

effect on respiration, 15

evaluation of, 15

in surgery of the neck, 44

sequelae of, 15

(see also Anaesthesia)

Ewald's one hour test meal, 119

stomach tube, 117

Ewing on incidence of sarcoma of the

stomach, 517

Exploratory laparotomy in peptic ulceration,

331

Extra-dural spinal block, 35

(see also Anaesthesia; Analgesia)

## F

Facial fistula, 1238

causes of, 1238

following acute appendicitis, 934

in tuberculous ulcerative colitis, 947

post-operative, 1238

treatment of, 1239, 1240

Faeces, examination of, in jaundice, 771

impaction of, as cause of acute intestinal obstruction, 1018

in dyspepsia, 82

in gastro-intestinal diseases, 85

in stricture of the rectum, 1383

occult blood in, 127

(see also Melena)

Family history in dyspepsia, 70

in gastric and duodenal ulcer, 70

Farquharson on prognosis in peptic

ulceration, 303

Fat necrosis in pancreatitis, 787, 788

Fedoroff's three clamp method of dealing

with the vascular pedicle in

splenectomy, 811

Femoral hernia, see Hernia, femoral

Femoral thrombosis, post-operative, 1257,

1258

Fenwick on distribution of duodenal car-

cinoma, 603, 621

Fever, intermittent hepatic, 760

Fibro-cellular tumour of anus, 1392

Fibroids, see Fibroma

Fibroma, of rectum, 1397

of small intestine, 596

of stomach, 521

- radio-therapy of, 1668  
soft, of anus, 1392  
Fibrous polypus of rectum, 1397  
Field block, Hackenbruch's rhombus, in,  
32  
in abdominal surgery, 55  
novocaine in, 28  
percaine in, 29  
technique of, 32, 33  
(*see also* Analgesia, local)  
Finney's operation of gastro-duodeno-  
stomy for duodenal ulcer, 352  
Finney and Rienhoff on results of total  
gastrectomy, 500  
Finochietto's technique of the Péan-  
Billroth I operation, 526  
anæsthesia, 527  
exploration of abdominal viscera, 531  
incision, 527  
instruments used in, 527  
operative complications, 526  
steps of the operation, 531  
Finsterer's operation for chronic duodenal  
ulcer, 315  
operation of partial gastrectomy, 492  
operation of pyloric exclusion with  
partial gastrectomy for gastric  
and duodenal ulcer, 336  
results in operations for gastric car-  
cinoma, 444  
Finzi on the treatment of menorrhagia by  
X-rays, 1667  
Fistula, ano-rectal, *see* Ano-rectal fistula  
hiliary, *see* Biliary fistula  
duodenal, *see* Duodenal fistula  
fecal, *see* Fæcal fistula  
gastric, *see* Gastric fistula  
in secondary peptic ulceration, 411  
treatment of, 412  
pancreatic, *see* Pancreatic fistula  
rectal, *see* Ano-rectal fistula  
Fistula in ano, *see* Ano-rectal fistula  
Fitz's paper on "appendicitis," 909  
Flatulence in dyspepsia, 79  
Flint on abnormalities of the bile-ducts  
and associated blood-vessels, 628  
Floating spleen, 850  
Forceps, Allis', 327  
Childes', 489  
cross action tetra-cloth, 328  
Desjardins' gall-stone, 665  
Littlewood's, 328  
Morant Baker's, 921  
mosquito, 327  
Moynihan's cholecystectomy, 665  
Moynihan's mesenteric, 327  
Scott Ridout, 332  
Foregger's carbon dioxide absorber, 8  
Foreign bodies in the intestines, 620  
Fossa, iliac, *see* Iliac fossa  
Fouchet's test for bile-pigment, 754  
Fowler's position, 926  
Fractional test meal, *see* Test meals  
Frankan's cases of strangulated hernia,  
1034, 1035  
Fraser's scheme of diet after gastro-  
enterostomy, 389, 390  
Freezing of skin by ethyl chloride, 10  
Friedman on incidence of anastomotic  
ulcer, 404  
Friedrich-Petz clamp, 329, 461  
Friedrichshain Hospital, cases of strangu-  
lated hernia, 1034, 1035
- ## G
- Gabriel's operation for anal fissure, 1302  
Galactose tolerance test, 774  
Gall-bladder, abnormalities of, 628  
absence of, 628  
carcinoma of, 638  
ætiology of, 639  
cholecystectomy in, 678  
cholecystitis in relation to, 639  
diagnosis of, 638  
gall-stones in relation to, 639, 730  
incidence of, 638  
treatment of, 638  
varieties of, 638  
X-ray diagnosis of, 1539  
cholecystography in examination of,  
1527  
cholesterosis of, 653, 654  
diseases of, 627  
constipation in, 84  
duodenal intubation in, 139  
nausea as a symptom in, 77  
pain in, 74, 75  
pre-operative treatment of, 661  
X-ray diagnosis of, 1534  
empyema of, in acute cholecystitis, 729  
enlargement of, in dyspepsia, 112  
in hæmolytic jaundice, 885

Gall-bladder, inflammation of, *see* Cholecystitis  
 injuries of, 627, 635  
 mucocele of, 648  
 operations upon, 663  
   causes of death after, 722  
   cholecystectomy, 678  
   cholecysto-duodenostomy, 692  
   cholecysto-gastrostomy, 689  
   cholecysto-enterostomy, 687  
   cholecysto-jejunostomy, 692  
   cholecystostomy, 672  
   cholecystotomy, 670  
   exploratory laparotomy in, 668  
   general considerations, 664  
   incisions in, 667  
   instruments for, 664  
   isolation of operative field in, 669  
   position of patient upon operating table in, 664  
   post-operative treatment, 718  
   rotation of liver in, 668  
   technique of, 664  
 operative trauma to, 628  
   treatment of, 634  
 perforation of, from disease, 636  
   etiology of, 636  
   incidence of, 636  
   mortality of operations for, 636  
   peritonitis in, 637  
 rudimentary, 628  
 rupture of, 635  
   cholecystectomy in, 678  
   in acute cholecystitis, 729  
   treatment of, 636  
   "strawberry," 653, 654  
 volvulus of, 628  
 X-ray diagnosis in diseases of, 1534, 1539  
 X-ray examination of, 1527

Gall-stones, 723  
   calcium carbonate, 725  
   causation of, 727  
   cholesterin, 723  
   cholesterol, 723  
   faceted, 723  
   location of, 627, 651  
   pigment stone, 725  
   single, 723  
   varieties of, 723  
     (*see also* Gall-stone disease)

Gall-stones colic, 730  
   obstructive jaundice following, 731

Gall-stone disease, 642  
   nente intestinal obstruction due to, 731, 1017  
   acute pancreatitis in, 783  
   age incidence of, 642  
   and cholecystitis, 727, 728  
   and dyspepsia, 72  
   carcinoma of the gall-bladder associated with, 639, 730  
   choice of operation in, 644, 647  
   cholecystectomy in, 678  
   cholecystostomy in, 672  
   cholecystotomy in, 670  
   cholesterol-free diet in, 662  
   clinical features of, 731  
   colic in, 730, 731  
   complications of, 728  
   contra-indications to operation in, 644  
   dislike of fats in, 82  
   exploration of common bile-duct in, 652  
   fistulae due to, 657  
   flatulent dyspepsia in, 731  
   hæmatemesis in, 82  
   ileus in, 1017  
   in relation to carcinoma of the gall-bladder, 639, 730  
   in women, 727  
   incidence of, 642  
   indications for operation in, 642  
   inflammation due to, 728  
   late results of operations for, 720  
   obstructive jaundice in, 730, 731, 758, 759  
   operations for, 647, 663  
   cholecystectomy, 678  
   cholecystostomy, 672  
   cholecystotomy, 670  
   exploration of common bile-duct in, 652  
   late results of, 720  
   mortality of, 646, 719  
   recurrence-rate after, 720  
   pain in, 75, 731  
   pathological complications of, 728  
   position of stones in, 647, 651  
   post-operative treatment, 718  
   recurrence-rate after operations, 720  
   sequelæ of, 760  
   symptoms of, 731  
   typhoid fever in relation to, 727



- with jaundice, 83, 646, 759
  - operations for, 648
  - pre-operative treatment of, 663
- without jaundice, 646, 648
  - operations for, 648
- X-ray diagnosis of, 761, 1535
- Gall-stone ileus, 1017
- Gas-oxygen anaesthesia, 6
- Gastrectomy, anaesthesia in, 55
  - partial, choice of operation in, 467
    - clamps used in, 460
    - diarrhoea after, 85
    - electric cautery in, 462
    - essentials of operations for, 468
    - examination of the stomach in, 465
    - exploration of the abdomen in, 465
    - for cancer of the stomach, 447
    - for *chronic duodenal ulcer without stenosis*, 314
    - for *chronic peptic ulcer*, 307, 309
    - for hour-glass stomach, 282
    - for perforated ulcer, 247
    - for pyloric obstruction, 296
    - general considerations in, 460
    - incisions for, 464
    - methods of performing, 384
      - anterior Polya methods, 468
      - Finochietto's method, 384, 526
      - Finsterer's operation, 492
      - Haberer-Finney operation, 384, 385
      - Lahey's operation, 495
      - Péan-Billroth I operation (Finochietto's technique), 526
      - Polya methods, 386
      - Polya-Balfour operation, 491
      - Polya-Moynihan operation, 468
      - posterior Polya methods, 492
      - Roux's operation in, 386
      - Schoemaker's operation, 384, 385
    - suction apparatus in, 462, 463
    - suture and ligature material used in, 460
- total, 500
  - causes of death following, 502
  - indications for, 501
  - Moynihan's operation, 503
  - operative mortality in, 500
  - pre-operative treatment in, 501
  - technique of, 503
- Gastric acidity, significance of, in gastro-intestinal diseases, 122
  - tests for, 121, 122
- Gastric analysis, histamine and alcohol test meals in, 563
  - in investigation of dyspepsia, 116
  - in ulcer-cancer of the stomach, 562
- Gastric and duodenal ulcer, 177
  - acute, 177, 178
    - diagnosis of, 196
    - perforation in, 226
    - symptoms of, 196
  - chronic, 180
    - aetiology and pathogenesis, 180
      - accessory factors, 187
      - acid factor, 183
      - dietetic factor, 70, 187
      - gastritis theory, 186
      - theory of neurogenic causation, 184
      - toxic factor, 184
    - ulcer diathesis, 181
    - vascular theory, 185
  - anaesthesia in operations for relief of perforation, 58
  - complications of, 220
  - co-existence of gastric and duodenal ulcers, 188
  - diagnosis of, 196
    - blood examination, 138
    - gastric analysis, 116
    - occult blood examination, 127
    - physical signs, 199
    - symptoms, 196
    - test meals, 119
    - Wassermann reaction, 138
    - X-ray examination, 202, 1490
  - diathesis in, 70
  - diet as aetiological factor in, 70, 187
  - dietetic treatment of, 574
    - post-ulcer regime, 576
    - strict ulcer treatment, 575
  - differential diagnosis of, 202
    - from appendicitis, 205
    - from benign growths of the stomach, 218
    - from carcinoma of the colon, 206
    - from carcinoma of the stomach, 211
    - from chronic colitis, 207
    - from chronic duodenal ileus, 215
    - from diverticulitis, 207
    - from duodenal bands, 218
    - from duodenal diverticula, 212

Gastric and duodenal ulcer, differential diagnosis of, from epigastric hernia, 207

from "functional dyspepsia," 203

from gall-bladder diseases, 205

from gastric crises of tabes, 201

from gastritis and duodenitis, 209

from ileo-cæcal tuberculosis, 209

from migraine, 201

from nervous gastric disorders, 203

from pancreatic disease, 209

from reflex dyspepsias, 205

from syphilis of the stomach, 210

from tuberculous ulceration of the stomach, 211

from visceroptosis, 208

gastric analysis in diagnosis of, 116

hæmatemesis in, 82, 198, 578

acute, 579

amenability of lesions to surgical measures, 580

blood-transfusion in, 582

chronic or recurrent, 583

comparative mortality of surgical and medical treatment, 580

diagnosis of, 579

treatment of, general principles in, 578

medical, 584

operative, 586

hunger pain in, 198

in relation to carcinoma of the stomach,

417

(see also Ulcer-cancer of the stomach)

incidence of, 177

indications for operation in, 305

intermittent dyspepsia in, 72

medical treatment of, 573

acute infections during, 575

alkalis in, 574, 577

diet in, 574

duration of, 573

post-ulcer regime, 576

strict ulcer treatment, 575

treatment of focal infection, 575

treatment of tonsillitis, 575

melæna in, 82, 83, 198, 199

mental and physical fatigue as factors in, 187

operations for, 314

excision of gastric ulcers, 340

cautery excision (Balfour's operation), 341

sleeve resection (Riedel-Rodman operation), 346

wedge excision, 342

exploratory laparotomy in, 331

gastro-jejunostomy, 359

anterior gastro-jejunostomy, 377

history of, 359

posterior gastro-jejunostomy, 363

retro-colic anterior or posterior, 380

hæmostasis in, 329

incisions in, 322

instruments for, 326

partial gastrectomy, 384

Finochietto's technique of the

Péan-Billroth I operation, 384,

526

Haberer-Finney operation, 384, 385

Polya types, 386

Roux's method in Y, 386

Schoemaker's operation, 384, 385

Péan-Billroth I operation (Finochietto's technique), 526

pyloric occlusion or exclusion, 333

Bier method, 336, 337

Devine method, 336, 337

Finsterer's operation of pyloric exclusion with partial gastrectomy, 336

Kelling-Mayo method, 334

Wilms method, 334, 337

pyloroplasty—gastro-duodenostomy, 348

Finney's operation, 352

Horsley's operation, 348

Jaboulay's operation, 357

Judd's operation, 352

skin disinfection in, 322

technique of, general considerations in, 322

pain in, 74, 76, 196

pathogenesis of, 181

pathology of, 188

associated gastric and duodenal lesions, 188

macroscopic characteristics, 190

microscopic characteristics, 195

number of lesions, 188

position of ulcers, 190

penetration of the pancreas in, 74

perforation in, 220

age incidence of, 222

- as a cause of peritonitis, 1125
- diagnosis of, 231
- following treatment, 250
- incidence of, 220
- operative treatment of, 236
  - excision of ulcer and suture (pyloroplasty), 246
  - partial gastro-duodenal resection, 247
  - simple suture, 237
  - suture followed by primary gastro-jejunostomy, 244
  - temporary gastrostomy or duodenostomy, 248
- post-operative complications of, 250
- post-operative treatment of, 250
- pre-operative treatment of, 236
- previous history of patients, 222
- prognosis of, 249
- sex incidence of, 221
- signs and symptoms of, 227
- site of ulcer, 224
- size of perforation, 225
- stage of peritonitis in, 230
- stage of prostration in, 228
- stage of reaction in, 230
- sub-acute, 254
- types of, 221
- physical signs in, 199
- post-operative complications of, 391
  - anæmia, 402
  - hæmorrhage, 391
  - retrograde jejuno-gastric intussusception, 399
  - vomiting, 394
- post-operative treatment of, 387
- pre-operative treatment, 319
  - administration of glucose and fluids, 321
  - eradication of septic foci, 321
  - gastric lavage, 320
  - sunlight treatment, 322
- pyloric obstruction in, 283
- secondary, 403
  - acid factor in production of, 407
  - ætiology and pathology of, 403
  - age incidence of, 405
  - complications of, 410
  - condition of bowels in, 409
  - duodenal obstruction in, 413
  - family history in, 405
  - fistula in, 411
  - hæmorrhage in, 409, 411
  - healing of, 406
  - incidence of, 403
  - infective factor in production of, 407
  - pathogenesis of, 407
  - perforation in, 410
  - sex incidence of, 405
  - site of ulceration in, 405
  - symptoms of, 409
  - technical errors at operation as cause of, 407
  - treatment of, 413
  - vomiting in, 409
  - wasting in, 409
  - X-ray findings in, 410
- site-determination of ulcers, 186
- ischæmia theory, 187
- lymphoid follicle theory, 186
- traumatic theory, 187
- sub-acute, 179
- sub-acute perforation in, 254
- symptoms of, 196
- time to operate in, 319
- tobacco smoking as a factor in, 187
- treatment of, 297
  - choice of operation and results, 306
  - dietetic, 574
  - factors influencing choice of treatment, 300
  - indications for operation, 305
  - medical, 573
  - operative, 314
  - post-operative, 387
  - pre-operative, 319
- weight in, 81
- X-ray diagnosis of, 202, 1490
- (see also Duodenal ulcer; Gastric ulcer; Ulcer-cancer of the stomach)
- Gastric crises of tabes, 204
- Gastric fistula, 257, 411, 412
  - external, 257
    - causes of, 257
    - direct and indirect types of, 258
    - operation for, 259
    - signs and symptoms of, 258
    - treatment of, 259
  - internal, 260
    - causes of, 261
    - diagnosis of, 261
    - symptoms of, 261
    - treatment of, 261
    - varieties of, 260

- Gastric juice, examination of, in dyspeptic states, 121
- Gastric lavage, in pre-operative treatment of carcinoma of the stomach, 446
- in pre-operative treatment of gastric ulcer, 320
- in pyloric obstruction, 320
- in treatment of inoperable carcinoma of the stomach, 445
- Gastric phlegmon, *see* Gastritis, acute phlegmonous
- Gastric polyposis, 522
- X-ray diagnosis of, 1502
- Gastric ulcer, appetite and nutrition in, 197
- choice of operation in, 306, 309
- complications of, 220
- dyspeptic symptoms in, 72
- family history in, 70
- hour-glass stomach in, 266
- (*see also* Hour-glass stomach)
- in relation to gastric carcinoma, 417
- (*see also* Ulcer-cancer of the stomach)
- indications for operation in, 305
- "letter-box" ulcers, 312
- operations for, 306
- cholecysto gastrostomy, 312
- excision, 310
- excision of ulcer combined with gastro-jejunostomy, 309
- gastro-duodenal resection for ulcers situated in the pyloric segment, 313
- gastro-jejunostomy, 312
- gastro-jejunostomy without excision of the ulcer, 310
- jejunostomy, 312
- partial gastrectomy, 310
- trans-gastric resection, 345
- operations for complications of, 313
- pain in, 72, 196
- secondary anæmia in, 92
- simulation of, by cirrhosis of liver, 98
- X-ray diagnosis of, 1490
- (*see also* Duodenal ulcer; Gastric and duodenal ulcer; Ulcer-cancer of the stomach)
- Gastritis, 72
- acute phlegmonous, 169
- ætiology of, 169
- circumscribed, 170
- diagnosis of, 171
- diffuse, 170
- incidence of, 169
- pathology of, 170
- primary, 169
- secondary, 169
- symptoms of, 170
- treatment of, 171
- anorexia in, 82
- nausea as a symptom in, 77
- pain in, 74, 76
- Gastro-anastomosis in hour-glass stomach, 274
- Gastro-colic fistula, 261
- (*see also* Gastric fistula)
- Gastro-duodenostomy, for chronic duodenal ulcer, Finney's operation, 352
- Jaboulay's operation, 357
- for chronic duodenal ulcer without stenosis, 314, 315
- in pyloric obstruction, 295
- Gastro-enterostomy, anæsthesia for, 55
- Fraser's post-operative diet chart for, 389, 390
- Gastro-gastrostomy in hour-glass stomach, 274
- Gastro-intestinal diseases, abdominal reflexes in, 103
- anæmia in, 92
- asthenic constitution in, 90
- body-build in, 90
- condition of bowels in, 84
- constipation in, 84
- diagnosis of, 63
- diarrhœa in, 84
- exploratory laparotomy in, 89
- gastric analysis in, 116
- jaundice in, 83, 92
- nutrition in, 91
- occult blood in feces in, 127, 129
- physical examination in, 90
- physique in, 90
- sex factor in, 91
- simulation of, by cardiac and pulmonary diseases, 93
- by coronary thrombosis, 94
- by pericarditis, 95
- by respiratory affections, 95
- skin in, 92
- sthenic constitution in, 90

- stools in, 85  
 syphilis in relation to, 138  
 test meals in diagnosis of, 119  
 visceral pain in, 104  
 weight in, 91  
 X-ray diagnosis of, *see* X-ray diagnosis  
 (*see also* Dyspepsia)
- Gastro-intestinal tract, blood supply of, 1190  
   innervation of, 1190, 1183  
     in relation to paralytic ileus, 1067  
   lymphatic supply of, 1191  
   muscular mechanism of, 1182  
   X-ray examination of, 1483
- Gastro-jejunal ulcer, pain in, 75, 76  
   X-ray diagnosis of, 1503  
 (*see also* Gastric and duodenal ulcer)
- Gastro-jejunostomy, anterior, for gastric and duodenal ulcer, 377  
   diarrhoea after, 85  
   double, in hour-glass stomach, 274  
   following suture of perforated duodenal ulcer, 244  
   in carcinoma of the stomach, 459  
   in duodenal fistula, 264  
   in gastric and duodenal ulcer, 307  
     effects of, 362  
     history of, 359  
     methods, 359  
     technique of, 363  
   in hour-glass stomach, 273  
   in inaccessible ulcer of the stomach, 312  
   in perforated ulcer, 1129  
   in pyloric obstruction, 295, 296  
   posterior, for gastric and duodenal ulcer, 363  
     causes of unsatisfactory results in, 374  
     errors in operative technique in, 376  
     technique of, 363  
   retro-colic anterior or posterior, for gastric and duodenal ulcer, technique of, 380, 382  
   retrograde jejuno-gastric intussusception following, 399  
   secondary peptic ulcer following, 401  
   size of stoma in, 361  
   with excision, cautery destruction, or infolding of the ulcer, for chronic duodenal ulcer without stenosis, 314
- Gastro-photography, 88
- Gastroplasty in hour-glass stomach, 276  
   Heineke-Mikulicz method, 276  
   Kammerer's method, 276
- Gastropotosis, as a cause of pyloric obstruction, 289  
   pain in, 74
- Gastroscope, Wolf-Schindler, 87
- Gastrosocopy, 87, 88
- Gastrostomy, anaesthesia in,  
   in carcinoma of the stomach, 447  
   in perforated gastric ulcer, 248  
   indications for, 447  
   Kader's operation, 454  
   Lepage-Janeway operation, 454  
   Marwedel's operation, 454  
   Stamm's operation, 448  
   Witzel's operation, 452
- Gastrotomy for foreign bodies in the stomach, 176
- Gatewood on hereditary factor in gastric carcinoma, 417  
   results in operative treatment of gastric carcinoma, 444
- Gaucher's disease, 856  
   aetiology of, 856  
   blood changes in, 858  
   blood examination in, 864  
   bone changes in, 858  
   causes of death in, 859  
   course of, 857  
   differential diagnosis of, 859  
   endocrine organs in, 859  
   enlargement of the liver in, 857  
   genetic aspects of, 857  
   incidence of, 861  
   lymphadenopathy in, 859  
   mortality of splenectomy in, 859, 861  
   nervous symptoms in, 859  
   ocular conditions in, 858  
   onset of, 856  
   pathological features of, 860  
   pigmentation of skin in, 858  
   sex incidence of, 857  
   splenectomy in, 861, 862  
   splenomegaly in, 857  
   symptoms of, 856  
   value of splenectomy in, 861, 862
- Geiser on distribution of duodenal cancer, 603, 621
- Gilmonr and Saint on partial gastrectomy for perforated ulcer, 248

- Glandular hyperplasia of small intestine, 597
- Glandular polypus of rectum, 1394
- Globocellularis tumour of small intestine, 613
- Glucose in treatment of operative shock, 23
- Goutre, toxic, anæsthesia in operations for, 45
- care of eyes in, 47
- pre-operative medication, 45
- preparation of patient, 45, 46
- Goodsall's ligature method in treatment of rectal prolapse, 1313
- observations upon ano-rectal fistulæ, 1333
- Gordon-Taylor on anæmia after partial gastrectomy, 402, 403
- results in sleeve resection of gastric ulcer, 346
- Graham on mortality of gastric carcinoma, 416
- Granulomata of liver, 747
- Gravocaine as local analgesic, 28
- Grid-iron incision in appendicectomy, 920
- Grout's X-ray diagnosis of jejunal carcinoma, 611, 621
- Guaiac test for occult blood, 128
- Gum acacia in treatment of operative shock, 23
- Gumma of liver, 747
- as a cause of obstructive jaundice, 763
- of spleen, 849
- Gunshot wounds of abdomen, 620
- Gynæcology, high voltage X-rays in, 1671
- radio-therapy in, 1625

## H

- Haberer-Finney operation of partial gastrectomy, 384, 385
- Haberlein on incidence of gastric carcinoma, 416
- Hackenbruch's rhombus in field blocking, 32
- von Hacker's method of gastro-jejuno-stomy, 359, 360
- Hæmangiomas of small intestine, 597
- Hæmatemesis, causes of, 579
- following gastric operations, 82, 391
- treatment of, 392

- in appendicitis, 82
- in benign growth of the stomach, 524
- in blood dyscrasias, 82
- in cirrhosis of liver, 82
- in diseases of the spleen, 82
- in gall-stone disease, 82
- in gastric and duodenal ulcer, 82, 198, 578
- acute, 579
- amenability of lesions to surgical measures, 580
- blood-transfusion in, 582
- chronic or recurrent, 583
- comparative mortality of surgical and medical treatment, 580
- diagnosis of, 579
- treatment of, general principles, 578
- medical, 584
- operative, 586
- in mesenteric occlusion, 1032
- in portal cirrhosis, 744
- Hæmatoma, localised, post-operative, 1219
- Hæmaturia, essential, due to purpura hæmorrhagica, 879
- Hæmochromatosis, differentiation of, from jaundice, 764
- Hæmoglobin, and bile-pigment formation, 748
- Hæmolytic icterus, *see* Hæmolytic jaundice
- Hæmolytic jaundice, 751, 764, 882
- acquired form of, 883, 888
- ætiology of, 885
- blood changes in, 884, 886, 887
- character of jaundice in, 883
- cholangitis in, 885
- cholecystitis in, 885
- clinical features of, 883
- congenital, 882
- crises in, 884
- definition of, 882
- familial, 84, 765
- formation of pigment gall-stones in, 885
- gall-stone formation in, 885
- in icterus neonatorum, 765
- in pernicious anæmia, 765
- pre-operative blood-transfusion in, 886, 888
- splenectomy for, 885
- mortality and late results of, 887

- splenomegaly in, 883
- symptoms of, 883
- treatment of, 885
- varieties of, 882
- Hæmorrhage following gastric operations, 391
- treatment of, 392
  - (see also Hæmatemesis)
- Hæmorrhagic purpura, *see* Purpura hæmorrhagica
- Hæmorrhoids, 1261
  - external, 1261
    - circumscribed blood extravasation at the anal margin in, 1261
    - ætiology, 1262
    - natural termination of, 1262
    - operative treatment, 1261
    - pathological anatomy, 1262
    - symptomatology, 1262
    - treatment of, 1263
  - dilated peri-anal veins, 1267
    - ætiology, 1267
    - after-treatment, 1269, 1286
    - operative treatment, 1269
    - pathological anatomy, 1268
    - symptomatology, 1267
    - treatment, 1268
  - redundant folds of peri-anal skin, 1264
    - ætiology, 1265
    - pathological anatomy, 1266
    - symptomatology, 1265
    - treatment, 1266
  - thrombotic, 1261
  - varieties of, 1261
  - venous, 1261
- internal, 1269
  - ætiology of, 1278
  - arterial supply of the lower portion of the rectum in relation to, 1271
  - bleeding from, 1282
  - constipation as ætiological factor in, 1279
  - effect of presence upon the tissues of the anal canal, 1279
  - indications for operation in, 1281
  - number which may develop, 1270
  - operative treatment of, 1281
    - after-care, 1286
    - change of dressings, 1288
    - choice of anæsthetic, 1284
    - diet during after-treatment, 1288
    - instruments required, 1284
    - micturition after, 1288
    - Miles's modification of Salmon's ligature operation, 1284
    - pre-operative preparation, 1283
    - selection of operation, 1282
    - Whitehead's operation, 1282
  - pathological anatomy of, 1270
  - position of, in relation to the circumference of the anal canal, 1272
  - post-operative complications, 1289
    - hæmorrhage, 1289
    - narrowing of the lumen of the anal canal, 1290
    - retention of urine, 1290
    - stricture of the anus, 1290
  - post-operative treatment, 1286
  - protrusion of, 1282
  - recurrence of, 1289
  - secondary, 1271, 1272
  - stages in development of, 1276
    - primary stage, 1276
    - intermediate stage, 1277
    - final stage, 1278
  - treatment of, 1281
    - operative, 1281
    - palliative, 1281
    - post-operative, 1286
- Hæmostasis in gastric operations, 329
  - in relation to wound infection, 1216
- Hæmostat, Maingot's, 327
- Haggard on incidence of sarcoma of the stomach, 517
- Hair-ball of stomach, 174, 175
- Handley on the spread of cancer, 1434
- Hanot's hypertrophic biliary cirrhosis, 764
- Harmer on results of radium therapy in cancer of the maxilla, 1591
- Harmer and Cade's results in radium therapy of cancer of the maxilla, 1595
- Harris's total rebreathing apparatus for gas-oxygen anæsthesia, 8
- Hartfall on occurrence of anæmia after gastric operations, 402
- Hartmann's pouch, 632, 633
- Head, anæsthesia in surgery of, 41, 42
- Headache after spinal anæsthesia, 38
- Heart, abdominal symptoms in diseases of, 93
  - diseases of, simulating dyspepsia, 93

Heart, failure, during anaesthesia, 13, 24, 25

massage of, in cardiac failure during anaesthesia, 25

puncture of, in cardiac failure during anaesthesia, 25

Heartburn in dyspepsia, 79, 80

Hebaral sodium as basal narcotic, 20

Heineke-Mikulicz operation for chronic duodenal ulcer, 316

Hepatic arteries, abnormalities of, 629

anatomy of, 629

operative trauma to, 628

Hepatic duct, common, buttonholing of, 630, 631, 635

operations for implantation of hepatic duct into the duodenum, 711

operative trauma to, 630, 631

Hepatic ducts, accessory, 628

operative trauma to, 630

Hepatic failure, *see* Cholemia

Hepatic fever, intermittent, 760

Hepatico-jejunostomy, 718

Hernia, anaesthesia in operations for, 53, 51

diaphragmatic, X-ray diagnosis of, 1501

epiplocele, 1043, 1045

femoral, strangulated, 1034

anaesthesia in, 1050

diagnosis of, 1016

incidence of, 1036

morbid anatomy of, 1037, 1038

mortality of, 1035

operative technique in, 1054

pathology of, 1036

inguinal, strangulated, 1034

anaesthesia in, 1050

diagnosis of, 1046

incidence of, 1036

morbid anatomy of, 1037, 1039

mortality of, 1035

operative technique in, 1054

pathology of, 1036

Maydl's, 1043

Richter's, 981, 1043, 1045

strangulated, 1034

age incidence of, 1037

causes of, 1036

comparison of duration and mortality of, 1035

constricting agent in, 1037

contents of sac in, 1037

definition of, 1037

diagnosis of, 1045

differential diagnosis of, 1046

incidence of the various types of, 1036

morbid anatomy of, 1037

morbid physiology of, 1043

mortality of, 1035

operative treatment of, 1049

anaesthesia in, 1050

herniotomy, 1052

in the various types of hernia, 1054

incisions for, 1054, 1055

of non-viable and doubtful intestine, 1056

by exteriorisation (resection and anastomosis by stages), 1060

by immediate resection and anastomosis, 1057

taxis, 1047

technique of, 1052

pathology of, 1036

post-operative complications, 1053

retention of urine, 1054

post-operative treatment of, 1053

pre-operative treatment of, 1049

anti-shock measures, 1049

blood-transfusion, 1049

gastric and duodenal drainage, 1049

prognosis of, 1035, 1047

"reduction en masse," 1047, 1048

sex incidence, 1037

stages in production of, 1038

symptoms of, 1045

time-factor in, 1035, 1047

treatment, 1047

operative, 1049

post-operative, 1053

pre-operative, 1049

unusual types of, 1042

umbilical, strangulated, 1034

anaesthesia in, 1051

diagnosis of, 1046

incidence of, 1036

morbid anatomy of, 1037

mortality of, 1035

operative technique in, 1055

pathology of, 1036

ventral, following splenectomy, 817

Hernial sacs, contents of, 1037

Herniotomy, local analgesia for, 1050

technique of, 1052



- Herpes zoster as a cause of abdominal pain, 96
- Heuer on results of operations for acute cholecystitis, 721
- Hewer's mouth prop for anaesthesia, 49
- Hexamine as a biliary antiseptic, 662
- Hiccough, persistent, following splenectomy, 817
- High voltage X-rays, *see* X-rays, high voltage
- Histamine test meal in diagnosis of ulcer cancer of the stomach, 563
- Holfelder Field Selector in high voltage X-ray therapy, 1676
- Holocaine as local analgesic, 29
- Horse serum in treatment of purpura hæmorrhagica, 877
- Horseshoe kidney, *see* Kidney, horseshoe
- Horsley on the theory of neurogenic causation in peptic ulcer, 184
- operation of "physiologic" pyloroplasty for duodenal ulcer, 316
- technique of, 348
- Hoskin and Sewell's bed-lifter, 926
- Hour-glass stomach, 266
- ætiology of, 266
- age incidence of, 266
- appetite in, 272
- diagnosis of, 268
- incidence of, 267
- loss of weight in, 81, 270
- operations for, 272
- double gastro-jejunostomy, 274
- gastro-jejunostomy, 273
- gastro-gastrostomy (gastro-anastomosis), 274
- gastroplasty (Heineke-Mikulicz method), 276
- gastroplasty (Kammerer's operation), 276
- partial gastrectomy, 282
- sleeve resection, 276
- Walton's operation, 280
- pain in, 76, 270
- pathology of, 267
- pre-operative regime in, 272
- sex incidence of, 266
- signs and symptoms of, 268
- treatment of, 272
- types of, 268, 271
- vomiting in, 270
- Howard on acute glossy œdema of pancreas, 786
- Hunger pain, in cholecystitis, 77
- in chronic appendicitis, 77
- in duodenal ulcer, 77
- in duodenitis, 77
- in dyspepsia, 77
- in gastric carcinoma, 77
- in hyperchlorhydria, 77
- in peptic ulceration, 198
- Hurst's method of treating chronic cholecystitis, 662
- on healing of secondary peptic ulcers, 406
- on incidence of secondary peptic ulcer, 404
- post-ulcer regime, 576
- strict ulcer treatment, 575
- Hurst and Stewart on secondary peptic ulcer, 413
- Hydatid cysts, of liver, 745
- as a cause of obstructive jaundice, 764
- of spleen, 846
- Hydrocele, anaesthesia in operations for, 53
- Hydronephrosis, X-ray diagnosis of, 1544
- Hyperchlorhydria, "hunger pain" in, 77
- significance of, in gastro-intestinal diseases, 122
- Hypernephroma, X-ray diagnosis of, 1549
- Hysterectomy, anaesthesia in, 54

## I

- Iccal in treatment of operative shock, 24
- Icterus, hæmolytic, *see* Hæmolytic jaundice
- Icterus index, 754
- Icterus neonatorum, 765
- Ileo-cæcal hyperplastic disease, 917
- Ileum, benign tumours of, 593
- carcinoma of, 608
- diagnosis, 609
- pathology, 608
- treatment, 611
- globocellularis or carcinoid tumour of, 613
- X-ray examination of, 1613
- Ileus, 1181
- "active," 1191, 1197
- feeding in, 1200

- Ileus, "active," Fowler position in, 1199  
     management of, 1198  
     promotion of, 1198  
     purgation in, 1200  
     radiant heat in, 1199  
     rectal saline in, 1198  
     sedatives in, 1199  
     starvation in, 1198  
 adhesive, 1073  
 causes of, 1197  
 chronic, 1204  
     circulatory factor in, 1205  
 chronic inhibitory impulses in, 1204  
 clinical consideration of, 1197  
 definition of, 1182  
 differentiation from intestinal obstruction, 1181  
 duodenal, *see* Duodenal ileus  
 from peritonitis, 1109, 1120, 1203  
 gall-stone, 1017  
 in acute obstruction, 1203  
 intermittent, 1204  
 mechanism of peristalsis in relation to, 1184  
 muscular mechanism of the alimentary tract in relation to, 1182  
 paralytic, 1066, 1194, 1200  
     etiology of, 1070  
     anatomy of peristalsis and, 1067  
     causes of, 1072  
     complicating diffuse peritonitis, 1109  
     prevention and treatment, 1120  
 following operation for acute appendicitis, 935  
 management of, 1085  
 morbid anatomy of, 1072  
 pathology of, 1067  
 physiology of peristalsis and, 1069  
 post-operative, 1076  
     diagnosis of, 1076  
     differentiation from adhesive obstruction, 1078  
     duration and course of, 1077  
     symptoms of, 1076  
 prevention of, 1079, 1200, 1201  
 treatment of, 1085, 1201  
     drugs in, 1086  
     operative, 1094  
     spinal anaesthesia in, 1092  
     splanchnic anaesthesia in, 1093  
 physiological consideration of, 1181  
 primary, 1182  
 right coloptosis and, 1208  
 significance of, in gastro-intestinal diseases, 1211  
 visceroptosis and, 1208  
 (*see also* Intestinal obstruction)  
 Iliac abscess, 1145  
     left iliac abscess, 1147  
     right iliac abscess, 1145  
         diagnosis of, 1146  
         differential diagnosis of, 1147  
         symptoms of, 1146  
         treatment of, 1148  
 Iliac fossa, actinomycosis of, 937  
 Incision, abdominal, in relation to post-operative eventration, 1225  
     Battle's, in appendicectomy, 920  
     grid-iron, in appendicectomy, 920  
     in appendicectomy, 920  
     in gastric surgery, 464  
     in operations for peptic ulcer, 322  
     in splenectomy, 806  
     in surgery of the gall-bladder and bile-ducts, 667  
     Kocher's, 668  
     post-operative rupture of, 1222, 1225  
 Indigestion, *see* Dyspepsia  
 Infantile pyloric stenosis, *see* Pyloric stenosis, infantile  
 Infiltration analgesia, by novocaine, 28  
     by percaïne, 29  
     technique of, 31, 32  
 Inguinal hernia, *see* Hernia, inguinal  
 Instruments, for examination of the stomach, 87  
     for gall-bladder surgery, 664  
     for gastric surgery, 326  
 Insufflation of rectum, in treatment of ulcerative colitis, 945  
 Insulin premedication in anaesthesia, 13  
 Intercostal muscles, abdominal symptoms in fibrositis of, 96  
 Intermittent hepatic fever, 760  
 Intestinal obstruction, acute, 973  
     abdominal auscultation in, 993  
     adhesive, 1008, 1073  
     diagnosis of, 1010  
     pathology of, 1008  
     recurrence of, 1010  
     signs and symptoms of, 1010  
     treatment of, 1010  
     etiology and classification of, 976  
     by obturation, 1017

- causes of, 978, 996
  - classification of types and causes of, 978
  - clinical features of, 990
  - congenital, 1015
    - direct causes of, 1015
    - indirect causes of, 1016
  - constipation in, 992
  - coprostasis in, 992
  - danger of sudden decompression in, 987, 989
  - diagnosis of, 989
  - diagnosis of the cause of obstruction, 996
    - discovery of acute obstruction, 990
    - examination and physical signs, 993
    - recognition of the type and level of obstruction, 994
  - X-rays in, 994, 1512, 1514
  - distension in, 985
  - examination and physical signs in, 993
  - exploratory laparotomy in, 1004
  - following appendicectomy, 1073
  - following splenectomy, 817
  - from carcinoma of the colon, 960
  - from enteroliths, 1018
  - from faecal impaction, 1018
  - from gall-stones, 731, 1017
  - from intussusception, 1019
  - from mesenteric embolism and thrombosis, 1030
  - from strangulated hernia, 1034
  - from stricture of the rectum, 1388
  - from volvulus, 1026
  - general consideration of, 973
  - high small-gut obstruction, morbid physiology of, 982
    - treatment of, 997
  - internal strangulation, by Meckel's diverticulum, 1012, 1013
    - by omental bands, 1012
    - by peritoneal bands, 1012
    - by visceral bands, 1012
    - diagnosis of, 1014
    - mechanism of, 1014
    - pathology of, 1011
    - through internal apertures, 1013
    - treatment of, 1015
  - large-gut obstruction, morbid physiology of, 987
    - treatment of, 1000
  - low small-gut obstruction, morbid physiology of, 984
    - treatment of, 999
  - morbid anatomy of, 979
  - morbid physiology of, 981
  - mortality of, 974
  - operative treatment of, 1002
    - after-treatment, 1007
    - anaesthesia, 1003
    - cæcostomy, 1007
    - enterostomy, 1005
    - preparation for, 1002
    - technique of, 1004
  - pain in, 990
  - pathology of, 979
  - post-operative treatment of, 1007
  - pre-operative treatment of, 1002
  - shock in, 988, 1001
  - simple obstruction, 976
    - blood changes in, 983
    - dehydration in, 982
    - morbid physiology of, 982
    - renal failure in, 984
    - symptoms of, 994
  - strangulation obstruction, 977
    - morbid physiology of, 988
    - symptoms of, 994
    - treatment of, 1001
  - symptoms of, 990
  - toxæmia in, 975, 986, 989
  - treatment of, 997
    - of high small-gut obstruction, 997
    - of internal strangulation, 1001
    - of large-gut obstruction, 1000
    - of low small-gut obstruction, 999
    - operative, 1002
    - types of, 978, 1008
    - vomiting in, 991
  - X-ray diagnosis of, 994
- adhesive, *see* Post-operative, *below*
- anaesthesia in operations for relief of, 58
- post-operative, 1066, 1073
- adhesive, 1073
  - ætiology of, 1073
  - diagnosis of, 1077
  - differential diagnosis from paralytic ileus, 1078
  - following appendicectomy, 1073
  - following splenectomy, 817
  - mechanism of, 1074
  - morbid anatomy of, 1074
  - simple obstruction, 1074

- Intestinal obstruction, post-operative,  
 adhesive, strangulation in, 1075  
 symptoms of, 1077  
 aetiology of, 1073  
 clinical types of, 1076  
 diagnosis of, 1076  
 differential diagnosis of, 1078  
 management of, 1085  
 mild inhibition of peristalsis in, 1076  
 prevention of, 1079  
   post operative prophylaxis, 1084  
   pre-operative prophylaxis, 1079  
   prophylaxis at operation, 1080  
   prophylaxis in appendix operations, 1083  
*symptoms of, 1076*  
 treatment of, 1085  
   by acetyl-choline, 1083  
   by drugs, 1086  
   by enemata, 1092  
   by hypertonic saline, 1091  
   by morphine, 1087  
   by pituitrin and eserine, 1086  
   by spinal anaesthesia, 1092  
   by splanchnic anaesthesia, 1093  
   operative, 1094  
   summary of, 1095  
 X-ray diagnosis of, 994, 1512, 1514  
*(see also Ileus)*
- Intestine, small, 593, 615  
 benign tumours of, 593  
   adenomata, 595  
   diagnosis of, 597  
   fibromata, 596  
   haemangiomas, 597  
   incidence of, 593  
   intussusception due to, 597, 599, 600  
   lipomata, 597  
   myomata, 596  
   pathology of, 594  
   polypi, 595  
   treatment of, 600  
   types of, 593  
 carcinoid tumour of, 613  
 carcinoma of, 602  
 foreign bodies in, 620  
 glandular hyperplasia of, 597  
 globocellularis or carcinoid tumour of, 613  
 injuries of, 615  
   closed injuries, 615  
   diagnosis of, 615  
   due to foreign bodies, 620  
   extent of tear in, 618  
   gunshot wounds, 620  
   involvement of mesentery in, 619  
   open injuries, 619  
   pneumatic rupture, 615  
   rupture of duodenum, 619  
   shock in, 617  
   symptoms of, 615  
   time-factor in, 619  
   treatment of, 617  
   sarcoma of, 613  
   tumours of, 593  
   volvulus of, 1029  
   X-ray examination of, 1513
- Intestines, innervation of, in relation to  
 paralytic ileus, 1067
- Intra-arterial local analgesia, 34
- Intra-bronchial anaesthesia in thoracic  
 surgery, 51
- Intra-neural block, 33
- Intravenous anaesthesia with evipan, 14,  
 15
- Intravenous local analgesia, 34
- Intubation, duodenal, *see* Duodenal in-  
 tubation
- Intubation in endotracheal anaesthesia, 17,  
 18
- Intussusception, acute, 1019  
 anaesthesia in operations for, 59  
 diagnosis of, 1022  
 differential diagnosis of, 1023  
 in infants, 1019, 1022  
 incidence of, 1019  
 Maunsell's operation for, 1025  
 mechanism of, 1019  
 morbid anatomy of, 1020  
 non-operative treatment of, 1026  
 operative treatment of, 1023  
 mortality in, 1022, 1023  
 procedure in difficult cases, 1024  
 technique of, 1024  
 post-operative treatment of, 1025  
 symptoms of, 1022  
 treatment of, 1023  
 varieties of, 1021
- from benign tumours of small in-  
 testine, 597, 599, 600
- retrograde jejuno-gastric, following  
 gastric operations, 399
- Iron in treatment of purpura haemorrhag-  
 ica, 877

Irradiation therapy, *see* Radio-therapy ;  
Radium therapy ; X-rays, high  
voltage

Ischio-rectal fistula, *see* Ano-rectal fistula

Islets of Langerhans, adenomata of, 789  
carcinoma of, 789

J

Jaboulay's operation of gastro-duodeno-  
stomy for duodenal ulcer, 357

von Jaksch's disease, 832

Jaundice, 748  
acholuric, *see* Hæmolytic jaundice  
bile-pigment formation and, 748  
blood in, 748, 754  
blood analysis in, 752, 754  
blood-coagulation time in, 775, 779  
catarrhal, *see* Catarrhal jaundice  
cholecystography in, 775  
classifications of, 750, 754  
clinical features of, 755  
combined, 754  
definition of, 748  
dietetic treatment of, 776  
differential diagnosis of, 770  
dissociated, 755  
duodenal intubation in, 140, 771  
epidemic, 84, 768  
Fouchet's test in, 754  
hæmolytic, *see* Hæmolytic jaundice  
icterus index in, 754  
in carcinoma of the common bile-duct,  
83  
in carcinoma of the pancreas, 83  
in carcinoma of the stomach, 440  
in cholæmia, 772  
in congenital syphilis, 769  
in dyspepsia, 92  
in eclampsia of pregnancy, 768  
in fevers, 84  
in gall-stone disease, 83, 646, 648, 663  
in gastro-intestinal diseases, 83, 92  
in infantile pyloric stenosis, 149  
in injuries to the bile-ducts, 635, 636  
in pancreatitis, 83  
in septic conditions, 81  
in syphilis, 84, 763, 768, 769  
infective, 766  
in specific diseases, 768  
irritation of the skin in, 779

latent, 753, 754  
liver function tests in, 773  
galactose tolerance test, 774  
levulose tolerance test, 774  
McNee's classification of, 750, 755  
medical treatment of, 775  
care of the bowels, 777  
cholagogues and biliary antiseptics,  
778  
diet, 776  
drugs, 778  
duodenal drainage, 778  
obstructive, 755  
causes of, 758  
cholecystography in, 761  
Courvoisier's law, 730  
due to amœbic abscess of liver, 764  
due to carcinomatosis of the peri-  
toneum, 763  
due to chronic perihepatitis, 763  
due to cirrhosis of the liver, 764  
due to gall-stones, 730, 731, 759  
due to gummatous disease of liver,  
763  
due to hydatid cyst of liver, 764  
due to malignant disease, 762  
due to peritoneal adhesions, 763  
due to secondary malignant deposits,  
763  
medical treatment of, 775  
symptoms of, 755  
X-ray examination in, 761  
obstructive hepatic, 750  
pathology of, 750  
physical examination in, 770  
pre-operative measures in patients with,  
779  
regurgitation, 754  
renal threshold for bilirubin, 753  
retention, 754  
Rich's classification of, 754  
salvarsan, 769  
skin irritation in, 779  
spirochætal, 768  
surgical aspects of, 779  
toxic and infective hepatic, 84, 751, 766  
ætiology of, 766  
catarrhal, 766  
due to chemical poisons, 769  
due to chloroform inhalation, 769  
due to heavy metals, 769  
medical treatment of, 779

Jaundice, toxic and infective hepatic,  
     pathology of, 766  
     symptoms of, 769  
 Van den Bergh reaction in, 750  
     indirect, 750, 751  
     interpretation of results, 753  
     technique of, 752  
     varieties of, 750  
 Jaw, radium therapy in cancer of,  
     1592  
 Jefferson on incidence of carcinoma of  
     small intestine, 603, 621  
 Jejuno-gastric intussusception, 399, 400  
     acute retrograde, 400  
     chronic, 401  
     treatment of, 402  
 Jejuno-ileum, benign tumours of, 598  
     carcinoma of, 608  
     diagnosis of, 609  
     pathology of, 608  
     treatment of, 611  
 Jejunostomy for duodenal fistula, 264  
     for gastric carcinoma, 458  
     for inaccessible ulcers of the stomach,  
     312  
 Jejunum, benign tumours of, 593  
     carcinoma of, 608  
     diagnosis of, 609  
     pathology of, 608  
     treatment of, 611  
     injuries of, 615  
     ulcer of, 403  
     (*see also* Gastric and duodenal ulcer,  
     secondary)  
 X-ray examination of, 1513  
 Johnson on incidence of carcinoma of  
     small intestine, 603, 621  
 Joll on pyloric gastric ulcer, 307  
 Jones' (Howard) hypoharic percaine  
     method of spinal anaesthesia,  
     36  
     spinal needle and stilette, 37  
 Judd on anastomotic ulcer following  
     operation for gastric carcinoma,  
     405  
     on carcinoma of the colon, 956  
     operation of pyloroplasty for duodenal  
     ulcer, 317, 352  
 Judd and Phillips' cases of perforation of  
     the gall-bladder, 656  
     on results of operations for acute  
     cholecystitis, 721

Judd and Priestley on recurrence of gall,  
     stones after operative treatment-  
     720  
     results in cholecystectomy for chronic  
     cholecystitis, 719  
 Junker's bottle, 14  
     inhaler for chloroform anaesthesia, 14

## K

Kader's method of gastrostomy, 454  
 Kahn test, value of, in abdominal disease,  
     138  
 Kammerer's operation of gastropasty in  
     hour-glass stomach, 276  
 Kappis' method in splanchnic block, 56  
 Kelling-Mayo method of pyloric occlusion  
     for gastric and duodenal ulcer,  
     334  
 Kellogg on technique of Coffey's modifica-  
     tion of Sullivan's operation of  
     end-to-side choledocho-duodeno-  
     stomy, 712  
 Kerocain, *see* Novocaine  
 Ketosis as a cause of vomiting in children,  
     98  
 Keynes on gastrostomy in perforated  
     gastric ulcer, 249  
     on use of radium in carcinoma of breast,  
     1601  
 Kidney, amyloid degeneration of, from  
     stricture of the rectum, 1389  
     calculi in, *see* Renal calculus  
     congenital anomalies of, X-ray diag-  
     nosis of, 1546  
     congenital polycystic, X-ray diagnosis  
     of, 1548  
     disease, in relation to dyspepsia, 98  
     enlargement of, in dyspepsia, 111  
     function tests, 131  
     horseshoe, X-ray diagnosis of, 1546  
     mobile, as a cause of pyloric obstruc-  
     tion, 289, 294  
     X-ray diagnosis in diseases of, 1544  
     X-ray examination of, 1540  
     (*see also* Renal colic; Urinary tract)  
 Kifa skin-clips, 326, 489  
 King on benign tumours of the intestines,  
     593  
 Knott on the spleen in Gaucher's disease,  
     853

- report on blood examination in a case of  
Gaucher's disease, 864
- Kny-Scheerer operating table, 46
- Kocher's incision in surgery of the gall-  
bladder, 968  
method of mobilising the duodenum,  
358, 700, 701  
operation of trans-duodenal chole-  
dochoostomy, 704
- Konjetzny on association of gastritis  
with gastric and duodenal ulcer,  
407  
on chronic gastritis and gastric car-  
cinoma, 417  
on duodenal involvement in cancer of  
the pylorus, 603
- Kupffer cells of the liver, 748, 749
- L
- Labat's syringe and needle for local  
analgesia, 29
- Lævulose tolerance test, 774
- Lahey's operation of partial gastrectomy,  
495
- Lake on the blood count after partial  
gastrectomy, 403
- Lang's frame, 408
- Langerhans, islets of, *see* Islets of Langer-  
hans
- Laparotomy, exploratory, in gastro-  
intestinal diseases, 89
- Larynx, anaesthesia in surgery of, 44, 45  
radium therapy in cancer of, 1596
- "Leather-bottle" stomach, 422  
X-ray diagnosis of, 1497
- Leitch's investigations upon cancer of the  
rectum, 1434
- Lenhartz diet in post-operative treatment  
of peptic ulcer, 388, 390
- Lepage-Janeway method of gastrostomy,  
454
- "Letter-box" ulcers of the stomach, 312
- Leucoplakia, radio-therapy in, 1670
- Leukæmias, radio-therapy in, 821
- Levi on alkalosis in infantile pyloric  
stenosis, 148
- Lewisohn on incidence of anastomotic  
ulcer, 404
- Ligatures for abdominal surgery, 460  
in relation to wound infection, 1217
- Linitis plastica, *see* "Leather-bottle"  
stomach
- Lip, radium therapy in cancer of, 1589
- Lipoma of anus, 1393  
of colon, 952  
of small intestine, 597  
of stomach, 522
- Liston sounds for dilating the common  
bile-duct, 666
- Littlewood's forceps, 328
- Liver, abscess of, 740  
after suture of perforated duodenal  
ulcer, 743  
choleangitic, 740  
portal pyæmic, 740, 744  
rupture of, 742, 743  
septicaemia, 740  
staphylococcal, 743  
tropical, 740  
complications, 742  
diagnosis, 740  
mortality of, 743  
open operation in, 742  
prognosis in, 743  
symptoms of, 740  
treatment, 741  
varieties of, 740
- actinomycosis of, 747  
aspiration of, 741  
carcinoma of, 745  
cirrhosis of, 744  
hæmatemesis in, 82  
in hæmolytic jaundice, 885  
in relation to dyspepsia, 98  
in splenic anaemia, 868  
jaundice in, 764  
simulating gastric ulcer, 98  
splenomegalic, 855  
surgical aspects of, 744
- Talma-Morison operation in, 744  
treatment of ascites due to, 744  
treatment of hæmatemesis from, 744
- effect of chloroform anaesthesia upon, 13
- efficiency tests, 773
- enlargement of, in dyspepsia, 110
- failure, *see* Chokemia
- formation of bile-pigment, 748
- function tests, 773  
ammonia-coefficient of urine, 773  
galactose tolerance test, 774  
lævulose tolerance test, 774  
urinary nitrogen-coefficient, 773

- Myoma, of colon, 932  
 of small intestine, 596  
 of stomach, 520  
 Myositis, suppurative, secondary hæmorrhage due to, 1220  
 Myxoma of rectum, 1406  
 Myxomatous polypus of rectum, 1406

## N

- Nagel on duodenal involvement in cancer of the pylorus, 603, 621  
 Narath's modification of the Talma-Morison operation for cirrhosis of the liver, 744  
 Narcosis, *see* Basal narcosis  
 Narcylen anaesthesia, 9  
 Nausea, and vomiting, after spinal anaesthesia, 38  
   in appendicitis, 77  
   in carcinoma of the stomach, 438  
   in dyspepsia, 77  
   in gall-bladder disease, 77  
   in gastric carcinoma, 77  
   in gastritis, 77  
 Neck, anaesthesia in surgery of, 44  
   endotracheal anaesthesia in surgery of, 16  
 Needle holder, Bozemann's, 327  
 Nembutal as basal narcotic, 20  
 Nêocaine, *see* Novocaine  
 Neothesiin as local analgesic, 29  
 Nerve block, 33  
   by novocaine, 28  
   (*see also* Analgesia, local)  
 Nervous disorders in relation to dyspepsia, 203  
 Nervous system, abdominal pain in diseases of, 97  
 New's results in radium therapy of cancer of the maxilla, 1594  
 Nielsen on prognosis of medical treatment of peptic ulcer, 303  
 Nitrous oxide anaesthesia, 3, 5  
   action of, 3  
   administration, 5  
   as preliminary to ether, 11  
   combined with local analgesia, 27  
   muscular relaxation obtainable with, 4  
   preparation of, 5  
   rate of elimination, 4  
   storage, 5  
   toxicity of, 4  
 Nitrous oxide and air anaesthesia, 5, 6  
   administration, 5  
   advantages of, 6  
   apparatus, 5  
   contra-indications to use of, 5  
 Nitrons oxide and oxygen anaesthesia, 6  
   administration, 6  
   advantages of, 6  
   apparatus, 6  
   closed-circuit apparatus, 6  
   continuous-flow apparatus, 6  
   in abdominal surgery, 53, 58, 59  
   in cranial surgery, 42  
   in operations upon the abdominal wall, 53  
   in surgery of the neck, 44  
   in thoracic surgery, 48  
   intermittent-flow apparatus, 6  
   premedication in, 6  
   with ether vapour, 12  
 Novocaine as local analgesic, 28  
   in abdominal surgery, 56, 57  
   in cranial surgery, 42  
   infiltration and field blocking by, 28  
   nerve blocking by, 28  
 Novtox, 28  
 Nutrition, degree of, in dyspepsia, 91  
   in gastro-intestinal disorders, 91

## O

- Occult blood, *see* Blood, occult,  
 Ochsner-Sherren (delayed) treatment of appendicitis, 924  
   care of bowels, 927  
   charts for, 926  
   diet in, 927  
   drugs in, 927  
   observation of patient in, 928  
   selection of cases for, 925  
   technique of, 926  
 (Esophagus, loss of weight in obstruction of, 81  
   varices of, in splenic anaemia, 867  
 Ogilvie, on Finsterer's operation of partial gastrectomy, 492, 493  
   on gastro-jejunostomy for peptic ulcer, 362  
   on incidence of anastomotic ulcer, 404



- on partial gastrectomy for duodenal ulcer, 309
- on the blood count after partial gastrectomy, 403
- on the technique of Finsterer's operation for gastric and duodenal ulcer, 338
- Olive oil, emulsified, in treatment of toxæmia, 1124
- Omentopexy in portal cirrhosis, 744
  - in splenic anæmia, 870
  - in splenomegalic cirrhosis, 855, 856, 857
- Omentum, functions of, 1103
  - strangulated, 1043, 1045
- Operation wounds, infected, *see* Abdominal wounds, infected
- Orth on pathology of duodenal carcinoma, 604, 621
- Ovary, anæsthesia in surgery of, 54
  - differential diagnosis of appendicitis and apoplectic, 919
  - radio-therapy in carcinoma of, 1664
- Oxygen insufflation of rectum in treatment of ulcerative colitis, 945
- Oxyuria *vermicularis* in the appendix, 911

## P

- Pain, abdominal, in affections of the central nervous system, 97
  - in affections of the spine, 97
  - in cardiac and pulmonary diseases, 93
  - in chronic cough, 96
  - in early phthisis, 96
  - in fibrositis of intercostal muscles, 96
  - in herpes zoster, 96
  - in Pott's disease, 97
  - in tabes dorsalis, 97
  - in thoracic conditions, 96
- constant, in gastric disease, 76
- "hunger pain," *see* "Hunger pain"
- in abdominal disease, 74
- in acute intestinal obstruction, 990
- in appendicitis, 75, 76
- in atonic dilatation of the stomach, 77
- in carcinoma of the stomach, 75, 76, 437
- in cholecystitis, 76
- in chronic colitis, 75
- in duodenal perforation, 76

- in duodenal ulcer, 74
- in dyspepsia, 74
  - accompanied by collapse, 76
  - associated with pyrexia, 76
  - character of, 75
  - constant, 76
  - "hunger pain," 77
  - position of, 74
  - radiation of, 74
- in gall-bladder disease, 74
- in gall-stone colic, 75, 731
- in gastric and duodenal ulcer, 74, 196
- in gastric perforation, 76
- in gastritis, 76
- in gastro-jejunal ulcer, 76
- in hæmorrhagic pancreatitis, 76
- in hour-glass stomach, 76, 270
- in pancreatic disease, 75
- in peritonitis, 1111
- in pyloric obstruction, 291
- in pyloric stenosis, 76
- in rupture of the spleen, 894
- in secondary peptic ulcer, 409
- in visceroptosis, 75
- post-operative, 1253
- visceral, in abdominal disease, 104
- Palate, radium therapy in epithelioma of, 1590
- Pancreas, abscess of, *see* Pancreatic abscess
  - acute glossy œdema of, 784
  - benign tumours of, 789
  - calculi of, 788
  - carcinoma of, 788
    - and pyloric obstruction, 293
    - as a cause of obstructive jaundice, 762
  - cholecysto-enterostomy in, 688
  - jaundice in, 83, 730, 762
- cysts of, 787
- enlargement of, in dyspepsia, 112
- fistula of, 788
- inflammation of, *see* Pancreatitis
- injuries of, operative, 783
- neoplasms of, 788
- pain in diseases of, 75
- penetration of, in peptic ulceration, 74
- pseudo-cysts of, 787
- Pancreatic abscess, 784
  - drainage of, 787
- Pancreatic fistula, 788
- Pancreatic insufficiency, diarrhœa in, 84

## Pancreatitis, 783

acute, aetiology of, 783

Cullen's sign in, 786

diagnosis of, 786

Grey Turner's sign in, 786

in gall-stone disease, 783

Loewi's test in, 786

pancreatic drainage in, 786

produced at operation, 783

symptoms of, 786

treatment of, 786

varieties of, 783

cholecysto-enterostomy in, 688

fat necrosis in, 787, 788

gangrenous, 783

haemorrhagic, 783

cholecystostomy in, 672

pain in, 76

jaundice in, 83

suppurative, 784

Pantocain as local analgesic, 29

Papilliferous carcinoma of rectum, 1409

Papilloma of anus, 1390

Paraldehyde as basal narcotic, 19

Paralyses after spinal anaesthesia, 38

Paralytic ileus, *see* Ileus, paralytic

Para-neural block, 33

Para-rectal suppuration, from stricture of the rectum, 1388

Parathormone in treatment of purpura haemorrhagica, 877

Paravertebral and splanchnic block in abdominal operations, 56, 57

Paris technique in radio-therapy of carcinoma of the cervix, 1653

Parotitis, acute suppurative, 1242

aetiology of, 1242

post-operative, 1242

predisposing factors, 1242

treatment of, 1243

Paterson on anastomotic ulcer, 403, 401

on anterior gastro-jejunostomy, 378

on gastro-jejunostomy for peptic ulcer, 362

on results of total gastrectomy, 500

results in gastro-jejunostomy, 378, 379

Paul's tubes, drainage by, 919, 950

Paul-Mikulicz operation, 963, 966

Payr clamp, 328, 461, 462

Péan-Billroth I operation of partial gastrectomy (Finochietto's technique), 526

anaesthesia for, 527

complications of, 526

exploration of abdominal viscera in, 531

incision in, 527

instruments for, 527

technique, by stages, 531

Pectenotomy for anal fissure, 1300

Pelvic abscess, with reference to appendicitis, 930

Pelvic peritoneal abscess, 930, 1150

causes of, 1150

diagnosis of, 1151

evacuation of, 1151

by rectal route, 1152

by vaginal route, 1153

symptoms of, 1151

treatment of, 1151

Pemberton on ligature of the coronary vein in splenectomy, 870

on omentopexy supplementary to splenectomy, 870

Penis, radium therapy in epithelioma of, 1612

Peptic ulcer, *see* Gastric and duodenal ulcer

Peptone injections, in treatment of purpura haemorrhagica, 877

Percaïne as local analgesic, 29

in abdominal surgery, 55

spinal analgesia by, 35, 36

Peri-anal skin, redundant folds of, 1264

Peri-anal veins, dilatation of, 1267

Pericarditis simulating abdominal disease, 95

Peri-colic abscess in tuberculous ulcerative colitis, 947

Perigastric adhesions, 255

Perigastric inflammation, and pyloric obstruction, 287, 291

Perihepatitis, chronic, and obstructive jaundice, 763

Perineum, anaesthesia in surgery of, 59, 60

Peristalsis, anatomy of, 1067

biochemical factors in, 1070

derangement of, 1187

effect of chloroform and ether anaesthesia upon, 1070

in relation to ileus, 1184

physiology of, in relation to paralytic ileus, 1067, 1069

post-operative weakening of, 1076

- Peritoneal abscess, 1142  
 anatomy of the sub-diaphragmatic spaces in relation to, 1154  
 diagnosis of, 1143  
*iliac, see Iliac abscess*  
 pelvic, *see* Pelvic peritoneal abscess  
 sites of, 1142  
 subphrenic, *see* Subphrenic abscess  
 symptoms of, 1143  
 treatment of, 1143
- Peritoneal spaces, 1099, 1101
- Peritoneum, abscess in, *see* Peritoneal abscess
- anatomy and functions of, 1098  
 carcinomatosis of, and obstructive jaundice, 763  
 cleansing of, in treatment of peritonitis, 1118  
 compartments of, 1099  
 functions of, 1102  
 lymph drainage, 1103  
 nerve supply, 1103
- Peritonitis, 1097  
*acute diffuse, 1104*  
 abscess in, *see* Peritoneal abscess  
 appendicular, 932, 1131  
 atypical cases of, 1113  
 bacteriology of, 1106, 1109  
 caused by acute appendicitis, 1131  
 caused by perforated gastro-duodenal ulcer, 1125  
 diagnosis, 1126  
 question of primary gastro-jejunostomy in, 1129  
 treatment, 1128  
 classification of, 1104  
 course of, 1111  
 diagnosis of, 1110  
 differential diagnosis of, 1114  
 fulminating type of, 1113  
 localising type of, 1113  
*morbid anatomy of, 1107*  
 paralytic ileus complicating, 1109  
 prevention and treatment of, 1120  
 pathology of, 1106  
 physical signs in, 1112  
 post-operative, 1132  
 aetiology and prophylaxis, 1132  
 diagnosis, 1134  
 treatment, 1135  
 prevention of, 1115
- secondary, 1101  
 aetiology, 1104  
 incidence of causes of, 1105  
 special varieties of, 1125  
 symptoms of, 1111  
 early stage, 1111  
 late stage, 1113  
 toxæmia of, 1109  
 treatment, 1123  
 treatment of, 1115  
 administration of fluids, 1123  
 anaesthesia, 1116  
 bacteriological, 1124  
 bacteriophage, 1124  
 blood-transfusion, 1124  
 cleansing the peritoneum, 1118  
 drainage, 1118  
 emulsified olive oil, 1124  
 Fowler position, 1123  
 of peritonitic toxæmia, 1123  
 operative, 1117  
 prevention and treatment of paralytic ileus, 1120  
 removal or closure of source of infection, 1116  
 vaccines, 1125  
 varieties of, 1125
- acute localised, 1097, 1098  
 autolytic, in injury of the liver, 739  
 chronic diffuse, 1137  
 chronic pyococcal peritonitis, 1139  
 encapsulating chronic peritonitis, 1139  
 tuberculous, 1137  
 diagnosis, 1138  
 treatment, 1138  
 chronic localised, 1098  
 general discussion of, 1097  
 in acute appendicitis, 932, 1131  
 in acute cholecystitis, 729  
 localised, 1097, 1098  
 primary, of children, 1135  
 diagnosis, 1136  
 mortality, 1135  
 treatment, 1136
- secondary, 1104  
 tuberculous, 1137  
 varieties of, 1097
- Permeation analgesia, 31
- Pernoxon as basal narcotic, 20
- Perrin and Lindsay on incidence of acute intussusception, 1019

- Petersen's operation of gastro-jejuno-stomy, 360
- de Petz clamp, 329
- Pharynx, anaesthesia in operations on, 44
- radium therapy in cancer of, 1596
- Phenoltetrachlorophthalein in the testing of liver function, 774
- Phlebitis, post-operative, 1256
- prevention of, 1257
- treatment of, 1258
- Phlegmon, gastric, *see* Gastritis, acute phlegmonous
- Photographic examination of the stomach, 88
- Phrenicotomy, anaesthesia in, 44
- Phthisis, abdominal manifestations in early, 96
- Physical examination in gastro-intestinal disorders, 90
- Physique in dyspepsia, 90
- Pick, on the value of splenectomy in Gaucher's disease, 861, 862
- Piles, *see* Haemorrhoids
- Pituitrin in treatment of operative shock, 24
- Planocaine, *see* Novocaine
- Pleural cavity, anaesthesia in surgery of, 49
- pressure changes in, during operations, 49, 50, 52
- Pleurisy, and appendicitis, differential diagnosis of, 918
- as a cause of abdominal pain, 95
- Pleurodynia, abdominal symptoms in, 96
- Pneumonia, and appendicitis, differential diagnosis of, 918
- lobar, simulating acute appendicitis in children, 95
- Polya types of partial gastrectomy for gastric ulcer, 386
- Polya-Balfour operation of partial gastrectomy, 491
- Polya-Moynihan operation of partial gastrectomy, 468
- Polycythæmia, X-ray therapy in, 822
- Polypi, of colon, 947, 953, 954
- of rectum, 1393
- fibrous, 1397
- glandular, 1394
- myxomatous, 1406
- of small intestine, 595
- of stomach, 522
- as a cause of pyloric obstruction, 286
- X-ray diagnosis of, 1503
- Polyposis, *see* Polypi
- Portal cirrhosis, *see* Liver, cirrhosis of pyæmia, 744
- vein, ligature of, in portal pyæmia, 744
- operative trauma to, 630
- phlebitis of, in acute cholecystitis, 729
- Post electric cauter, 328, 462
- Post-operative complications in abdominal surgery, 1215
- evisceration, 1222
- treatment, after gastric operations, 387
- Pott's disease, abdominal pain in, 97
- Pregnancy, and appendicitis, differential diagnosis of, 919
- appendicitis in, 933
- ectopic, and appendicitis, differential diagnosis of, 919
- Procaine, *see* Novocaine
- Procidentia recti, *see* Rectum, prolapse, complete
- Proctolysis, 1252
- Prolapse of rectum, *see* Rectum, prolapse of
- Propylene anaesthesia, 9
- Prostate, radium therapy in carcinoma of, 1621
- Prostatectomy, anaesthesia in, 54
- Protein shock in treatment of purpura hæmorrhagica, 877
- Pruritus ani, 1303
- etiology of, 1303
- symptomatology of, 1304
- treatment of, 1304
- Pseudo-leukæmic anaemia of infants, 852
- Psoas abscess in appendicitis, 919
- Purgatives, abuse of, as ætiological factor in appendicitis, 913
- Purpura hæmorrhagica, 871
- acute form of, 872
- alternative titles of, 871
- blood-platelet count in, 875
- blood-transfusions in, 873, 876
- case-reports, 878
- chronic form of, 874
- definition of, 871
- diagnosis of, 874
- diagnostic tests for, 874
- differential diagnosis of, 876
- essential hæmaturia due to, 879
- forms of, 872
- history of, 871
- incidence of, 872

- indications for splenectomy, *n.*, 872
  - menorrhagia in, 879
  - nervous symptoms in, 881
  - recurrence of, after splenectomy, 878
  - sex incidence of, 872
  - size of the spleen in, 875
  - splenectomy in, 877
    - advantages of, 873
    - indications for, 872
    - operative mortality of, 873
    - results of, 878
  - symptoms of, 871
  - synonyms of, 871
  - treatment of, 876
    - administration of blood, 876
    - by drugs, 877
    - dietetic, 877
    - medical, 876
    - non-specific protein shock, 877
    - radio-therapy, 876
    - splenectomy, 877
    - use of coagulents, 877
  - Purpura, thrombocytolytic, *see* Purpura hemorrhagica
  - Pyelitis, and appendicitis, differential diagnosis of, 918
  - Pyelograms, interpretation of, 1540
  - Pyelography, 1540
  - Pylephlebitis, complicating acute appendicitis, 934
    - suppurative, complicating acute cholecystitis, 729
  - Pyloric exclusion with partial gastrectomy for gastric and duodenal ulcer, *Finsterer's operation*, 336
  - Pyloric obstruction, 282
    - ætiology of, 282
      - gastroptosis, 289
      - mobile kidney, 289
      - new growths of the stomach, 284
      - peptic ulceration, 283
      - perigastric inflammation, 287
      - polypus, 286
      - pressure on duodenum or pylorus by a diseased neighbouring viscus, 287
      - scirrhus cancer, 285
      - simple stenosis of the pyloric canal, 286
      - traction upon or kinking of the duodenum, 289
    - appetite in, 291
    - bowels in, 292
    - diagnosis of, 292
    - differential diagnosis between benign and other types of obstruction, 293
    - gastric lavage in pre-operative treatment of, 320
    - loss of weight in, 292
    - pain in, 291
    - pathology of, 283
    - physical examination in, 292
    - symptoms of, 290
    - thirst in, 292
    - treatment of, medical, 577
      - operative, 295
      - pre-operative, 295
    - urine in, 292
    - vomiting in, 291
  - Pyloric occlusion for gastric and duodenal ulcer, 333
    - Bier method, 336, 337
    - Devine method, 336, 337
    - Kelling-Mayo method, 334
    - Wulms method, 334, 337
  - Pyloric stenosis, constipation in, 84
    - infantile, 145
      - ætiology of, 145
      - alkalosis in, 148
      - anæsthesia in operations for, 59
      - constipation in, 148
      - diagnosis of, 149
      - factors which have lowered death rate in, 151
      - incidence of, 145
      - jaundice in, 149
      - loss of weight in, 148
      - mortality of, 150
      - pathological anatomy of, 146
      - physical examination in, 149
      - post-operative complications of, 156
      - signs and symptoms of, 147
      - tetany in, 149
      - treatment of, 150
        - by Rammstedt's operation, 150, 153
        - post-operative, 152
        - pre-operative, 152
      - vomiting in, 147
      - X-ray diagnosis of, 1506
    - loss of weight in, 81
    - pain in, 76
    - simple, as a cause of pyloric obstruction, 286
- (*see also* Pyloric obstruction)

- Petersen's operation of gastro-jejuno-stomy, 360
- de Petz clamp, 329
- Pharynx, anaesthesia in operations on, 44
- radium therapy in cancer of, 1598
- Phenoltetrachlorophthalein in the testing of liver function, 774
- Phlebitis, post-operative, 1256
- prevention of, 1257
- treatment of, 1258
- Phlegmon, gastric, *see* Gastritis, acute phlegmonous
- Photographic examination of the stomach, 88
- Phrenicotomy, anaesthesia in, 44
- Phthisis, abdominal manifestations in early, 96
- Physical examination in gastro-intestinal disorders, 90
- Physique in dyspepsia, 90
- Pick, on the value of splenectomy in Gaucher's disease, 861, 862
- Piles, *see* Haemorrhoids
- Pituitrin in treatment of operative shock, 24
- Planocaine, *see* Novocaine
- Pleural cavity, anaesthesia in surgery of, 49
- pressure changes in, during operations, 49, 50, 52
- Pleurisy, and appendicitis, differential diagnosis of, 918
- as a cause of abdominal pain, 95
- Pleurodynia, abdominal symptoms in, 96
- Pneumonia, and appendicitis, differential diagnosis of, 918
- lobar, simulating acute appendicitis in children, 95
- Polya types of partial gastrectomy for gastric ulcer, 386
- Polya-Balfour operation of partial gastrectomy, 491
- Polya-Moynihan operation of partial gastrectomy, 468
- Polycythemia, X-ray therapy in, 822
- Polypi, of colon, 947, 953, 954
- of rectum, 1393
- fibrous, 1397
- glandular, 1394
- myxomatous, 1406
- of small intestine, 595
- of stomach, 522
- as a cause of pyloric obstruction, 286
- X-ray diagnosis of, 1502
- Polyposis, *see* Polypi
- Portal cirrhosis, *see* Liver, cirrhosis of pyæmia, 744
- vein, ligature of, in portal pyæmia, 744
- operative trauma to, 630
- phlebitis of, in acute cholecystitis, 729
- Post electric cauterly, 328, 462
- Post-operative complications in abdominal surgery, 1215
- evisceration, 1222
- treatment, after gastric operations, 387
- Pott's disease, abdominal pain in, 97
- Pregnancy, and appendicitis, differential diagnosis of, 919
- appendicitis in, 933
- ectopic, and appendicitis, differential diagnosis of, 919
- Procaine, *see* Novocaine
- Procidencia recti, *see* Rectum, prolapse, complete
- Proctolysis, 1252
- Prolapse of rectum, *see* Rectum, prolapse of
- Propylene anaesthesia, 9
- Prostate, radium therapy in carcinoma of, 1621
- Prostatectomy, anaesthesia in, 54
- Protein shock in treatment of purpura hæmorrhagica, 877
- Pruritus ani, 1303
- etiology of, 1303
- symptomatology of, 1304
- treatment of, 1304
- Pseudo-leukæmic anaemia of infants, 852
- Psoas abscess in appendicitis, 919
- Purgatives, abuse of, as ætiological factor in appendicitis, 913
- Purpura hæmorrhagica, 871
- acute form of, 872
- alternative titles of, 871
- blood-platelet count in, 875
- blood-transfusions in, 873, 876
- case-reports, 878
- chronic form of, 874
- definition of, 871
- diagnosis of, 874
- diagnostic tests for, 874
- differential diagnosis of, 876
- essential hæmaturia due to, 879
- forms of, 872
- history of, 871
- incidence of, 872

- indications for splenectomy in, 872
- menorrhagia in, 879
- nervous symptoms in, 881
- recurrence of, after splenectomy, 878
- sex incidence of, 872
- size of the spleen in, 875
- splenectomy in, 877
  - advantages of, 873
  - indications for, 872
  - operative mortality of, 873
  - results of, 878
- symptoms of, 871
- synonyms of, 871
- treatment of, 876
  - administration of blood, 876
  - by drugs, 877
  - dietetic, 877
  - medical, 876
  - non-specific protein shock, 877
  - radio-therapy, 876
  - splenectomy, 877
  - use of coagulents, 877
- Purpura, thrombocytolytic, *see* Purpura hæmorrhagica
- Pyelitis, and appendicitis, differential diagnosis of, 918
- Pyelograms, interpretation of, 1540
- Pyelography, 1540
- Pylephlebitis, complicating acute appendicitis, 934
  - suppurative, complicating acute cholecystitis, 729
- Pyloric exclusion with partial gastrectomy for gastric and duodenal ulcer, Finsterer's operation, 336
- Pyloric obstruction, 282
  - ætiology of, 282
  - gastroptosis, 289
  - mobile kidney, 289
  - new growths of the stomach, 284
  - peptic ulceration, 283
  - perigastric inflammation, 287
  - polypus, 286
  - pressure on duodenum or pylorus by a diseased neighbouring viscus, 287
  - scirrhus cancer, 285
  - simple stenosis of the pyloric canal, 286
  - traction upon or kinking of the duodenum, 289
- appetite in, 291
- bowels in, 292
- diagnosis of, 292
- differential diagnosis between benign and other types of obstruction, 293
- gastric lavage in pre-operative treatment of, 320
- loss of weight in, 292
- pain in, 291
- pathology of, 283
- physical examination in, 292
- symptoms of, 290
- thirst in, 292
- treatment of, medical, 577
  - operative, 295
  - pre-operative, 295
- urine in, 292
- vomiting in, 291
- Pyloric occlusion for gastric and duodenal ulcer, 333
  - Bier method, 336, 337
  - Devine method, 336, 337
  - Kelling-Mayo method, 334
  - Walms method, 334, 337
- Pyloric atresia, constipation in, 84
- infantile, 145
  - ætiology of, 145
  - alkalosis in, 148
  - anæsthesia in operations for, 59
  - constipation in, 148
  - diagnosis of, 149
  - factors which have lowered death rate in, 151
  - incidence of, 145
  - jaundice in, 149
  - loss of weight in, 148
  - mortality of, 150
  - pathological anatomy of, 146
  - physical examination in, 149
  - post-operative complications of, 156
  - signs and symptoms of, 147
  - tetany in, 149
  - treatment of, 150
    - by Rammstedt's operation, 150, 153
    - post-operative, 152
    - pre-operative, 152
    - vomiting in, 147
  - X-ray diagnosis of, 1506
- loss of weight in, 81
- pain in, 76
- simple, as a cause of pyloric obstruction, 286
- (*see also* Pyloric obstruction)

- Pyloric ulcer, 224, 246  
 choice of operation in, 307  
 simulating duodenal ulcer, 76  
*(see also Gastric and duodenal ulcer)*  
 Pyloroplasty, combined with gastro-  
 gastrostomy in hour-glass  
 stomach, 274  
 for chronic duodenal ulcer, Horsley's  
 operation, 348  
 Judd's operation, 352  
 for chronic duodenal ulcer without  
 stenosis, 314, 316  
 for perforated peptic ulcer, 246  
 Pylorus, carcinoma of, and pyloric  
 obstruction, 284, 293  
 congenital hypertrophic stenosis of, *see*  
 Pyloric stenosis infantile  
 duodenal involvement in carcinoma of,  
 603  
 hypertrophy of, X-ray diagnosis in,  
 1506  
 obstruction of, *see* Pyloric obstruction  
 scirrhus cancer of, 285  
 stenosis of, *see* Pyloric stenosis  
 ulcer of, *see* Pyloric ulcer  
 Pyrosis in dyspepsia, 79, 80

## Q

- Quick's results in radium therapy of  
 cancer of the maxilla, 1591  
 results in radium treatment of cancer of  
 the tongue, 1587  
 Quick and Martin's results in Lepage-  
 Janeway operation of gastro-  
 stomy, 458  
 Quinine and urea hydrochloride as local  
 analgesic, 29

## R

- Radiation therapy, *see* Radio-therapy;  
 Radium therapy  
 Radiography, *see* X-ray diagnosis; X-ray  
 examination  
 Radiology, *see* X-ray diagnosis; X-ray  
 examination  
 Radio-therapy, cavitory method, 1635  
 clinical observations on the action of  
 different wave-lengths, 1632

- comparison of Gamma and X-radia-  
 tions, 1630  
 distance radiation, 1638  
 dosage in, 1638  
 units of, 1641  
 high-voltage X-rays in, *see* X-rays, high  
 voltage  
 in abnormal menopause, 1665  
 in benign conditions of the female  
 genital tract, 1665  
 in carcinoma of the cervix uteri, 1643  
 ætiology, 1643  
 diagnosis, 1645  
 definition of International Stages 1-4,  
 1647  
 history, 1650  
 Paris technique, 1653  
 pathology, 1644  
 results, 1655  
   at Marie Curie Hospital, 1657  
   at Mayo Clinic, 1656  
   at Mount Vernon Hospital, 1657  
   at Munich, 1656  
   at Stockholm, 1656  
 statistical results, 1656  
 Stockholm technique, 1650  
 technique, 1649  
 in carcinoma of the corpus uteri, 1658  
 in carcinoma of the ovary, 1664  
 in carcinoma of the vagina, 1662  
 in carcinoma of the vulva, 1663  
 in chorion epithelioma, 1660  
 in diseases of the spleen, 820  
 in gynecology, 1625  
   clinical aspects of, 1612  
 in leucoplakia, 1670  
 in the leukæmias, 821  
 in lymphadenoma, 823  
 in menorrhagia or irregular hæmo-  
 rrhage, 1665  
   production of artificial menopause in  
   young patients, 1669  
   results of treatment of abnormal  
   menopause, 1668  
   technique of, 1667  
   treatment by X-rays, 1667  
 in polycythæmia, 822  
 in purpura hæmorrhagica, 876  
 in sarcoma of the uterus, 1661, 1662  
 in tuberculosis of the cervix and vagina,  
 1670  
 in uterine cancer, 1643



- in uterine fibroids, 1668
- in uterine hæmorrhage, 1665
- interstitial irradiation, 1637
- methods of application, 1635
  - cavitary method, 1635
  - distance radiation, 1638
  - interstitial irradiation, 1637
  - surface irradiation, 1635
- nature of X-radiation, 1629
- physics of, 1625
- radio-sensitivity, 1633
- surface irradiation, 1635
- units of dosage, 1641
- (see also Radium therapy; X-rays, high voltage)
- Radium, 1557, 1625
  - Alpha rays, 1557, 1626
  - Beta rays, 1558, 1627
  - biological action of, 1631
  - choice between radon and, 1553
  - Gamma rays, 1558, 1627
  - products of, 1558
- Radium bomb, 1570
  - method of application, 1572
  - Westminster Hospital bomb, 1572
  - (see also Radium therapy)
- Radium burns, 1665
  - delayed, 1566
  - immediate, 1566
- Radium emanation, see Radon
- Radium needles, 1568
- Radium therapy, 1557
  - biological effects of radiation, 1561
  - cavitary irradiation, 1567
  - choice between radon and radium, 1559
  - distance radiation, 1570
  - dosage, notation of, 1561
  - effects of radiation, 1564
    - destruction, 1565
    - inhibition, 1564
    - radium burns, 1565
    - stimulation, 1564
  - filtration, 1560
  - in cancer, 1557
    - compared with surgery, 1574
    - final results of, 1574
    - value of, 1574
      - as a palliative measure, 1573
  - in cancer of the anal canal, 1619
  - in cancer of the bladder, 1619
  - methods of treatment, 1629
  - results of, 1620
- in cancer of the breast, 1599
  - age factor, 1601
  - combined with surgery, 1602
  - compared with surgery, 1600
  - post-operative treatment, 1606
  - pre-operative radium treatment, 1606
  - results of, 1607
  - selection of treatment, 1601
  - technique of treatment, 1602
    - first stage: interstitial irradiation, 1602
    - after-treatment, 1604
    - second stage: surface irradiation, 1605
  - treatment of post-operative recurrences, 1606
  - value and results of, 1600, 1601
- in cancer of the buccal mucosa, 1588
- in cancer of the larynx and pharynx, 1596
- in cancer of the lip, 1589
- in cancer of the maxilla, 1592
  - results of, 1594
- in cancer of the prostate, 1621
- in cancer of the rectum, 1614
  - intra-rectal irradiation, 1618
  - operability and, 1614
  - peri-rectal irradiation, 1617
  - principles of irradiation of the rectum, 1616
  - technique, 1617
  - use of radon seeds, 1619
- in cancer of the skin, 1607
- in cancer of the tongue, 1576
  - changes produced by treatment, 1582
  - oral toilet, 1576
  - results, 1586
  - technique of treatment of the primary growth, 1578
  - treatment of the lymphatic area, 1584
- in cancer of the tonsil, 1595
- in epithelioma of the palate, 1590
- in epithelioma of the penis, 1612
- in epithelioma of the skin, 1610
- in malignant disease, 1557
- in purpura hæmorrhagica, 876
- in rodent ulcer, 1608
- interstitial irradiation, 1568
- mass radiation, 1570
- methods of, 1567
  - cavitary irradiation, 1567

Radium therapy, methods of, distance radiation, 1570  
 interstitial irradiation, 1568  
 surface irradiation, 1569  
 notation of dosage, 1561  
 palliative value of, 1575  
 physics of, 1557  
 physiological effects of, 1562  
 products of radium, 1558  
 radiation from an X-ray tube, 1629  
 radium bomb, 1570  
 sensitivity to, 1562  
 surface irradiation, 1569  
 teluradium, 1570  
 time factor in, 1563  
 types of radiation, 1557  
 value of, 1574  
   as a palliative measure, 1575  
 (see also Radio-therapy; Radon; X-rays, high voltage)  
 Radon, 1558, 1626  
   choice between radium and, 1559  
 (see also Radium therapy)  
 Rammstedt's operation for infantile pyloric stenosis, 150  
   technique of, 153  
 Rankin and Newell on benign tumours of the intestines, 593  
   results of operations for benign tumours of small intestine, 600, 621  
 Récamier's operation for anal fissure, 1302  
 Rectal examination in abdominal disease, 114  
 Rectal fistula, see Ano-rectal fistula  
 Recto sigmoid colon, stricture of, 917  
 Recto-sigmoidectomy for prolapse of the rectum, 1324  
 Rectum, 1261  
   adenoma or glandular polypus of, 1394  
   diagnosis of, 1395  
   physical examination in, 1395  
   symptoms of, 1395  
   treatment of, 1397  
     post-operative, 1397  
   arterial supply of, in relation to hæmorrhoids, 1271  
   benign tumours of, 1393  
     varieties of 1394  
   carcinoma of, 1409  
     adenoid carcinoma, 1411  
     ætiology of, 1414

anæsthesia in operations for, 51, 60, 1456  
 anatomy of the lymphatics in relation to spread of, 1431  
 classification of, 1409  
   Dukes', 1412  
 clinical varieties of, 1409  
 colloid or mucoid carcinoma, 1412  
 constipation as early symptom in, 1414  
 differential diagnosis of, 1423  
 digital examination in, 1425  
 Dukes' classification of, 1412  
 examination of the cancerous rectum, 1425  
 inoperable, ascites in, 1480  
 colostomy in, 1474  
   growths involving the anal canal, 1477  
   growths situated at the recto-sigmoidal junction, 1475  
   growths situated in the ampulla, 1476  
   hæmorrhage in, 1478  
   increased secretion of rectal mucus, 1478  
   palliative treatment of, 1478  
   pelvi-rectal and ischio-rectal sup-puration in, 1479  
   pressure upon or involvement of nerves of the sacral plexus, 1479  
   recto-vesical or recto-vaginal fistula, 1479  
   treatment of, 1473  
 lymphatic system in relation to spread of, 1431  
   extra-mural lymphatics, 1432  
   intermediary lymphatics, 1432  
   intra-mural lymphatics, 1431  
   melanotic carcinoma, 1412  
   metastasis of, see Spread of, below  
   operability of, 1445  
   bearing of pathology upon, 1448  
   determination of, by abdominal exploration, 1447  
   by digital examination, 1416  
 operations for, 1451  
   abdomino-anal operation, 1453  
   abdomino-perineal operation, 1453  
   anæsthesia for, 54, 60, 1456  
   blood-pressure estimation before, 1456

- Coffey's two-stage operation, 1453, 1454
- Miles' one-stage operation, 1453
- mortality of the one-stage operation, 1471
- operability rate of, 1471
- post-operative treatment, 1470
- preparatory treatment, 1455
- recurrence rate of the one-stage operation, 1472
- survival rate, 1473
- technique of, by stages, 1456
- anæsthesia in, 54, 60, 1456
- perineal excision, 1452
- perineal resection, 1452
- perineo-abdominal operation, 1473
- radical, *see* Abdomino-perineal operation, *above*
- vaginal resection, 1452
- operative treatment, bearing of pathology upon, 1448
- palliative treatment of inoperable cases, 1478
- papilliferous carcinoma, 1409
- pre-operative treatment of, 1455
- proctoscopic or sigmoidoscopic examination in, 1427
- radical operation for, 1453  
(*see also* Operations for, *above*)
- radium therapy in, 1614
- recurrence rate after radical operation for, 1472
- situation of, 1426
- spread of, 1428
- by means of the lymphatic system, 1431
- downward zone of spread, 1436
- lateral zone of spread, 1437
- upward zone of spread, 1438
- direct extension through continuity of tissue, 1428
- through the venous system, 1430
- symptomatology of, 1414
- during pre-ulcerative stage, 1418
- during the progress of surface ulceration, 1420
- of a cancerous growth at the recto-sigmoidal junction, 1416
- of carcinoma arising in the anal canal, 1417
- of carcinoma of the ampulla, 1418
- when the growth has extended beyond the fascia propria and has implicated neighbouring structures, 1422
- when the rectal wall has been penetrated and infiltration of the peri-rectal tissues has occurred, 1422
- treatment of, 1450
- of inoperable cases, 1473
- examination of, in suspected cases of cancer, 1425
- excision of, *see* Rectum, carcinoma, operations for, *above*
- fibroma or fibrous polypus, 1397
- diagnosis of, 1398
- physical examination in, 1398
- symptoms of, 1398
- treatment of, 1398
- fistula of, *see* Ano-rectal fistula
- glandular polypus of, 1394
- lymphatics of, in relation to the spread of carcinoma, 1431
- myxoma or myxomatous polypus of, 1406
- symptoms of, 1406
- treatment of, 1407
- oxygen insufflation of, in treatment of ulcerative colitis, 945
- polypus of, 1304, 1397, 1406
- prolapse of, 1307
- complete, 1314
- etiology of, 1314
- differential diagnosis of, 1318
- exciting causes in adults, 1315
- in children, 1315
- operative treatment of, 1320
- contracting the lumen of the rectum, 1322
- fixation of the rectum to the sacrum, 1322
- narrowing the anal orifice, 1324
- recto-sigmoidectomy, 1324
- mortality and end-results of, 1326
- post-operative treatment in, 1326
- technique of, 1324
- suspension of the rectum from the abdomen, 1323
- palliative treatment of, 1319
- pathological anatomy of, 1314, 1316

- Rectum, prolapse of, predisposing causes of, 1314  
 symptomatology of, 1316  
 treatment of, 1319  
 partial, 1307  
   ætiology of, 1307  
   differential diagnosis of, 1310  
     from complete prolapse, 1311  
     from internal piles, 1310  
   operative treatment of, 1312  
     after-treatment, 1313  
     technique of, 1313  
   palliative treatment of, 1311  
     improving the tone of the sphincter muscles, 1312  
     prevention of recurrence of the protrusion, 1312  
     reduction of the protruded bowel, 1311  
   pathological anatomy of, 1309  
   symptomatology of, 1309  
   treatment of, 1311  
 sarcoma of, 1408  
 stricture of, *see* Stricture of anus and rectum  
 villous tumour of, 1399  
   ætiology of, 1401  
   operative treatment of, 1405  
   pathological anatomy of, 1403  
   post-operative treatment of, 1405  
   recurrence of, 1406  
   symptoms of, 1401  
   treatment of, 1404  
 Régaud's results in radium treatment of cancer of the tongue, 1587  
 Regurgitation in dyspepsia, 79, 80  
 Rehfuß' stomach tube, 117  
 Reid, Zininger and Merrell's method of suture by silver wire, 1232  
 Renal calculus, X-ray diagnosis of, 1544  
   colic and appendicitis, differential diagnosis of, 918  
   disease in relation to dyspepsia, 98  
   unction, tests for, 131  
   pelvis, X-ray examination of, 1540, 1517, 1550  
   (*see also* Kidney)  
 Respiration, artificial, in primary cardiac failure during anæsthesia, 25  
   effect of evipan anæsthesia upon, 15  
 Respiratory affections as a cause of abdominal pain, 95  
   diffusion in basal narcosis, 21  
   obstruction and endotracheal anæsthesia, 16  
 Retention of urine, *see* Urine, retention of  
 Reticulo-endothelial system, 748  
   changes in, following splenectomy, 818  
 Retractor, Deaver's, 665  
 Retro-colic gastro-jejunostomy for gastric and duodenal ulcer, 380, 382  
 Rihs, anæsthesia in resection of, 50, 51  
 Rich on the pathology and classification of jaundice, 754  
 Richter's hernia, 981, 1043, 1045  
   (*see also* Hernia)  
 Riedel-Rodman operation for gastric ulcer, 316  
 "Rodent ulcer" of the small intestine, 613  
 Rodent ulcer, radium therapy in, 1608  
 Roeder on results of total gastrectomy, 500  
 Roentgenography, *see* X-ray diagnosis; X-ray examination  
 Rolleston on distribution of duodenal cancer, 603, 621  
 Roscoe, Graham and Lewis on hyperchlorhydria in cases of stomal ulcer, 407  
 Rosenthal on splenic anæmia, 835, 837  
 Rnux's method in Y for gastro-jejunostomy, 360, 386  
   secondary peptic ulcer following, 404  
 Rovsing's sign in diagnosis of appendicitis, 916  
 Rowlands on mortality of gastric carcinoma, 416  
 Rowlands and Simpson on anæmia following gastric operations, 402  
 Rupture of gall-bladder, 729  
   of liver abscess, 743  
   of spleen, 890  
   of stomach, 173  
 Ryle's stomach tube, 118

## S

- Sacs, hernial, *see* Hernial sacs  
 Saint on glandular hyperplasia of small intestine, 597, 621  
   on multiple hæmangiomas of the jejunum, 597, 621

- Salmon's ligature operation for hæmorrhoids, Miles' modification of, 1284
- Salpingitis and appendicitis, differential diagnosis of, 919
- Salvarsan in treatment of purpura hæmorrhagica, 877
- jaundice due to, 769
- Sarcoma of rectum, 1408
- of small intestine, 613
- of spleen, 847
- of stomach, 517
- Sargent's depressor, 920
- Scalpels as source of wound infection, 1218
- Schimmelbusch's syringe, 527
- Schoemaker's operation of partial gastrectomy for gastric and duodenal ulcer, 384, 385
- Schofield on radon treatment of duodenal carcinoma, 607, 621
- Scopolamine preliminary to anæsthesia, 19
- Scott Ridout forceps, 332
- Scurocaine, *see* Novocaine
- Secondary peptic ulcer, *see* Gastric and duodenal ulcer, secondary
- Sedative drugs before operation, 19
- "Sentinel pile," 1292
- Sepsis following abdominal operations, 1215
- Septicæmia, jaundice in, 84
- Sewell and Hoskin's bed-lifter, 926
- Sex factor in gastro-intestinal disorders, 91
- Sherren's delayed (Ochsner-Sherren) treatment of appendicitis, 924
- operation of gastro-jejunostomy, 360
- skin triangle in appendicitis, 916
- stomach clamp, 328, 461, 462
- Shipway's warm ether-vapour apparatus, 11
- Shock, operative, 22
- causes of, 22
- drugs in treatment of, 24
- due to anæsthetic overdosage, 23
- fluids in, 22, 23
- loss of temperature in, 23
- oxygen deprivation and, 23
- pathology of, 22
- signs of, 22
- traumatic, 22
- treatment of, 23, 24
- Stalo-adenitis, acute septic, *see* Parotitis, acute suppurative
- Sigmoid colon, carcinoma of, 956, 960
- resection of, 965
- stricture of, 917
- volvulus of, 948, 1027
- Sigmoidoscopy in ulcerative colitis, 945
- Simpson and Rowlands on anæmia following gastric operations, 402
- Sinclair on gastric polyposis, 522
- Skin, appearance of, in gastro-intestinal disease, 92
- disinfection of, in operations for peptic ulcer, 322
- freezing of, by ethyl chloride, 10, 33
- hyperæsthesia and hyperalgesia of, in abdominal disease, 105
- preparation of, for abdominal operations, 446
- in relation to wound infection, 1216
- radium therapy in cancer of, 1607
- in epithelioma of, 1610
- Sleeve resection of the stomach, 276
- Small intestine, *see* Intestine, small
- Smith's (Durden) results in radium therapy of carcinoma of the bladder, 1620
- Smoking, *see* Tobacco smoking
- Snake venom in treatment of purpura hæmorrhagica, 877
- Sodium amytal as basal narcotic, 20
- Sodium evipan, *see* Evipan
- Sodium morrhuate in treatment of hæmatemesis due to portal cirrhosis, 744
- Sodium soneryl as basal narcotic, 20
- Somnoform anæsthesia, 10
- Souttar's cases of acute intestinal obstruction, 974
- Spectroscopic method for detection of occult blood, 129
- Sphincterotomy for anal fissure, 1299
- Spinal analgesia, *see* Analgesia, local, spinal
- Spinal block, *see* Analgesia, local, spinal
- Spine, abdominal pain in diseases of, 97
- Spinocain, 28
- Splanchnic block, Brann's technique in, 56
- in abdominal surgery, 55, 56, 57
- Kappis' method, 56
- (*see also* Analgesia)

## Spleen, 803

abscess of, 842

ætiology of, 843

diagnosis of, 843, 844

drainage of, 845

examination of the blood in, 844

rupture of, 844

splenectomy for, 844

splenic puncture in, 844

symptoms of, 843

treatment of, 844

X-ray diagnosis of, 844

accessory, 809

in cases of purpura hæmorrhagica, 878

benign growths of, 847

blood-platelets in relation to, 829

blood-vessels of, 806

cysts of, 845

dermoid cysts of, 846

ectopic, 850

effects of X-rays on, 820

enlargement of, in dyspeptic states, 111

floating, *see* Spleen, wandering, *below*

gumma of, 849

hæmatemesis in diseases of, 82

hydatid cysts of, 846

irradiation of, in the leukæmias, 821

in lymphadenoma, 823

in polycythæmia, 822

in purpura hæmorrhagica, 876

malarial, 849

new growths of, 847

puncture of, 844

radio-therapy in diseases of, 822

removal of, *see* Splenectomy

rupture of, 890

causes of, 890

clinical types of, 893

"delayed" type of, 895

due to direct injuries, 890

due to indirect injuries, 890

incidence of, 890

naked-eye appearances in, 893

operative mortality in, 897

splenectomy in, 896

technique of, 897

spontaneous, 890, 891, 892

symptomatology of, 894

traumatic, 890

treatment of, 896

sarcoma of, 847

size of, in purpura hæmorrhagica, 875

surgical diseases of, 842

syphilis of, 848

torsion of, 851

tuberculosis of, 847

wandering or floating, 850

causes of, 850

complications of, 851

diagnosis of, 851

symptoms of, 851

treatment of, 852

X-ray therapy in diseases of, 821

## Spleniculus, 809

excision of, in splenectomy, 809

growth of, after splenectomy, 809

## Splenectomy, blood changes after, 817

blood-platelet count after, 832

blood-platelet increase after, 831

blood-platelets in relation to, 837

burst abdomen following, 817

complications of, 816

empyema after, 817

hæmorrhage complicating, 816

in Egyptian splenomegaly, 899

technique of, 902

in Gaucher's disease, 861, 862

in hæmolytic jaundice, 883

mortality and late results of, 887

in purpura hæmorrhagica, 877

advantages of, 873

indications for, 872

operative mortality of, 873

recurrences after, 878

results of, 878

in rupture of the spleen, 896, 897

in splenic anæmia, 869

ligature of coronary vein in, 870

mortality and late results of, 871

pre-operative measures in, 869

in splenomegaly, technique of, 812

incision and choice of incision, 806

indications for, 803

intestinal obstruction following, 817

ligature of splenic artery as alternative to, 818, 810

mortality of, 824

peritoneal effusion after, 816

persistent hæmorrhage following, 817

physiological effects of, 817

pleural effusion after, 817

pneumonia after, 817

post-operative shock in, 816

- pulmonary collapse after, 817  
 removal of accessory spleen or splenuli in, 809  
 results of, at the Mayo Clinic, 824  
 sub-diaphragmatic suppuration after, 816  
 technique of, 805  
   in Egyptian splenomegaly, 902  
   in splenomegaly, 812  
 thrombosis after, 816, 832  
 ventral hernia following, 817  
 X-ray treatment as alternative to, 821
- Splenic anemia (Banti's disease), 865  
 aetiology and pathology of, 865  
   ascites in, 868  
   blood changes in, 866  
   blood-platelets in, 835, 837, 838  
   bone-marrow in, 868  
   chronicity of, 865  
   cirrhosis of the liver in, 868  
   dyspeptic symptoms in, 868  
   haemorrhage in, 867  
   lymph glands in, 868  
   mortality of splenectomy for, 871  
   pathology of, 865  
   post-operative thrombosis in, 835, 837, 838  
   rupture of oesophageal varices in, 867  
   splenectomy for, 869  
     mortality and late results of, 871  
     pre-operative measures in, 869  
   splenomegaly in, 865  
   stages of, 865  
   symptoms of, 865  
   treatment of, 868
- Splenic artery, aneurysm of, 842  
 ligature of, as alternative to splenectomy, 818, 840
- Splenic puncture, 844
- Splenomegalic cirrhosis, 855
- Splenomegaly, anemia in infancy and childhood and, 852  
 Banti's disease and, 865  
 cirrhosis and, 855  
 Egyptian, 898  
   aetiology of, 898  
   blood picture in, 899  
   parasitology of, 898  
   splenectomy for, 899  
     after-treatment, 901  
     anaesthesia, 902  
     mortality of, 899  
     technique of, 899, 902  
     stages of, 898  
   symptomatology of, 898, 899  
 Gaucher's disease and, 855  
 in haemolytic jaundice, 883  
 in splenic anaemia, 865  
 von Jaksch's disease and, 852  
 ligature of splenic artery as alternative to splenectomy in, 818  
 malarial, 849  
 pseudo-leukaemic anaemia of infants and, 852  
 splenic anaemia and, 865  
 technique of splenectomy in, 812  
 thrombophlebitic, 853
- Stamm's method of gastrotomy, 448
- "Steer-horn" stomach, 182
- Stenosis, of pylorus, *see* Pyloric stenosis  
 of rectum, *see* Stricture of anus and rectum
- Stewart on adenomata of the stomach, 521  
 on incidence of ulcer-cancer of the stomach, 561
- Sthenic constitution in gastro-intestinal diseases, 90
- Stiles' operation of gastro-jejunostomy, 360
- Stitch abscess, 1217, 1219
- Stockholm technique in radio-therapy of carcinoma of the cervix, 1650
- Stoma in gastro-jejunostomy, 361
- Stomach, acidity of, in relation to dyspepsia, 122  
 acute suppurative cellulitis of, *see* Gastritis, acute phlegmonous  
 adenoma of, 521  
 anaesthesia in surgery of, 55  
 angioma of, 522  
 atonic, and pyloric obstruction, 294  
 benign growths of, 519  
   physical examination in, 524  
   symptoms of, 523  
   treatment of, 525  
   X-ray diagnosis of, 1502  
 carcinoma of, 416  
   adenomata and, 418, 521  
   aetiology of, 416  
   age incidence of, 417  
   anaemia in, 91, 439  
   anorexia in, 82, 439  
   benign growths in relation to, 418

- Stomach, carcinoma of, cachexia in, 91  
 causes of delay in diagnosis of, 441  
 chronic gastric ulcer and, 417  
 (see also Ulcer-cancer of the stomach)  
 chronic gastritis and, 417  
 clinical features of, 431  
 clinical types of, 432  
 condition of the bowels in, 440  
 constipation in, 81  
 curability of, 443  
 determination of operability in, 465  
 diagnosis of, 428  
 differential diagnosis of, 441  
 distaste for meat in, 82  
 dyspepsia in, 72  
 dysphagia in, 440  
 emaciation in, 438  
 exciting factors in, 418  
 gastric analysis in diagnosis of, 116  
 heredity in, 417  
 "hunger pain" in, 77  
 incidence of, 416  
 individual symptoms in, 437  
 inoperability in, 440  
 inoperable, management of, 445  
 jaundice in, 440  
 loss of weight in, 81  
 lymphatic glands in, 466  
 melena in, 82  
 modes of extension of, 425  
 nausea as symptom in, 77, 438  
 operations for, 447  
 pain in, 74, 76, 437  
 palliative operations for, 447  
   excision of growth, 460  
   exclusion of growth (Devine's operation), 459  
   gastro-jejunostomy, 459  
   gastrostomy, 447  
   jejunostomy, 458  
 palliative treatment of, 447  
 palpable tumour in, 440  
 pathology of, 418  
 peripheral venous thrombosis in, 440  
 pre-operative treatment of, 445  
 pyloric obstruction in, 284  
 pyrexia in, 440  
 radical operations for, 447, 460  
   partial gastrectomy, 460  
   total gastrectomy, 500  
 recurrence of, X-ray diagnosis of, 1504  
 sex incidence of, 417  
 spread of, 425  
 symptoms of, 431  
 treatment of, 444  
 weight in, 438  
 X-ray diagnosis of, 1494, 1504  
 (see also Ulcer-cancer of the stomach)  
 colloid cancer of, 418  
 cysts of, 523  
 dilatation of, 200  
   acute, 158  
   aetiology of, 159  
   diagnosis of, 164  
   incidence of, 158  
   pathological anatomy of, 161  
   post-operative, 160  
   prognosis of, 164  
   signs and symptoms of, 162  
   treatment of, 165  
   vomiting in, 162  
   atonic, pain in, 77  
 diverticulum of, X-ray diagnosis of, 1489  
 examination of, with special instruments, 87  
 fibroma of, 521  
 fistula of, see Gastric fistula  
 foreign bodies in, 174  
   gastrotomy for, 176  
   symptoms of, 175  
   treatment of, 175  
   X-ray examination in, 175  
 fungating and polypoid tumours of, 420  
 gastro-photography in examination of, 88  
 gastroscopic examination of, 87  
 hair-ball in, 174, 175  
 hour-glass, see Hour-glass stomach  
 inflammation of, see Gastritis  
 injuries of, 172  
 (see also Stomach, rupture of, below)  
 instruments for examination of, 87  
 "leather-hottle," 422  
   X-ray diagnosis of, 1497  
 lipoma of, 522  
 malignant ulcer of, 418  
 (see also Ulcer-cancer of the stomach)  
 myomata of, 520  
 nerve supply of, in relation to ileus, 1183  
 new growths of, 416  
 normal, X-ray examination of, 1483



- operations on, hæmatemesis after, 82
- post-operative complications of, 391
- post-operative treatment, 387
- perforation of, pain in, 76
- perigastric adhesions, 255
- polyposis of, 522
- X-ray diagnosis of, 1502
- rupture of, 173
- sarcoma of, 517
  - age incidence of, 517
  - diagnosis of, 519
  - incidence of, 517
  - pathology of, 517
  - treatment of, 519
- sleeve resection of, in hour-glass stomach, 276
- spasm of, X-ray appearances in, 1486
- "steer-horn," 182
- syphilis of, 210
  - X-ray diagnosis of, 1501
- tuberculosis of, 211
  - X-ray diagnosis of, 1502
- ulcer of, *see* Gastric ulcer; Gastric and duodenal ulcer
- ulcer-cancer of, *see* Ulcer-cancer of the stomach
- wounds of, 172
- X-ray examination, in diseases of, 1489
  - of the normal stomach, 1483
  - (*see also under* Gastric)
- Stomach tubes, 117
  - technique of passing, 118
- Stomal ulcer, *see* Gastric and duodenal ulcer, secondary
- Stools, *see* Fæces
- Stovaine as local analgesic, 29
- Strangulated hernia, *see* Hernia, strangulated
- Streptococcic carriers in relation to wound infection, 1218
- Streptococcus fecalis, associated with appendicitis, 913
- Stricture of anus and rectum, 1373
  - amyloid degeneration of kidneys due to, 1389
  - cicatricial or fibrous, 1373
  - complications of, 1388
  - following operations for hæmorrhoids, 1290
  - intestinal obstruction due to, 1388
  - linear or annular stricture, 1373
  - ætiology of, 1373
  - pathological anatomy of, 1375
  - symptomatology of, 1374
  - treatment of, 1376
    - when the stenosis is situated at the anal orifice, 1376
    - when the stenosis is situated at the level of the upper border of the internal sphincter, 1377
    - when the stenosis is situated at a level of three or more inches above the anal orifice, 1378
- para-rectal suppuration due to, 1388
- sequelæ of, 1388
- tubular stricture, 1379
  - ætiology of, 1379
  - chronic inflammation of the uterus as a causative factor in, 1381
  - pathological anatomy of, 1381
  - symptomatology of, 1382
  - syphilis and, 1380
  - treatment of, 1385
    - by dilatation, 1386
    - general, 1388
    - local, 1385
  - varieties of, 1373
- Subarachnoid spinal block, 35
- Subphrenic abscess, 221, 1153
  - anatomy of the sub-diaphragmatic spaces in relation to, 1154
    - left antero-inferior intra peritoneal space, 1157
    - left postero-inferior intra-peritoneal space, 1157
    - left superior intra-peritoneal space, 1157
    - retroperitoneal spaces, 1158
    - right antero-superior intra-peritoneal space, 1156
    - right inferior intra-peritoneal space, 1156
    - right postero-superior intra-peritoneal space, 1155
  - appendicular origin of, 1163
  - bacteriology of, 1159
  - biliary origin of, 1165
  - causes of, 1162
    - appendicular, 1163
    - gastro-duodenal, 1164
    - hepatico-biliary, 1164
  - clinical features of, 1167
  - clinical varieties of, 1166
  - complicating acute cholecystitis, 729

Subphrenic abscess, diagnosis of, 1165  
 differential diagnosis of individual space infections, 1170  
 fate of untreated abscesses, 1159  
 gastro-duodenal origin of, 1164  
 hepatico-biliary origin of, 1164  
 incidence of abscess in the various subphrenic spaces, 1160  
 morbid anatomy of, 1159  
 mortality of, 1153  
 pathology of, 1159  
 physical signs in, 1168  
 prevention of, 1169  
 routes of infection in, 1161  
   direct infection, 1161  
   intra-peritoneal spread, 1161  
   lymphatic spread, 1161  
   spread along retroperitoneal cellular tissues, 1162  
   spread via the blood stream, 1162  
 rupture of, 1160  
 symptoms of, 1168  
 thoracic complications of, 1160, 1171  
 treatment of, 1169  
   conservative, 1171  
   operative, 1171  
     anterior extra-peritoneal operation, 1177  
     by transperitoneal route, 1173  
     by transpleural route, 1172  
     by trans-serous route, 1172  
     extra-serous approach in, 1173  
     methods of approach in, 1172  
     posterior extra-peritoneal operation, 1174  
 Subphrenic infection, 1167  
   suppuration, *see* Subphrenic abscess  
 Suction apparatus for gastric operations, 329, 462  
 Sullivan's operation of end-to-side choledcho-duodenostomy (Coffey's modification), 712, 714  
 Sunlight, artificial, in pre-operative treatment of gastric carcinoma, 446  
   in pre-operative treatment of peptic ulcer, 322  
 Suppurative parotitis, *see* Parotitis, acute suppurative  
 Suprarenal gland, enlargement of, in dyspepsia, 112  
 Surface analgesia, 31

Suture, by silver-wire, for closure of disrupted wounds, 1232  
 Connell's, 330  
   in relation to wound infection, 1217  
   loop-on-the-mucosa, 330, 331  
   material for abdominal surgery, 460  
   Mayo-Connell, 330  
 Syncaine, *see* Novocaine  
 Syphilis, and dyspepsia, 70  
   as a cause of stricture of the rectum, 1380  
   congenital, jaundice in, 769  
   in relation to abdominal disease, 138  
   jaundice in, 84, 763, 768, 769  
   of the spleen, 848  
   of the stomach, 210  
   X-ray diagnosis of, 1501  
 Syringe, Schimmelbusch's, 527

## T

Tabes dorsalis, abdominal manifestations in, 97  
   gastric crises of, 204  
 Talma-Morison operation, *see* Omentopexy  
 Teleradium therapy, 1570  
   (*see also* Radium therapy)  
 Test meals, 119  
   Ewald's one hour meal, 119  
   fractional, 119  
     examination of fasting juice, 121  
     interpretation of results, 121  
     technique of, 119  
   with histamine or alcohol, in diagnosis of ulcer-cancer of the stomach, 563  
 Tetany in infantile pyloric stenosis, 149  
 Tetra-cloth forceps, 328  
 Thirst, in pyloric obstruction, 292  
   post-operative, 1251  
 Thoracic wall, anaesthesia in operations on, 48, 49  
 Thoracotomy, anaesthesia for, 49  
 Thorax, anaesthesia in surgery of, 9, 48, 51  
   diathermy in surgery of, 52  
   premedication in surgery of, 48  
 Thrombocytolytic purpura, *see* Purpura hæmorrhagica  
 Thrombocytopenia, *see* Purpura hæmorrhagica

- Thrombopenia, essential, *see* Purpura hæmorrhagica
- Thrombosis, femoral, post-operative, 1257, 1258  
 following splenectomy, 816, 832  
 of mesenteric blood-vessels, 1030  
 peripheral venous, in carcinoma of the stomach, 440  
 post-operative, 834  
   blood-platelets in relation to, 833, 834  
   in splenic anemia, 835, 837  
   low protein diet in, 834
- Thyroid extract in treatment of purpura hæmorrhagica, 877
- Thyroid gland, anaesthesia in surgery of, 44
- Thyroidectomy, partial, avertin narcosis in, 27
- Tobacco smoking, and dyspepsia, 70  
 during treatment of peptic ulcer, 576  
 in relation to peptic ulceration, 187, 302
- Tongue, radium therapy in cancer of, 1576
- Tonsil, radium therapy in cancer of, 1595
- Trans-sacral block in perineal surgery, 60
- Tremor during ether anaesthesia, 12
- Trichobezoar, 174, 175
- Tripartite rubber bag for operating tables, 666
- Trocar and cannula, Mayo's, 665
- Tropacocaine, 29
- Tropical abscess of liver, *see* Liver, abscess, tropical
- Tuberculosis, of colon, 947  
 of kidney, X-ray diagnosis of, 1544  
 of liver, 747  
 of spleen, 847  
 of stomach, 211, 1502
- Tuberculous colitis, *see* Colitis, tuberculous  
   peritonitis, *see* Peritonitis, tuberculous
- Tubes, Paul's, 949, 950
- Tumours, *see* Carcinoma; Sarcoma, etc.
- Turner (G. Grey) on incidence of stomal ulcer, 404  
   sign of acute pancreatitis, 786
- Tutocaine, 29
- Typhoid fever, in relation to gall-stones, 727  
   spontaneous rupture of the spleen in, 891
- U
- Ulcer, duodenal, *see* Duodenal ulcer  
   gastric, *see* Gastric ulcer  
   peptic, *see* Gastric and duodenal ulcer  
   rodent, *see* Rodent ulcer  
   stomal, *see* Gastric and duodenal ulcer, secondary
- Ulcerative colitis, *see* Colitis, ulcerative
- Ulcer-cancer of the stomach, 418, 561  
   definition of, 561  
   diagnosis of, 428, 436  
     accessory methods in, 562  
     detection of faecal occult blood, 564  
     gastric analysis, 562  
   incidence of, 561  
   pathology of, 565  
     macroscopical appearances, 565  
     microscopical appearances, 566  
   symptoms of, 436, 562  
   treatment of, 444  
   X-ray appearances in, 1496  
   (*see also* Stomach, carcinoma of)
- Ulcer diathesis, 181
- Ultra-violet light in treatment of purpura hæmorrhagica, 876
- Umbilical hernia, *see* Hernia, umbilical
- Urea concentration test, 133  
   tolerance test, 134
- Ureters, congenital anomalies of, X-ray  
   diagnosis of, 1551  
   dilatation of, X-ray diagnosis of, 1551  
   X-ray examination of, 1540, 1551
- Urinary tract, pyelography of, 1540, 1541  
   X-ray examination of, 1540
- Urine, ammonia-coefficient of, in hepatic disease, 773  
   bile-pigment in, 751, 753  
   in dyspepsia, 131  
   in pyloric obstruction, 292  
   retention of, following herniotomy, 1054  
     following operations for hæmorrhoids, 1290  
   post-operative, 1245  
     causes of, 1247  
     incidence of, 1245  
     treatment of, 1247  
   urinary nitrogen-coefficient in hepatic disease, 773
- Uterine hæmorrhage, radio-therapy of, 1665

- Uterus, carcinoma of, diagnosis of, 1659  
     radio-therapy of, 1643, 1658, 1660  
     treatment of, 1660  
 chorion epithelioma, of, 1660  
     diagnosis of, 1661  
     radio-therapy of, 1661  
     treatment of, 1661  
 chronic inflammation of, as causative factor in stricture of the rectum, 1381  
 fibroids of, radio-therapy of, 1668  
 radio-therapy in carcinoma of, 1643, 1658, 1660  
 sarcoma of, radio-therapy in, 1661, 1662  
 (see also *Cervix uteri*)

## V

- Vagina, carcinoma of, radio-therapy in, 1662  
     tuberculosis of, radio-therapy in, 1670  
 Vaginal examination in abdominal disease, 115  
 Van den Bergh reaction, 750  
     indirect, 750, 751, 752  
     interpretation of results of, 753  
     technique of, 752  
 Varicocele, anaesthesia in operations for, 53  
 Varicose veins of the oesophagus in splenic anaemia, 867  
 Vater, ampulla of, carcinoma of, 603, 604, 640  
 Verbrugghen on the spread of gastric carcinoma, 425, 501  
*Vermiform appendix*, see *Appendix*  
 Vick's cases of acute intestinal obstruction, 974, 979  
     of acute intussusception, 1022  
     of strangulated hernia, 1034  
     of volvulus, 1026  
 Villous tumour of rectum, 1399  
 Vinethene anaesthesia, 13  
 Viscera, enlargement of, in dyspepsia, 110  
     transposition of, X-ray diagnosis of, 1527  
 Visceral pain, 104  
     tenderness, in abdominal disease, 106  
 Visceroptosis, and duodenal ileus, 622, 624  
     and ileus, 1203

- constipation in, 84  
 dyspepsia and, 72  
 pain in, 75  
 Vitamins B and C, in treatment of purpura hæmorrhagica, 877  
 Volvulus, 1026  
     aetiology of, 1027  
     acute, of colon, 948  
     neonatorum, 1030  
     of the caecum, 1030  
     of the colon, 948  
     of the sigmoid, 1027  
     clinical features of, 1027  
     pathology of, 1027  
     treatment of, 1028  
     of the small intestine, 1029  
     aetiology of, 1029  
     clinical features of, 1029  
     diagnosis of, 1029  
     pathology of, 1029  
     treatment of, 1029  
 Vomiting, after gastric operations, 394  
     late causes of, 399  
     palliative measures in, 395  
     secondary operative measures in, 396  
     after apical analgesia, 38  
     in acute dilatation of the stomach, 162  
     in acute intestinal obstruction, 991  
     in benign growths of the stomach, 524  
     in dyspepsia, 77, 78  
     in hour-glass stomach, 270  
     in infantile pyloric stenosis, 147  
     in peptic ulcer, 76  
     in pyloric obstruction, 291  
     ketosis as cause of, in children, 98  
     nervous causes of, 97, 98  
     reflex, 97  
 Vulva, carcinoma of, radio-therapy in, 1663

## W

- Walters' (Waltman) cases of transplantation of external biliary fistula, 711  
     on incidence of sarcoma of the stomach, 517  
     on treatment of obstruction of the common bile-duct, 659  
 Walton on acute dilatation of the stomach, 160  
     on incidence of anastomotic ulcer, 404

- on pathology of duodenal carcinoma, 604, 621
- operation for hour-glass stomach, 280
- operation of indirect implantation of the common bile-duct into the duodenum, 712
- results in cholecystectomy for chronic cholecystitis, 719
- Wandering spleen, *see* Spleen, wandering
- Wangensteen's method of duodenal drainage by suction-siphonage, 398
- Ward and Smith's results in radium treatment of cancer of the tongue, 1586
- Wassermann reaction, value of, in abdominal disease, 138
- Water-brash, in chronic duodenal ulcer, 199
  - in dyspepsia, 80
- Watersheds, abdominal, 110, 1199, 1212
- Westminster Hospital radium bomb, 1572
- Weight, in carcinoma of the stomach, 438
  - in dyspepsia, 81
  - in gastric and duodenal ulcer, 81
  - in gastro-intestinal diseases, 91
  - loss of, in carcinoma of the stomach, 81
    - in hour-glass stomach, 270
    - in infantile pyloric stenosis, 148
    - in obstruction of the common bile-duct, 81
    - in œsophageal obstruction, 81
    - in pyloric obstruction, 292
    - in pyloric stenosis, 81
    - in stricture of the rectum, 1284
- Whitehead's operation for hæmorrhoids, 1292
- Wilkie's method of closing a mid-line epigastric incision, 322, 323
  - on chronic cholecystitis, 643, 653
  - on chronic duodenal ileus, 622, 624
  - on incidence of carcinoma of the gall-bladder and bile-ducts, 638
  - operation for gastro-colic fistula, 313
  - operations for gall-stones, 642
  - results in cholecystectomy for chronic cholecystitis, 719
  - results in gastro-duodenostomy, 315
  - two-stage operation for gastro-jejunal fistula, 412
- Wilms' method of pyloric occlusion for gastric and duodenal ulcer, 334, 337
- Witzel's jejunostomy, 458
  - for duodenal fistula, 264
  - method of enterostomy for acute intestinal obstruction, 1006
  - method of gastrostomy, 452
- Wolf-Schindler gastroscope, 87
- Wolfer's operation of gastro-jejunostomy, 359
- Women, radio-therapy in diseases of, 1625
- Worms, intestinal, as ætiological factor in appendicitis, 912
- Wound infection, *see* Abdominal wounds, infected
- Wright (Garnett) on anastomotic ulcer, 403, 404, 405, 409

## X

- X-ray diagnosis, 1483
  - of actinomycosis of the colon, 1521
  - of acute intestinal obstruction, 994
  - of appendicitis, 1515
  - of benign tumours of the colon, 1523
    - of the duodenum, 1510
    - of the stomach, 1502
  - of carcinoma of the colon, 1524
    - of the duodenum, 1510
    - of the gall-bladder, 1539
    - of the jejunum, 611
    - of the stomach, 1494
      - carcinomatous ulcer, 1496
      - differential diagnosis, 1500
      - direct signs of cancer, 1498
      - indirect signs of cancer, 1498
      - "leather-bottle" stomach, 1497
      - polypoid growth, 1495
  - of cholecystitis, 1534
  - of colitis, 1520
  - of congenital anomalies of the colon, 1526
    - of the kidney, 1546
    - of the ureters, 1551
  - of congenital hypertrophic stenosis of the pylorus, 1506
  - of diaphragmatic hernia, 1504
  - of dilatation of the ureters, 1551
  - of diseases of the bladder, 1553
    - of the colon, 1520
    - of the duodenum, 1508
    - of the kidney, 1544
    - of the stomach, 1483

X-ray diagnosis, of diverticula of the duodenum, 1511  
 of diverticulum of the stomach, 1489  
 of diverticulitis, 1521  
 of duodenal delay, 1513  
 of duodenal ileus, 623  
 of duodenal obstruction, 1512  
 of duodenal ulcer, 1508  
 of duodenitis, 1508  
 of embryoma of the kidney, 1550  
 of gall-bladder disease, 1534  
 of gall-stones, 1535  
 of gastric cancer, 1494  
 of gastric diseases, 1489  
 of gastric ulcer, 1490  
   inactive gastric ulcer, 1494  
   indirect signs of gastric ulcer, 1492  
   multiple ulcers, 1494  
   ulcers above the incisura angularis, 1490  
   ulcers below the incisura angularis, 1490  
 of gastro-jejunal ulcer, 1503  
 of hydrocephrosis, 1544  
 of hypernephroma, 1549  
 of kidney diseases, 1544  
 of "leather-bottle" stomach, 1497  
 of left-sided colon, 1526  
 of megacolon, 1526  
 of new growths of the gall-bladder, 1539  
 of papilloma of the renal pelvis, 1550  
 of peptic ulcer, 202, 1490, 1508  
 of pyloric hypertrophy, 1506  
 of pyloric stenosis, 1506  
 of recurrent carcinoma of the stomach, 1504  
 of renal calculus, 1544  
 of secondary peptic ulcer, 410  
 of syphilis of the stomach, 1501  
 of transposition of viscera, 1527  
 of tuberculosis of the kidney, 1544  
   of the stomach, 1502  
 of tuberculous colitis, 1520  
 of tumours of the kidney, 1548

of ulcer-cancer of the stomach, 1496  
 of ulcerative colitis, 1520  
 (see also X-ray examination)

X-ray examination, 1483  
 of the alimentary tract, 1483  
 of the appendix, 1515  
 of the bladder, 1540, 1552  
 of the colon, 1518  
 of the duodenum, 1507  
 of the gall-bladder, 1527  
 of the ileum, 1513  
 of the jejunum, 1513  
 of the kidneys, 1540  
 of the small intestine, 1513  
 of the stomach, 1483  
 of the ureters, 1540, 1551  
 of the urinary tract, 1540  
 (see also X-ray diagnosis)

X-ray therapy, see Radio-therapy  
 X-rays, high voltage, in treatment of malignant disease in gynaecological cases, 1671

apparatus, 1674  
 condition of the skin area to be irradiated, 1672  
 dosage, 1676  
 extent and environment of primary lesion, 1673  
 field selection, 1675  
 immediate and after-effects of irradiation, 1680  
 immediate object of treatment, 1674  
 methods of application, 1675  
 pathology in relation to, 1674  
 physical factors in, 1675  
 physics of, 1674  
 precautions taken during irradiation, 1680  
 preparation of the patient, 1671  
 site of primary lesion, 1673  
 technique of, 1677  
 unit of measurement, 1675  
 (see also Radio-therapy; Radium; Radium therapy)